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ENVIRONMENTAL LEAD EXPOSURE AMONG INNER-CITY CAPE TOWN CHILDREN: A STUDY OF ASSOCIATED RISK FACTORS

BY

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ABSTRACT

Risk factors for lead exposure among children in South Africa have not been well documented. This study elucidated important factors which co-vary with lead in increasing children's risk of exposure.

Two study designs were used. First, a cross-sectional analytical study involving first grade school children was executed. Venous blood samples from children were analysed for lead using atomic absorption spectrophotometry. In addition other haematological and anthropometric measurements were conducted. A pre-tested questionnaire administered to parents identified risk factors for lead exposure. Statistical analyses, including log-linear models, were used to determine the relationships between biological, environmental, social factors and blood lead. In an environmental study, daily air and dust samples were collected over a year from several sites in the study area, contemporaneously with the blood and questionnaire surveys. Spatial and temporal variations in atmospheric lead were determined. In stage two a case control study was carried out to determine whether risk factors for lead exposure differed among cases (blood lead $\geq$ 24 ug/dl) and controls (blood lead $\leq$ 14 ug/dl). Levels of lead in air, water, paint and dust samples from children's homes were determined and the state of housing evaluated. Home interviews were conducted with parents to assess the role of economic, demographic, cultural, and behavioural factors in increasing children's risk.

13% of coloured children, but no white children were identified with blood lead levels $\geq$ 25 ug/dl. Air lead levels ranged from $<$ 0.5 ug/m^3 to $>$ 1.5 ug/m^3 and dust lead levels from $<$ 550 ppm to $>$ 3 000 ppm. Environmental lead levels were significantly elevated near heavy traffic, particularly during winter months. Baseline exposure was of significance in influencing blood lead levels of children attending schools in direct proximity to heavy traffic, where blood lead levels were elevated. In cases, direct inhalation of aerosols, and ingestion of lead in water were not found to be important risk factors. Hand contamination and mouthing were associated with increased risk. Sources of elevated lead were found in the homes of both cases and controls, but were not accessible in the homes of controls.

A conceptual framework for lead exposure in children is proposed, to illustrate how social and environmental factors may act to increase risk. The results have implications for primary and secondary prevention strategies aimed at the community.

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CHAPTER 1

INTRODUCTION AND REVIEW

This chapter puts the present study in perspective, starting with a general historical outline of lead usage and human exposure, and moving to the specific situation relating to lead exposure in children and the South African context. This is necessary in order to understand the background and nature of the topic and the gaps in knowledge as pertain to the current topic.

Lead is a naturally occurring element which is ubiquitous in nature. It is found in the earth's crust and in trace quantities in the biosphere. Through the weathering of rocks, by igneous activity and from the radioactive decay of radon, lead is released to the earth's surface. By far the greatest input to the environment, however, has resulted from human activity (Southwood, 1983).

Lead is a heavy, dense metal which is soft and malleable and resistant to corrosion and weathering. The principal lead bearing ore is galena, which is accessible and easy to smelt. It is found mostly in deposits associated with other minerals such as zinc, but the proportion of different metals like copper,

silver, zinc, differs in the ores of various countries (WHO, 1977).

Compared to other metals such as aluminium and iron, lead is relatively rare, but is much more abundant than toxic heavy metals such as cadmium and mercury. In terms of tonnage consumed, lead ranks fifth among metals after iron, copper, aluminium and zinc. It is produced in much larger quantities than other toxic heavy metals (EPA, 1986b). It also has a long history of use.

1.1 HISTORY OF LEAD USAGE AND HUMAN EXPOSURE

Lead is one of the seven metals which have been known since antiquity and was probably one of the first metals to have been smelted (Krysko, 1979, in Smith, 1986). Lead ores were mined extensively throughout ancient times, mainly for their silver content, which was a by-product of the lead smelting process.

Evidence for the use of lead dates back about 6 000 years. Small beads of metallic lead have been found with other artefacts dating to about 7 000 to 6 500 B.C., on the Central Anatolian Plain (Krysko, 1979, in Smith, 1986). A lead figure in the British museum dates to before 3 800 B.C. (Hunter, 1978).

Up until Roman times, lead was a relatively unimportant metal and was used only in small quantities. It was used mainly for small decorative objects (figurines, rings, beads), but had other more practical uses as well. Its soft and malleable properties made it suitable for use in weights and sinkers in fishing nets, and it was also used in products such as cosmetics, medicines, paint and glazes. It is thought that lead was used for glazing pottery by the Egyptians as long ago as 7 000 to 5 000 B.C. (Nriagu, 1978a).

Powdered galena was used as eye paint in Ancient Egypt and the Ancient Chinese and Classical Greeks used white lead on the face (Smith, 1986). Lead was also added to bronze and was used as a solder. In Assyria, about 2 000 B.C., lead was used as a form of currency (Waldron, 1973). Pigments were also used for decoration or writing. One of the earliest references to lead is found in Egyptian hieroglyphics of about 1 500 B.C. and in the Old Testament there are references to lead as an item of trade with the Phoenicians (Moore et al., 1977).

In Roman times lead was used in abundance. British mines were used extensively by the Romans, and Hadrian's Wall is thought to have been built partly to protect the mines in Northumberland and Cumberland (Moore et al., 1977). Lead was used mainly for lining water pipes and cisterns. Lead sheet was also used for roofing and for covering keels of ships. It was used in jewellery, ornaments, cosmetics and medicines. Lead compounds

were used as pigments for colour and in glass. Lead was also used to make or line sarcophagi (Smith, 1986).

The Greeks and Romans coated bronze or copper cooking pots with lead, which contributed to the contamination of food and drink. Contamination of food also derived from lead, pewter and lead soldered cooking implements (Nriagu, 1983). A lead-containing sweetening agent called sapa was added to food and, among other things, to wine. This boiled-down grape syrup was prepared in a lead pot and was added to wine to enhance its colour, sweetness, bouquet and preservation (Nriagu, 1983). It was also introduced to wine in many other ways, according to Gilfillan (1965). Pliny noted at the time that "genuine, unadulterated wine is not to be had now, not even by the nobility" (in Waldron, 1973). The average annual consumption of wine in Ancient Rome has been estimated to be about one to five litres per day per person (Nriagu, 1983).

Due to the widespread use of lead in Roman times, it is likely that lead poisoning was a relatively common occurrence. Dioscorides noted that corrected wine was "most hurtful to the nerves" and Pliny said, "From the excessive use of such wines arise dangling ... paralytic hands" (in Waldron, 1973). The effects of lead in water pipes were noted in the writings of Vitruvius:

> "... water is much more wholesome from earthenware than from lead pipes. For it seems to be made injurious by lead because cerusse is produced by it; and this is

> said to be harmful to the human body. Thus if what is produced by anything is injurious, it is not doubtful but that the thing is unwholesome in itself. We may take example by the workers in lead who have complexions affected by pallor. For when, in casting, the lead receives the current of air, the fumes from it occupy the members of the body and rob the limbs of the virtues of the blood. Therefore it seems that water should not be brought in lead pipes if we desire to have it wholesome"

(in Waldron, 1973).

Gilfillan (1965) noted that the low birth rate and high child mortality among the aristocracy in particular may have been caused by lead poisoning, which could eventually have contributed to the fall of the Roman Empire. He suggested that chronic lead poisoning among Romans may have been prevalent around the first and second centuries B.C. from food and drink contamination, particularly after 150 B.C., when rules forbidding wives to drink wine were relaxed. According to Waldron (1973) however, there is little evidence to substantiate Gilfillan's theory of the fall of Rome, which was a slow, gradual process with many contributing factors.

Nriagu has estimated that more than 40 tons of lead had been produced by the time of the fall of Rome (in Chisholm, 1985). Nriagu also noted that saturnine gout was probably prevalent among Roman aristocrats and that lead poisoning may indeed have contributed to the fall of the Roman Empire (Nriagu, 1983). Certain Roman bone samples have been found to have a high lead content, although not uniformly so (Nriagu, 1983).

Whilst there is substantial evidence that the Greeks and Romans were familiar with the symptoms of lead poisoning, descriptions of lead poisoning by ancient writers are uncommon. In 370 B.C. Hippocrates described what was probably lead colic in a man who extracted metals, but the earliest clinical account of lead poisoning (which included descriptions of constipation, colic, pallor, paralysis and ocular disturbances) was written by Nicander in the second century B.C. (Hunter, 1978).

With the fall of Rome in the fifth century, the use of lead declined dramatically but during the early Middle Ages, the demand for lead increased and many old Roman mines were re-opened. As before, the main uses of lead were in roofing and piping. Much domestic ware was made of pewter, some of which contained up to 60% lead (Smith, 1986). In the 17th century there was a new demand for lead in the building of statues. Lead was used for large scale ornamentation for the first time during the building of Versailles between 1668 and 1671, where it was used in many statues and fountains (Smith, 1986).

Several lead poisoning epidemics occurred during the Middle Ages. These were often caused by the ingestion of acidic drinks such as juice, wine, rum and cider, which were kept in lead glazed earthenware vessels or contaminated during the production process.

One of the first lead poisoning epidemics was described by Paul of Aegina in the seventh century. Other outbreaks of lead colic occurred sporadically throughout history and are known as the colic of Poitou, the entrapado of Spain, the huttenkatze of Germany, the bellain of Derbyshire, the dry bellyache of the Americas and the colic of Devonshire (Waldron, 1973).

Although the symptoms of these epidemics of colic were well recognised, in many cases the cause was not found to be lead until many years later. In 1616 Francois Citois published an account of the epidemic called colica Pictonum (later known as Poitiers colic) whose cause was not recognised for 150 years (Smith, 1986). The Devonshire colic lasted for many decades and ended in 1767, when Baker discovered lead to be the cause. In the beginning the cause was attributed to the wind, soils or stars, but Baker later found the source to be the cider mills which were contaminated with lead. He also showed lead to be present in the cider.

According to McCord (1953), Baker's report in 1767 was possibly one of the first achievements in scientific epidemiology. Baker was at first condemned by the clergy, the mill-owners and by members of the medical profession but was later knighted and became President of the College of Physicians.

A quote from the time reveals something of the nature of the epidemic:

> "... there were found throughout the town, people who were just like ghosts or statues. Walking artificially, pallid, squallid, lean, with their hands crooked and hanging under their own weight, not being raised to the mouth and other higher parts except by effort (practice, skill); their feet not their own, but the muscles of their shanks making their gait laughable if (it were) not so pitiable ..."

(McCord, 1953).

Benjamin Franklin was also aware of lead poisoning and recognised that "dry gripes" was caused by the distillation of rum stored in lead-lined containers, the rum becoming contaminated from distillation through lead worms and still heads. The drinking of contaminated rum was probably the main source of lead poisoning in the American colonies. Although statistics of the disease are not available, evidence is that among the colonists "the bellies 'roared' and the extremities 'dangled' from New Hampshire through Georgia" (McCord, 1954). Other more unusual causes of lead poisoning included red lead in pepper, litharge in snuff, tea caddies lined with lead, metal foil on foods (McCord, 1954).

During the industrial revolution, lead poisoning from occupational exposure increased. Many of the modern uses for lead, such as the lead-based storage battery, were discovered during this time.

Franklin analysed lead poisoning cases from a hospital in Paris according to their occupations, and found that nearly all worked

in lead related industries (McCord, 1953). Tanquerel des Planches published a treatise on lead poisoning in 1838 on a series of 1 200 cases from the same hospital in Paris. Many had been employed in a wide variety of lead-using occupations and were painters, potters and manufacturers of lead compounds (Nriagu, 1983, in Smith, 1986).

In the Uncommercial Traveller by Charles Dickens there is a quote which relates to the plight of women workers in the white lead mills of London: "Some of them gets lead pisoned [sic] soon, and some of them gets lead pisoned [sic] later, and some, but not many, niver [sic]" (in Smith, 1986).

Alice Hamilton, one of the first American doctors to study industrial hazards and working conditions in America in the early 1900s, recognised the wide array of symptoms and effects of lead poisoning. This was illustrated in 1925 in her book Industrial Poisons in the United States.

Since the mid twentieth century concern has arisen over the health impact on the population at current levels of lead in the environment. The United Kingdom Royal Commission on Environmental Pollution (Southwood, 1983) observed :

> "What makes lead different from other heavy metals is the very much greater scale on which it is used, dispersed and accumulated. This factor, when coupled with its known and postulated effects on health in amounts which may be encountered in the environment and found in the human body, makes lead unique amongst environmental pollutants."

The main properties of lead are summarised in Table 1.1.

TABLE 1.1

PROPERTIES OF ELEMENTAL LEAD (EPA 1986b)

Property	Description
Atomic weight	207.19
Atomic number	82
Oxidation states	+2, +4
Density	11.35 g/cm^3 at 20 ^oC
Melting point	327.5 ^oC
Boiling point	1740 ^oC
Covalent radius (tetradehral)	1.44 Å
Ionic radii	1.21 Å (+2),0.78 Å (+4)
Resistivity	21.9 x 10^{-6} ohm/cm

1.2 SOURCES OF LEAD IN THE ENVIRONMENT

Today the transport industry accounts for the largest proportion of lead used: it is added to petrol, used in storage batteries, and in the solder for joints (Urbanowicz, 1986). Lead is used in the building industry in sheet and pipes. In small amounts lead is used in solder and bearings, in the sheathing of cables, in radiation shielding, plastics, ceramics, glass, paint, ammunition, rubber and inks.

Over five million metric tons of lead are used annually, with six countries accounting for 60% of the world's consumption (Urbanowicz, 1986). In addition, it has been estimated that over a third of the total world supply comes from secondary sources, of which lead acid batteries are the most important (WHO, 1977).

1.2.1 LEAD IN PETROL

In 1921 tetraethyl lead was synthesised in the United States of America by Thomas Midgley; it was added to petrol in 1923 (Rosner and Markowitz, 1985). Tetraethyl and tetramethyl lead additives in petrol increase the octane rating and reduce 'knocking' in the engine, enhancing engine performance. They delay the abnormal state of partial oxidation in which the end-gas ignites, thus allowing engines to operate at higher compression ratios, under greater loads, before knocking occurs. Lead in petrol also protects engine parts from excessive wear by providing a protective and lubricating film which prevents 'valve recession' or erosion of valve seats (Southwood, 1983). Today, internal combustion engines have been redesigned to run on unleaded petrol; many countries have moved towards the introduction of unleaded petrol.

1.2.2 LEAD IN PAINT

Lead pigments have been used in paints since early times. Lead can withstand corrosion and weathering and has been widely used in the building and construction industries. Since the middle of this century, much less lead has been used in domestic paints and today most paint sold for domestic use contains little lead.

In the United Kingdom a voluntary agreement exists between the government and the Paintmakers' Association that warning labels are placed on tins of paint containing more than one percent of lead in the dry film. There is also an European Economic Communities (EEC) directive to this effect (Moore, 1986c). In the United Kingdom lead levels in toys and pencils are stringently regulated by law. In the United States of America, lead in paint is limited to 0,06% in the dry film. Apart from stringent legislation, the United States of America also has extensive lead abatement programmes.

From the mining, smelting and refining of lead and from the use and production of lead-based products, lead is emitted to the environment. From these emission sources, lead becomes deposited in the air, food, dust, soil and water. Studies have shown that, over time, lead levels have risen over the whole of the earth's surface, especially since the industrial revolution and the addition of lead additives to petrol (Murozumi et al., 1969).

1.3 PATHWAYS OF LEAD IN THE ENVIRONMENT

1.3.1 LEAD IN THE ATMOSPHERE

Lead is routinely released to the atmosphere by the burning of lead alkyls in petrol and, on a global scale, to a much lesser degree, from industrial processes like lead smelting, iron and steel production and coal combustion. The release of lead from natural sources is small, about four percent, compared to anthropogenic emissions (Nriagu, 1978c). According to the Environmental Protection Agency (EPA 1986b), lead from automobile exhausts accounts for between 75% and 90% of airborne lead, although the relative contribution varies from place to place. In the United States of America, 89% of lead emissions derive from the combustion of petrol (EPA, 1986b). The relative contribution of petrol-derived aerosols to the total airborne lead load has remained high, despite the introduction of low lead fuels and lead-free petrol, as emissions from stationary sources have decreased at a similar rate (EPA, 1986b). The use of unleaded petrol in the United States of America increased from 10% in 1975 to 59% in 1984. During the period 1976 to 1980, a continuous downward trend in air lead levels which corresponded to decreases in the lead content of petrol over this period, occurred throughout the United States of America (Annest et al., 1983).

The amount of lead in the atmosphere varies spatially and in time. Atmospheric lead concentrations today are thought to be many times higher than those that existed in prehistoric times. Global concentrations have increased most markedly since the 1920s when lead was added to petrol. The average atmospheric lead concentration in a particular area is, in general, related to population density. For instance, atmospheric lead concentrations are usually highest in urban areas, particularly around streets with heavy traffic densities. The exact concentration of lead particulates in a particular place and time depends on many factors including the following:

- Traffic density and distance from the source;
- Operating mode of vehicle;
- Type of vehicle and vehicle maintenance;
- Meteorological variables such as wind speed and direction, relative humidity, frequency and duration of temperature inversions, rain, snow;
- Vegetation and surface topography.

(Nriagu, 1978c; EPA, 1986b).

High air lead concentrations may also be found around certain industrial sources, particularly in the vicinity of lead smelters. In remote areas or areas away from urban-industrial centres, lead concentrations may be two to three times lower than urban areas. In confined areas such as tunnels or parking

garages, atmospheric lead concentrations may be much higher than near roadways or urban areas. Concentrations indoors (for example, inside houses) are generally lower than outdoors.

There is a wide variation in the particle size and chemical composition of lead aerosols which reflects both the source characteristics and the aging history of the leaded aerosols (Nriagu, 1978c). In general, airborne lead consists mainly of inorganic lead particulates, but a small amount (< 10%) exists in the form of gases (lead alkyls). A bimodal distribution of large and small particles occurs near a roadway; this distribution changes with time and distance to a single peak of intermediate sized particles (0.2 to 0.3 um) (EPA, 1986b). In urban atmospheres about five times more lead is associated with smaller particles than larger particles (EPA, 1986b) and lead aerosols from automobiles are generally sub-micron.

The chemical composition of automotive lead particulates is related to particle size. In addition to lead bromochloride, the larger particles also contain lead oxides and oxyhalides. The finer particles contain mostly lead bromochloride as well as minor amounts of the lead ammonium halide $2PbBrCl.NH_4Cl$. The lead halides are photochemically decomposed in the atmosphere (Nriagu, 1978c).

The deposition of the emitted particles depends mainly on their particle size. Whilst the larger particles have limited

atmospheric lifetimes (Harrison and Laxen, 1981), and are deposited in the vicinity of the source, smaller particles may remain suspended in the air for seven to 30 days, and may travel long distances from their source (Chamberlain et al., 1979, in EPA, 1986b).

1.3.2 LEAD IN SOIL AND DUST

Levels of lead in the soil depend on the physical and chemical properties of the soil and on the form of the lead deposited. Soils in urban areas may contain significant amounts of lead from a variety of atmospheric and non- atmospheric sources, the major sources being petrol-derived aerosols and flaking paint chips from old deteriorating housing. Natural sources include the weathering of rocks, ore deposits, volcanoes, fires and blowing dust (Nriagu, 1978b). Soils are the major sink for lead in the environment. Lead may have a residence time of several decades in soil (Southwood, 1983).

There is a curvilinear decrease in the concentration of lead from a highway. Factors which affect the concentration of lead in soil near roads include the nature of the traffic, traffic density, meteorological factors, vegetation and topography (Nriagu, 1978b). Most of the atmospheric lead is retained in the top two to five centimetres of soil, especially soils with

five percent or more organic matter and a pH of five or above (EPA, 1986a).

Factors which affect the lead concentration in street dust at any one site include the nature of the surrounding land use, the time since the street was last cleaned, the local traffic volume and character, the street surface type and condition, public works' practices and the season of the year (Sartor and Boyd, 1972, in Nriagu, 1978b). In street dust, lead is concentrated mainly in the smaller particles, less than 250u. Roadside soils have a similar lead particle distribution (Nriagu, 1978b).

Lead levels in urban-industrial soils and dusts, and in the vicinity of mines, are generally higher than in rural areas with little industry and traffic. Dust lead in and around households may derive from flaking paint, the atmosphere, or from the workplace via workers' clothing.

1.3.3 LEAD IN FOOD

The lead present in water, air, soil and plants can contribute to the amount of lead present in food, but it is uncertain as to the relative inputs of these factors. Lead in air may be deposited directly onto crops, or in some cases, may be taken up via the soil and by the roots of plants. It may also contribute to the lead content of the water. Lead may also be taken up in the

preparation and processing of food. Some researchers have claimed that lead in air is the chief source of lead in food both through direct contamination of crops and through the food chain. Others maintain that even in heavily contaminated produce from the vicinity of heavily travelled roads or factories, most of the lead is removed in food preparation (DHSS, 1980). Food in cans with lead solder may have increased lead levels, especially where the food is acidic. Particularly relevant is canned milk in infant feeding.

1.3.4 LEAD IN DRINKING WATER

The amount of lead present in drinking water depends on the amount of lead with which it is in contact, and the degree of softness of the water (Southwood, 1983). Where lead pipes are present, lead levels can be significantly increased in domestic water supplies, particularly where water is acidic (plumbosolvent). Other factors such as the temperature of the water and the period of time the water has been allowed to stand are also important. Most reservoirs of water have relatively low concentrations of lead (WHO 1977, in Moore, 1986b) but certain homes may have lead lined tanks which release lead. Lead soldered joints in copper piping may also be a source of lead.

1.4 UPTAKE OF LEAD IN THE HUMAN BODY

In non-occupationally exposed populations, lead is absorbed into the body mainly by ingestion and inhalation. The main route is via the gastrointestinal tract. This includes lead which is deposited in the upper respiratory tract and swallowed. The first studies of lead uptake by humans were carried out by Kehoe in 1937. Between 1937 and 1972 Kehoe's studies involved over 21 000 days of measured exposure in humans (Gross, 1979). He showed that there was considerable individual variability but, from other metabolic balance and isotope studies, it is generally agreed that in adults, between 10% and 15% of ingested lead is absorbed (EPA, 1986a). The rate is increased under fasting conditions (Rabinowitz et al., 1980). Many other factors affect the rate of absorption from the gastrointestinal tract. Deficiency in elements such as calcium, iron, zinc and magnesium, can increase the amount of lead absorbed.

Infants and pre-school children have a much higher rate of dietary lead intake than adults relative to their body weight. They absorb between 40% and 50% of lead via ingestion (Alexander, 1974; Ziegler et al., 1978; Duggan, 1983a). However, the relative rates of absorption may be very variable in individuals (Barltrop and Strehlow, 1978, in EPA, 1986c).

The uptake of particulate lead by inhalation is a function of the particle size and solubility of the aerosol inhaled. The

fractional deposition of lead in the lungs depends mainly on the particle size of the aerosol 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 (Hammond, 1977).

Most of the lead aerosols to which the general population is exposed are submicron and thus are easily absorbed into the blood. The studies of Kehoe (1961a, 1961b, 1961c) and Gross (1981) as well as others (Chamberlain et al., 1978, in EPA, 1986c) have indicated that for the general population, the deposition rate of particulate airborne lead in the lungs is between 30% and 50%, depending on particle size and ventilation rates. All lead deposited in the lower respiratory tract is ultimately absorbed. Children have a greater respiratory intake relative to their body weight than adults.

After absorption, lead is transferred by the blood to the soft tissues and bones. Highest concentrations are found in the liver and kidneys, but some goes to other organs such as the heart, lungs and brain. The half life of lead in the soft tissues is a few months (Moore, 1986a). Following constant exposure over a relatively short time, the blood and the soft tissues reach a dynamic equilibrium with a half life of about 60 days (Moore, 1986a).

More than 90% of lead absorbed is eventually transferred to the bone, where it is present as relatively insoluble lead phosphates. Lead in bone has a much longer half life than in the soft tissues. In children, less lead is deposited in bone and a greater proportion is present in a mobile form in the soft tissues. (About 73% of the total body burden of children is present in bone (EPA, 1986a)). In certain circumstances (such as stress), lead can be remobilised from the bone (Waldron and Stoefen, 1974).

Lead can cross the placental barrier and concentrations in the foetus increase during pregnancy. Cord blood lead levels generally correlate with those of the mother's blood, but are usually lower. Lead accumulates in the body with age, up until approximately 60 years, thereafter a decrease occurs (EPA, 1986a).

Studies of uptake from water and diet have shown that there is a curvilinear relationship between exposure levels and blood lead levels, with the rate of increase at the lower levels of exposure being much greater than at higher levels of exposure. It is likely that the curvilinear relationship also applies to air (Hammond et al., 1981; Brunekreef, 1984; Moore, 1986a). Within usual levels of exposure, a linear relationship normally applies (Lacey et al., 1985, in Moore, 1986a).

1.5 EFFECTS OF LEAD

There is no known biological function for lead and a continuum of harmful or potentially harmful effects exists over a wide range of exposure. Many different organ systems and biochemical/physiological processes may be affected. At relatively low levels, biochemical changes are detectable and impairment of central nervous system function may be caused.

The clinical manifestations of lead poisoning have been well known for centuries. Lead poisoning is usually chronic and accumulation of the body burden occurs over a period of time. Exposure to very high levels of lead, however, can result in the accumulation of toxic levels within weeks. Disruption of the normal functioning of the haematopoietic system, the kidney, liver, heart and central nervous system as well as other organ systems may be caused. At extreme levels, severe brain damage (encephalopathy) occurs and death may result.

Children are at high risk of lead poisoning compared to adults as there is more mobile lead in the soft tissues (where it exerts its toxic effects), and their metabolic pathways are incompletely developed, in particular the blood-brain barrier. In children clinical symptoms of lead poisoning include loss of appetite, vomiting, irritability, abdominal pain and constipation. At high levels, muscle co-ordination may be affected, and loss of consciousness and coma can occur. There is however,

considerable variation in the individual lead levels at which certain effects occur (Southwood, 1983).

One of the most serious effects is irreversible central nervous system damage. In young children lead encephalopathy or death can occur at blood lead levels around 80 to 100 ug/dl. Lead encephalopathy may result in permanent mental retardation and other neurological deficits in children who survive. Thomas and Blackfan (1914) were among the first to document the occurrence of lead encephalopathy in children. Byers and Lord (1943) reported that neurological impairment could occur later in life in children who had previously had lead poisoning.

There has been much debate about the effects on children at relatively low levels of environmental exposure. In a recent major review, the EPA (1986d) summarised the effects of most significance for children. The most important health effects (excluding neuropsychological effects) include haem biosynthesis effects, other biochemical and haematological effects and impairment of nervous system functions. These are briefly discussed below (references quoted are all in EPA, 1986d).

At several stages in the metabolic pathway, lead affects the synthesis of haem. Two enzymes in particular are inhibited: ferrochelatase and delta-aminolaevulinic acid dehydratase (ALA-D). The inhibition of ferrochelatase results in an increase in zinc protoporphyrin (ZPP) in the red blood cells which is

exponentially correlated with blood lead levels in children (Piomelli et al., 1973; Sassa et al., 1973; Lamola et al., 1975a, 1975b; Roels et al., 1976). Levels of ZPP in the blood of children start to increase at levels around 15 ug/dl (Roels et al., 1976; Piomelli et al., 1977; Piomelli et al., 1982). Larger elevations occur as blood lead levels exceed 30 ug/dl.

Iron deficiency is also associated with elevation of ZPP, particularly in younger children. Anaemia may also occur as a result of lead poisoning at levels around 40 ug/dl in children (WHO, 1977).

The threshold level for the inhibition of ALA-D has yet to be determined and the inhibition of ALA-D, which catalyses the conversion of ALA to porphobilinogen, occurs at blood lead levels as low as 10 to 15 ug/dl. The enzyme activity of ALA-D is inversely correlated with blood lead values (Hernberg and Nikkanen, 1970; Wada et al., 1973; Nieberg et al., 1974; Roels et al., 1975b; Alessio et al., 1976b).

The activity of the enzyme Py-5-N is particularly sensitive to inhibition by lead (Angle and McIntyre, 1978; Angle et al., 1982) and may be affected in children at blood lead levels below 30 ug/dl with no apparent threshold. At levels around 12 ug/dl, levels of the vitamin D hormone, 1,25 dihydroxy vitamin D may be effected (Rosen and Chesney, 1983; Chesney et al., 1983).

At moderately elevated blood lead levels, electrophysiological effects may also occur. In children at blood lead levels around 30 ug/dl, peripheral nerve dysfunction effects, indicated by slowed nerve conduction velocities, have been detected. In some asymptomatic children, studies have shown changes in EEG brainwave patterns and CNS invoked potential responses (Burchfiel et al., 1980; Otto et al., 1981, 1982, 1983). This is an important method of study, as measures of EEG are less likely to be affected by confounding variables such as socio-economic status than are psychological measures (Yule, 1986). Caution however, is necessary in interpreting these results.

Effects on children's intelligence and behaviour may also occur at moderately raised blood lead levels, but assessments of low level lead exposures are complex. Rutter (1980, 1983) classified studies of the neuropsychological effects of lead in children according to a number of categories: clinic-based studies, studies of children from the general population, studies of children living close to smelters, studies of mentally retarded or behaviourally deviant children and chelation studies.

Most of the clinic-based studies found lead to be associated with a decrease of three to four Intelligence Quotient (I.Q.) points at blood lead levels around 60 ug/dl or more (Lansdown, 1986). According to Rutter (1980), despite differences in interpretations of the results of certain studies, "... it is very likely that psychological impairment occurs in some

asymptomatic children with repeated blood lead levels in the 40 to 80 ug/100 ml range ...". According to the EPA (1986a) there is clear evidence of neuropsychological effects in the 30 to 50 ug/dl range (a decrease of about four I.Q. points). In the 50 to 70 ug/dl range, there is evidence of a decrease of about five I.Q. points.

Evidence of effects at blood lead levels below 30 to 40 ug/dl are less clear. The neuropsychological abnormalities most often associated with moderately raised levels of lead in children are those related to attention and intelligence: distractability, poor concentration, dreaminess, IQ deficits and other symptoms. Whilst studies prior to 1980 looked mainly at children with blood lead levels above 35 to 40 ug/dl, studies carried out subsequent to this time have looked at children with lower blood lead levels.

Some studies of the 10 to 30 ug/dl range have shown small effects on children's behaviour (Needleman et al., 1979; Yule et al., 1981; Needleman, 1983; Bellinger and Needleman, 1983; Lansdown et al., 1983; Winneke, 1983; von Schirnding and Fuggle, 1984a; Fulton et al., 1987), whilst other studies have failed to find any statistically significant effects on children with low levels (McBride et al., 1982; Yule et al., 1983; Smith et al., 1983; Harvey et al., 1983). Nevertheless, some of these studies found persistent one to two point I.Q. deficits in children with moderately raised lead levels, although these did not reach

statistical significance after social factors had been controlled for (Smith et al., 1983).

The reasons for the discrepancies in many of these studies have largely to do with methodological problems encountered in the design and execution of studies. Several recent reviews have alluded to such issues (Rutter, 1980; DHSS, 1980; Needleman, 1980; Needleman and Landrigan, 1981; Rutter, 1983).

Studies have suffered from one or more of the following problems highlighted by Rutter (1983) and others: weak and insensitive measures of neuropsychological functioning, inadequate markers of exposure to lead, small samples, multiple tests, confounding variables (e.g. pica and socio-economic status), selection bias resulting from use of clinic samples, volunteer subjects, loss of follow-up, and inadequate statistical measures.

More recently, sophisticated longitudinal prospective studies on the effects of lead on prenatal and postnatal development have been designed and are being carried out in many countries throughout the world (Bornschein and Rabinowitz, 1985). These differ in design from the earlier cross-sectional studies referred to above, and use common measures of exposure and developmental outcomes, considering also numerous covariates and confounding variables.

Bellinger et al. (1984) were the first to report on the relationship between low level prenatal exposure and developmental outcomes. Other studies have subsequently been reported by Bellinger et al., 1985, 1986; Ernhart et al., 1985, 1986; Dietrich et al., 1986, 1987 (all in Davis and Svendsgaard, 1987); Bellinger et al., 1987, and others. In most of these studies, foetal exposure levels (as indicated by maternal or umbilical cord blood lead concentrations at birth) have averaged around 10 ug/dl. Results have shown a relationship between lead exposure during foetal development and deficits in neurobehavioural performance. At blood lead levels as low as 10 to 15 ug/dl, effects such as impaired neurobehavioural development, reduced gestational age and lowered birth weight may occur (Davis and Svendsgaard, 1987). The degree to which the effects of prenatal exposure persist in children remains to be seen.

Due to the increased evidence of significant effects of lead on the nervous system and other important physiological processes at decreased levels, the action level for lead in blood of children has recently been lowered to 25 ug/dl in the United States of America (CDC, 1985).

1.6 CHILDHOOD LEAD EXPOSURE

Young children are known to be liable to have the highest blood lead levels of any non-occupationally exposed group. The reasons for this are many-fold. As indicated earlier, physiological factors predispose children to greater rates of intake into the respiratory and gastrointestinal tracts, and children have greater lead absorption and retention rates. Nutritional deficiencies known to potentiate lead toxicity are also more likely to occur in young children. Differences in certain habits, such as normal hand-mouth behaviour and pica (the abnormal extension of mouthing behaviour, whereby children purposefully ingest non-food items) may result in ingestion of lead contaminated dust, dirt or paint chips. Finally, children are most vulnerable to lead's toxic effects. For these reasons, young children are considered to be the group at highest risk for both high and low level lead exposure (Lin-Fu, 1975). The following section discusses the history of lead poisoning in children.

Unlike the mills and mines, few children were employed in the lead industry, though it is likely that they were indirectly exposed to lead via working parents, or via food and drink throughout the ages. The rate of spontaneous abortion, stillbirth and premature delivery was high among lead workers, as was the infant mortality rate in children of lead workers (Oliver, in Smith, 1986).

Around the turn of the century, many sources of lead exposure in young children were recognised, such as chrome yellow in food dyes (Stewart, 1895), lead food receptacles, lead medicinal ointments and others (Dufour-Labastide in Holt, 1923). Cases of lead poisoning among infants were relatively rare at this time. A few cases, caused by the use of lead ointments by nursing mothers were reported in the literature (Holt, 1923). Cadman, in 1902, was the first to identify lead nipple shields used by nursing mothers as a source of poisoning in infants (in Gordon and Whitehead, 1949). Other workers reported similar cases (Wilcox and Caffey, 1926; Findlay, 1935, in Clark, 1950; Rapaport and Kenney, 1939; Gordon and Whitehead, 1949).

In Japan, lead salts used in toilet powders were thought to have been the source of unrecognised lead poisoning incidents in infants, such as were first described in 1784 and later diagnosed as "meningitis of unknown aetiology" (Kato, 1932). It was Hirai, in 1923, who first suggested that this disease was lead poisoning, caused by the use of toilet powders by nursing mothers (Suzuki and Kaneko, 1924; Hirai, 1927; Kato, 1932). Between 1925 and 1930, 59 cases of lead poisoning were reported in infants in Tokyo (Kato, 1932).

1.6.1 PICA, PAINT, POVERTY AND PLUMBISM

Incidents of lead poisoning in young children were common in Australia during the early part of this century (Turner, 1909; Gibson, 1917; B.M.A., 1922). The first diagnosed cases were recorded in 1892 in Queensland by Gibson and co-workers. Ten reported cases occurred between 1891 and 1892, whilst one case occurred in 1890 (B.M.A., 1922). Between 1898 and 1903, 85 cases of lead poisoning were deteced in Queensland.

It was only in 1904 that Gibson first proposed that painted walls and railings were the most likely source of lead in these children (Gibson, 1904). He suggested that young children touching freshly painted surfaces, or more particularly, weathered painted veranda railings or walls, could become exposed by ingestion of the powdery lead paint. He implicated the "biting of finger-nails or sucking of fingers" as the main mechanism of exposure. He also recognised that paint might contaminate dust and be inhaled or ingested. Although Gibson's theory regarding the aetiology was initially met with scepticism, it soon gained widespread acceptance (B.M.A., 1922). By 1908, 262 cases of lead poisoning had been admitted to the Children's Hospital in Brisbane (Turner, 1909).

In the United States of America, childhood lead poisoning was infrequently identified during this period. The first cases were reported by Stewart (1895); Thomas and Blackfan (1914);

Blackfan (1917); Strong (1920); Holt (1923) and others. In most cases, lead paint was identified as the chief source.

Ruddock, in 1924, was one of the first to draw attention to the role of pica in childhood lead poisoning. The repeated ingestion of paint on objects such as window sills, railings, furniture and toys was recognised as being an important pathway of lead exposure among young children (Ruddock, 1924; McKhann, 1926).

There was an increase in awareness in the United States of America during the 1930s of the problem of lead poisoning in childhood, which was associated in part with improved methods of radiographic diagnosis (Park et al., 1931; McKhann and Vogt, 1933). From 1924 to 1933 there were 86 cases of lead poisoning among children aged five years and below in children's hospitals in Boston (McKhann and Vogt, 1933). Whilst in 1920 Strong had stated that lead poisoning "does not seem to be common in children" (Strong, 1920), in 1940 Holt observed that lead poisoning was "one of the common and most serious forms of intoxication recognised in childhood" (in Clark, 1950).

Lead poisoning in children was associated with a high mortality rate, and from 1931 to 1940, 202 deaths from lead poisoning in children under the age of 15 years were reported in the United States registration area (McDonald and Kaplan, 1942). During the 1950s in particular, there was an increase in the number of

reported cases of lead poisoning in children, which was associated with improved methods of case-finding and accuracy of clinical diagnosis among health workers (McLaughlin, 1956; Byers, 1959). The incidence of lead poisoning during this time is difficult to determine, as in many places lead poisoning was not a reportable disease and the diagnosis was made with difficulty (Greenberg et al., 1958). In New York city, where lead poisoning was a notifiable disease, an increase in the number of cases occurred from one in 1950 to 80 in 1954 (McLaughlin, 1956). Associated with this increase in the number of cases was a decline in mortality; the case fatality rate dropped from 55% in 1952 to 16,3% in 1954 in New York. By 1957, 416 cases of lead poisoning had been reported in New York city (Greenberg et al., 1958).

Whilst numerous case reports on childhood lead poisoning had appeared in the American literature by the early 1950s, there were only a few sporadic reports on lead poisoning among children published in the British literature during this time (Clark, 1950). These included case reports by Stephenson (1898); Cadman (1902, in Gordon and Whitehead 1949); Rodgers et al. (1934); Findlay (1935, in Clark 1950); Gordon and Whitehead (1949); Clark (1950) and Burrows et al. (1951). Between 1900 and 1952, only 19 cases of lead poisoning among children over a period of 50 years had been reported in Great Britain. About 50% of these cases were caused by the ingestion of paint on woodwork, mainly on cots (Millichap et al., 1952).

Data published in the United States of America during the 1950s and 1960s indicated that lead poisoning in children, caused primarily by pica, was prevalent among socially-deprived inner city children living in dilapidated housing with flaking lead paint (Mellins and Jenkins, 1955; Tanis, 1955; Bradley et al., 1956; Chisholm and Harrison, 1956; McLaughlin, 1956; Cohen and Ahrens, 1959; Byers, 1959; Jacobziner and Raybin, 1962; Christian et al., 1964; Griggs et al., 1964; Greengard, 1966a).

Whilst most published studies were based on case-series reports, Griggs et al. (1964) carried out an epidemiological study of children living in old and new housing. These researchers confirmed the relationship between increased lead absorption and residence in old deteriorating housing. Studies in certain slum areas showed that 50% to 70% of old houses in parts of the United States of America contained flaking lead pigment paints (Schucker et al., 1965, in American Academy of Pediatrics, 1969).

The repeated ingestion of lead paint chips over a period of three months or more could lead to the onset of clinical symptoms and eventually to lead encephalopathy (American Academy of Pediatrics, 1969). The case-fatality rate for lead poisoning in American children during the 1960s was around 15% to 20%, and neurological and mental disturbances from encephalopathy occurred in more than 25% of those children who survived (Jacobziner, 1966).

Pica for paint among poor children living in old housing was also recognised as an important aetiological factor in childhood lead poisoning in countries such as Great Britain (Moncrieff et al., 1964) and Australia (Freeman, 1970).

During the late 1960s and early 1970s, mass screening programmes (as opposed to the earlier case-finding activities) were undertaken in North America to detect high risk children (Lin-Fu, 1975). One of the first of these large screening studies was carried out on 68 744 inner city children in Chicago between 1967 and 1968. 5,7% of children aged one to six years were detected with blood lead levels $\geq$ 50 ug/dl (Blanksma et al., 1969).

Though lead poisoning was largely a phenomenon of the large inner cities, it was also known to occur outside of these areas. One of the first studies to determine the prevalence of raised blood lead levels in smaller cities was carried out in Illinois, United States of America, in 1971 (Fine et al., 1972). This study revealed that 18,6% of 6 151 children aged one to six years, living in areas of substandard housing, had blood lead levels $\geq$ 40 ug/dl.

Lead intoxication was also known to occur in rural areas (Greengard, 1966b). Cohen et al. (1973) were one of the first to determine the blood lead levels among 230 rural children, aged one to five years, living in New York State and Connecticut.

Nine percent of these children were found to have blood lead levels $\geq$ 40 ug/dl.

Whilst lead poisoning occurred mainly among young children aged one to six years, the prevalence among one to three year old children was highest (Chisholm and Harrison, 1956; U.S. Public Health Service, 1971). This age group also had the highest mortality (U.S. Public Health Service, 1971).

In the United States of America, particularly in cities such as New York, a large number of cases occurred among Puerto Rican and Negro children (McLaughlin, 1956; Jacobziner and Raybin, 1962). This was attributed to the fact that most of these children lived in high-risk areas.

Jacobziner in 1966 compared lead poisoning in children to an iceberg - the tip representing cases of lead encephalopathy and the larger portion representing asymptomatic children (Jacobziner, 1966).

1.6.2 OTHER PATHWAYS OF LEAD EXPOSURE

Whilst ingestion of lead paint was regarded as being the most important pathway to high level exposure among children, it was thought that other sources and pathways of lead might be responsible for exposure, particularly in some children with only

moderately raised lead levels. Dufour-Labastide, in 1902, observed a case of lead poisoning in an infant due to the inhalation of lead in dust (in Holt, 1923). In 1932 in Baltimore, United States of America, 40 cases of lead poisoning occurred, mainly among children from poverty-stricken homes, which were thought to have been caused by the inhalation of fumes from the burning of storage battery casings for fuel (Williams et al., 1933). Other outbreaks of lead poisoning from the burning of battery casings occurred in Canada in 1952 (Wyllie, 1954) and Great Britain in 1954 (Gillet, 1955; Travers et al., 1956). It was suggested by Gillet that ingestion, rather than inhalation, may have been the chief route of exposure in the United Kingdom outbreak.

1.7 EPIDEMIOLOGY OF LOW LEVEL LEAD EXPOSURE

1.7.1 LEAD IN THE ENVIRONMENT

In the late 1960s attention turned to environmental sources of lead, particularly lead in petrol. One of the first to draw attention to this source was Patterson. He said that urban dwellers may have body lead burdens some 100 times greater than are likely to have existed in prehistoric times. This was due, he said, to the accumulation of lead wastes in urban areas from lead alkyls, as well as from other sources (in Chisholm and Kaplan, 1968). Patterson's statement may have been among the

first which started the controversy over lead in petrol in the 1970s. As early as the 1920s however, concern existed over the health effects of the introduction of lead in petrol.

In 1922 Thomas Midgley and co-workers discovered that lead was a powerful anti-knock compound. One of the earliest 'epidemiological' studies on environmental exposure to lead in petrol was carried out in the 1920s. Results of a study of garage and filling station attendants and chauffeurs carried out in Cincinnati revealed no immediate hazardous effects from lead, but the authors warned of the possibility of long term effects and potential ill-effects associated with increased use of leaded petrol in the future (Rosner and Markowitz, 1985).

In 1968 Chisholm and Kaplan stated that there was increasing concern over the problem of environmental pollution (Chisholm and Kaplan, 1968). In 1973 Chisholm said that blood lead levels in the lower ranges may reflect passive exposure to 'low dose' environmental sources, rather than active ingestion of lead from high dose sources such as lead paint (Chisholm, 1973).

Studies of prehistoric samples of bone and/or teeth show that lead levels in current urban populations are indeed many times higher than those that existed in pre-metallurgic societies (Shapiro et al., 1975; Grandjean et al., 1979; Fosse and Wesenberg, 1981). Several studies have also looked at blood lead levels in remote populations and have found that they are

considerably below those of other populations (Piomelli et al., 1980; Poole et al., 1980).

Studies that have compared blood lead levels of adults and children living in urban and suburban or rural communities have found that lead levels in people living in urban areas are higher than others (Hofreuter et al., 1961; Goldwater and Hoover, 1967; Thomas et al., 1967; Cohen et al., 1973; Okubo et al., 1978; Drossos et al., 1982; Brunekreef et al., 1983).

In many studies however, populations of children from urban areas have not been properly matched with those from other areas. There is therefore a great range in the blood lead levels reported in the various studies and in many cases the populations are not comparable. For instance in Cohen et al.'s comparative study of urban and rural children, the rural group was primarily white middle income children, whereas the urban group consisted of black low income children (Cohen et al., 1973). Some studies have shown no differences between urban and rural groups (Taskinen et al., 1981; Elwood et al., 1982).

The lack of data on potentially confounding factors makes many studies difficult to interpret. Data from the Second National Health and Nutrition Examination Survey (NHANES II) in the United States of America are useful in providing information on this issue. In this nation-wide survey of a representative sample of the United States population, blood lead levels were found to

increase with the degree of urbanisation, for all groups except black adults (Mahaffey et al., 1982).

Other researchers have examined the blood lead concentrations of populations living near and away from traffic or major roadways. Some have found higher blood lead levels close to major roadways (Daines et al., 1972; Caprio et al., 1974; Johnson et al., 1975) whilst others have not (Elwood et al., 1982). However, in many of these studies, no information on confounding factors is given.

Whilst it is thus recognised that in general urban children have higher blood lead levels than other children, the reasons for raised blood lead levels have been inadequately studied. Recent studies from various parts of the world which have examined multiple sources and pathways have indicated that the relationship between blood lead levels and sources is likely to be multifactorial, the exact relationship depending on many different factors and circumstances. Due to the multiplicity of sources and pathways of exposure, it is extraordinarily difficult to quantify the contribution of different factors.

Pathways of lead - in air, dust, soil, food and water - to the body, have in general not been adequately studied. It has proved exceedingly difficult to obtain reliable measures of the exposure of individuals to various sources. Not only does the distribution of lead in sources such as air, soil and dust vary

considerably in the environment (both spatially and temporally), but so too does the behaviour of individuals, which puts them in a different relationship to sources of lead in the environment. Some of the methodological problems in studies investigating the blood lead : environmental lead relationship (with reference to lead in air) are discussed below.

1.7.2 EXPOSURE TO LEAD IN AMBIENT AIR

1.7.2.1 Direct Inhalation Pathway

Studies which have looked at the direct contribution of atmospheric lead include, among others, experimental studies in which volunteers are exposed to varying amounts of lead in the air (Kehoe, 1961a, 1961b, 1961c; Griffin et al., 1975, in EPA, 1986c; Gross, 1981). Stable isotopes have also been used to quantify the amount of lead in the air transferred to blood (Rabinowitz et al. 1974; Chamberlain et al., 1975; Manton, 1977; Yaffe et al., 1983; Tera et al., 1985).

The biases in such experimental studies, however, may be significant. Often the number of subjects studied are of necessity small, sometimes involving only a few individuals who are not representative of the general population. Also, they may be exposed to very high concentrations of lead in the air, which do not represent ambient conditions.

Another approach has been to determine air lead-blood lead correlations in the population (Daines et al., 1972; Azar et al., 1973, in Russell Jones and Stephens, 1983; Johnson et al., 1975; Tepper and Levin, 1975). The method of air sampling varies considerably in these respective studies. Azar et al. used personal samplers to measure exposure whereas Tepper and Levin used fixed air monitors. Some studies have relied on estimates of atmospheric lead which might be entirely inappropriate, both in terms of spatial and temporal relationships, to the population being studied. Goldsmith and Hexter (1967) developed a linear regression relationship plot of log blood lead versus log air lead. Experimental and epidemiological data tended to converge on the same regression relationship but air lead samples were not necessarily taken at the same time and place as the blood samples.

In other cases, populations such as those living around lead smelters are studied (Landrigan et al., 1975; Roels et al., 1976, 1980; Yankel et al., 1977; Brunekreef et al., 1981; Wilson et al., 1986). Such situations are not representative of usual ambient concentrations of lead.

1.7.2.2 Indirect Pathways

Studies which have concentrated only on the contribution of inhaled lead to the body burden ignore other important indirect pathways of atmospheric lead such as lead in food and dust. In children particularly, hand-mouth activities can introduce significant amounts of lead into the body. In some studies the hands of inner city children have been found to have greater concentrations of lead-rich dust than those of other children (Sayre et al., 1974; Lepow et al., 1974). Studies of exposed groups such as taxi drivers (Flindt et al., 1976) and traffic policemen (Aggarwal et al., 1979) do not provide information on indirect sources of atmospheric lead.

Whilst serious problems have occurred in studies which have examined the blood-lead : air-lead inhalation relationship, even fewer studies have succeeded in determining the nature of the relationship between other pathways of lead in the environment such as dust, soil, food and water. Difficulties exist in measuring these parameters, in sampling, and in most studies, as with those investigating the contribution of air lead, they are based on exposure to very high concentration levels in the environment (Elwood, 1986).

Some studies have looked at time trends in petrol lead, air lead and blood lead (Billick et al., 1979, 1980). Such studies take into consideration the contribution from both direct and indirect

routes. From Billick et al.'s studies in New York city blood lead has been found to correlate more closely with petrol lead than air lead, but air lead determinations were based on one sampler only (Russell Jones and Stephens, 1983).

The NHANES II study detected a significant downward trend in blood lead levels over time (37%) which correlated with decreases in lead in petrol and air over the period (Annest et al., 1983). This association held after controlling for effects of race, sex, age, region of country, season, income and degree of urbanisation. Nevertheless, certain reservations remain as to whether this strong association represents a causal one. Other studies have also detected downward trends in blood lead levels over time (Elwood, 1983; Hinton et al., 1986; Skerfving et al., 1986) but decreases in lead in petrol have not accounted for such trends in some cases (Elwood, 1983; Hinton et al., 1986).

Sinn (1980, 1981) carried out a study in Frankfurt which examined blood lead levels before and after the level of lead in petrol had been reduced. Blood lead levels declined slightly. Waldron (1975) carried out a study which examined blood lead levels before and after the opening of a major motorway interchange. After the opening, blood lead levels increased significantly. Only a relatively small proportion of the original population was followed up, however, and no control population was studied (Elwood, 1984).

An unusual experiment was the isotopic lead experiment carried out in Turin, Italy, which attempted to quantify the contribution of both inhaled and non-inhaled sources of atmospheric lead. In this study the geological source of lead for anti-knock compounds was switched to another source in order to change the isotopic composition of lead in the atmosphere (Garibaldi et al., 1975; Facchetti 1979; Facchetti and Geiss 1982, all in EPA, 1986c). The isotopic composition of the blood from members of the population was subsequently monitored, but only in a small unrepresentative group of adults. It was determined that the minimum contribution of petrol lead to blood lead varied from an average of 24% in Turin to 12.5% in the nearby countryside, to 11.0% in the remote countryside.

The failure to include relevant variables is one of the most serious problem in evaluating studies on lead in human populations. Studies also differ with respect to demographic characteristics, the type of outcome and exposure indices used and the type of environment studied. In addition, because the nature of the various sources may differ considerably from one area to another, it is difficult to extrapolate conclusions from one study to another. According to Elwood (1986), the evaluation of the relative importance of sources is nearly impossible. Accordingly, results of studies which have attempted to quantify the contribution of individual sources should be viewed with caution.

1.7.3 DEMOGRAPHIC FACTORS

Demographic factors which are associated with low level environmental exposure include age, sex, ethnic group and socio-economic status.

1.7.3.1 Age

Children are born with low cord blood lead levels, similar to their mothers, but infant blood lead levels rise sharply after a few months. Prospective studies which have been carried out on children have monitored blood lead levels in infants and young children from birth, taking serial blood samples. Hammond et al. (1983, in Hunter 1986) showed that children are born with low cord blood lead levels which rise steeply between the age of three to six months.

Children aged two to three years have higher blood lead levels than older children and adults (Mahaffey et al., 1982, Duggan 1983b). There is evidence, however, that in children isolated from normal environments (for example, hospitalised children) there is no peak in blood lead levels at two to three years (Duggan, 1983b; Kawai et al., 1987).

The NHANES II survey found that for children under six years of age there was no statistical association between age and mean blood lead concentration, but for children aged six to 17 years, there was a statistically significant association between age and mean blood lead concentration (Mahaffey et al., 1982). Mean blood lead levels decline with increasing age until late adolescence when they gradually increase.

Among adults, there is a significant trend in blood lead levels with age, especially in men. Blood lead levels are positively associated with age until middle age, and then decline.

1.7.3.2 Gender

Blood lead concentrations are not significantly different for young boys and girls under the age of six years. Between the ages of six and 17 years, the difference in mean blood lead levels between the sexes increases with age, with boys having higher blood lead levels than girls. More significant and pronounced differences start with adolescence. Among adults, blood lead levels are consistently and significantly higher in men than in women. This was recently confirmed in the WHO cross-cultural study (Hunter, 1986).

1.7.3.3 Ethnic Group

The NHANES II study found that in the United States of America, blacks have significantly higher blood lead levels than whites (Mahaffey et al., 1982). Among both sexes of all ages (but particularly younger children) and across income groups and residential areas, significant differences between the races have been found. In the United Kingdom, average blood lead concentrations among people from India, Pakistan and Bangladesh have been found to be slightly higher than whites, but due to the relatively small proportion of non-whites sampled, the results should be interpreted with caution (Quinn, 1985).

1.7.3.4 Socio-Economic Status

In the United States of America, raised blood lead levels are associated with low socio-economic status. The NHANES II study, using family income as a measure of socio-economic status, found that high blood lead levels were associated with low income levels (Mahaffey et al., 1982). In the United Kingdom, on the other hand, there is no clear, consistent trend in the blood lead-social class relationship in the population (Quinn, 1985). Children of manual workers have been found to have slightly higher blood lead levels than children of non-manual workers, but the differences are very small (around eight percent) (Quinn, 1985).

1.8 THE SOUTH AFRICAN SETTING

1.8.1 SOURCES OF LEAD EXPOSURE

In South Africa, lead was first discovered at the Maitland River in 1782 (Snodgrass, 1986). Lead deposits were exploited mainly in the Transvaal, near Argent station, where a lead smelter was erected in 1893. Between 1922 and 1925 about 360 tons of crude lead per month were produced. The Transvaal deposits were mined until 1957, by which time a total of 16 250 tons of metallic lead had been produced.

Today, South Africa produces four percent of the world's supply of lead and 53% of Africa's (Russell, 1986). The biggest deposits of lead occur at Aggeneys in Namaqualand, North Western Cape. In 1980, South Africa became a net exporter of lead for the first time. In 1984, 94 764 tons of lead were produced.

Table 1.2 gives the main uses of lead in South Africa. As can be seen, batteries account for about half the consumption. A wide range of chemicals are also produced from lead in South Africa, as are other lead-containing materials such as solder-bearing metals, lead foil, printers metal and ammunition (Snodgrass, 1986).

TABLE 1.2

CONSUMPTION OF LEAD IN SOUTH AFRICA - (SNODGRASS 1986)

Uses	Consumption kg	Percentage of total
Batteries	27.7	53.2
Cable sheathing	10.6	20.3
Chemicals	6.5	12.5
Lead sheet	2.5	4.8
Wire rope	1.6	3.1
Ammunition	1.2	2.3
Solder	1.0	1.9
Other	1.0	1.9
Total	52.1	100.0

Lead is used in numerous and varied products in South Africa. Lead-based paints were in use until relatively recently and are still used to a lesser degree today. There are no regulations or standards regarding the lead content of paint used in South Africa. A recent nation-wide survey, conducted by the Department of Health (1979), revealed that 20,5% of interior walls with leaded paintwork contained between five percent and 12,5% of acid leachable lead.

Until the early part of this century, lead plumbing was in general use in South Africa and cases of lead poisoning were reported in Cape Town. During the demolition of District Six, a suburb adjacent to central Cape Town, houses with old lead pipes were discovered (Retief-Steyn, 1976).

At the time of the present study (1983-1988), South Africa has one of the highest levels of lead in petrol in the Western world. During 1982, about 3 600 tons of lead were added to petrol (Scott and Stassen, 1983). The lead content in petrol has recently been reduced from 0.836 g/litre to 0.6 g/litre.

1.8.2 LEAD IN THE SOUTH AFRICAN ENVIRONMENT

Lead in the atmosphere is routinely monitored by the Council for Scientific and Industrial Research (CSIR) and by some local municipalities in South Africa. It is known that some South African cities have levels of lead in the atmosphere comparable to those found in large urban and industrial centres of the world. According to Wells et al. (1983), out of the 22 sampling sites operated for the Department of Health in South Africa, the lead concentrations at a particular site in Cape Town are elevated and have been increasing. For most of the other urban and industrial sites, there has been no marked trend upwards or downwards in air lead concentrations (Wells et al., 1983).

It is probable that the major source of atmospheric lead in the densely populated areas of South Africa is petrol derived (Scott and Stassen, 1983). In 1972 a study carried out in South Africa by Siegfried et al. revealed that lead levels in the bones of urban doves were approximately seven times more per unit weight of tissue than their rural counterparts. The authors concluded that this difference might reflect the relatively greater degree of lead in the atmosphere in urban areas, "presumably as a result of motor car exhaust emissions".

1.8.3 EPIDEMIOLOGY OF LEAD EXPOSURE IN SOUTH AFRICA

Whilst numerous studies on the impact of lead in the environment on human populations have been carried out in various parts of the world, in southern Africa little is known about the extent of lead absorption in the population at large.

Lead poisoning is a notifiable disease in this country. According to Davies (1985), no cases of lead poisoning were documented in 1984. He attributed this to the fact that doctors were either unaware of their legal obligations, were not diagnosing lead poisoning, or, alternatively, that conditions in industry have improved rapidly. (Lead is one of the ten most important metals in South African industry, according to the Erasmus Report of 1976). Surveys conducted by the National Centre for Occupational Health have indicated that lead exposure

in industry is commonly associated with excessive blood lead levels (Davies, 1985). In one survey, 87% of 110 workers employed in lead industries had blood lead levels above 40 ug/dl. The authors concluded that excessive exposure to lead is common in South Africa (Torrance et al., 1985).

Whilst it is known that workers may be exposed to a considerable degree to lead, there is little information about childhood lead exposure in South Africa. Between 1972 and 1981 only one case of lead poisoning among children was reported. In Cape Town, from 1970 to 1979, there were no reported cases among children. A few cases of lead poisoning in African children due to the burning of battery casings for fuel have nevertheless been reported in the literature (Harris, 1976). No cases of childhood lead poisoning from the ingestion of lead paint have been reported (Department of Health, 1979).

According to the Department of Health (1979), "... there is little doubt children with tendencies to pica in our country are in danger of being contaminated". It stated further that "... it is possible that we are now in the same position as New York prior to 1950, when cases of lead poisoning were simply not diagnosed by uninformed doctors and ailments were ascribed to more familiar causes" (Department of Health, 1979).

The first epidemiological study designed to determine the extent of increased lead absorption among children in parts of Cape Town

was carried out by von Schirnding in 1982 (von Schirnding 1982, von Schirnding and Fuggle, 1986a). In a lead screening study of 1 234 coloured first and second grade children attending schools in the Cape Peninsula, it was found that, among children from urban-industrial areas, there was an approximate two-fold increase in the prevalence of raised zinc-protoporphyrin (ZPP) and blood lead levels compared to children from suburban areas. This suggested that children from urban-industrial areas are at increased risk of exposure to lead.

Von Schirnding (1982) also examined the blood lead levels of children at two schools with the highest and lowest ZPP levels respectively (one situated in the urban area of Woodstock, close to the Central Business District of Cape Town, the other a control school in the semi-rural area of Hout Bay). It was found that blood lead levels at the urban school were on average twice as high (22 ug/dl) as those at the control school (11 ug/dl). 17% of children from the school in Woodstock had blood lead levels greater than or equal to 30 ug/dl; no children from Hout Bay had blood lead levels in this range. Pilot investigations suggested that there was no obvious lead source such as lead plumbing or water with a high lead content in the homes of these high risk children (von Schirnding, 1984).

In a preliminary study by White et al. (1982) of blood and tooth lead levels among clinic- and hospital-based Cape children, it was found that some children had accumulated lead to an extent

comparable to that in large Western cities. Blood lead levels were determined in 226 children who came from the Red Cross War Memorial Children's Hospital in Cape Town, from a hospital in a Ciskei township and from a dental clinic in Cape Town. Tooth lead determinations were also performed on these children. It was not stated how the children were selected for study. Children attending the dental clinic had higher mean blood lead levels than children attending the Red Cross War Memorial Hospital; the blood lead levels of these children were in turn similar to those of the hospital-based Ciskei children, although the Red Cross children were on average three years older (mean age: five years). No children had blood lead levels above 30 ug/dl. Only limited information on potential sources in nine high dentine lead children were mentioned in an addendum to the study.

In a subsequent study of tooth lead levels by van Wyk and Grobler (1983) in two groups of primary school children living in areas of heavy and light industry respectively, it was revealed that 27 children living in the vicinity of large industrial plants had higher tooth lead levels than 21 children living near light industries. No information on how the children were selected, nor of their respective ages was given, although all children were from primary schools in the areas.

1.9 RATIONALE FOR STUDY

The previous introduction and review of the topic has indicated that there are uncertainties regarding the risk factors for lead exposure among children. In South Africa in particular, very little is known about the nature of lead exposure, especially in children.

South African studies carried out up to 1983 point to the fact that increased lead absorption occurs among Cape children living in certain urban areas. No systematic attempt however, has been made to determine the risk factors for lead exposure in the children studied. There is virtually no information on sources of lead in the environments studied, nor is there adequate information regarding demographic and other characteristics associated with exposure to lead in the groups studied in this country.

The South African situation is of particular epidemiological interest with regard to the wide distribution of different socio-economic groupings among the coloured population living in inner city areas. Due to the Group Areas Act, and the resulting restriction on access to urban residential areas in particular, social stratification is not reflected in residential differentiation (Morris, 1980). Variation in socio-economic status may thus be considerable. This demographic feature has implications for the design of epidemiological studies, in

particular for investigating the relationship between social and environmental risk factors.

Preliminary data from the urban Woodstock area have suggested that the determinants of lead in this inner city community are complex. There is therefore a need to conduct more complete studies on an exclusively inner city population. The Woodstock area provides the opportunity to examine social and environmental risk factors in inner city school age children for whom there is a lack of information on exposure to lead. It was therefore decided to carry out a study on environmental lead exposure among children living in this area. This study relates primarily to coloured children, but as a significant number of white children were living in the study area, it was decided to include them in the study.

1.10 STUDY DESIGN

This study is concerned with risk factors for lead exposure among inner-city Cape Town children. Risk factors are factors associated with an increased probabilty of lead exposure. The presence of such an association however, does not necessarily imply causality.

The study was undertaken in two stages. First, a cross-sectional analytical study among first-grade school children was

undertaken. An environmental study was carried out simultaneously to determine sources of variation in environmental lead. This was followed, in stage two, by an in-depth case control study to determine sources of lead exposure in the home environments among children with high concentrations of lead in their blood (cases) as compared to children with low concentration levels (controls).

In the cross-sectional analytical study, blood lead levels were examined in relation to environmental and social risk factors, which were measured using a questionnaire. In addition, anthropometric surveys were carried out and the relation between lead absorption and nutritional status was examined. Measurements of haematological parameters, such as haemoglobin and haematocrit levels were also undertaken.

The cross-sectional analytical survey design was considered to be an appropriate design to use for investigating simultaneously the relationship between blood lead levels and risk factors in the study area. It would enable determination of the prevalence of increased blood lead levels in the area, and in addition, it would enable information on risk factors in a 'normal' school age population to be obtained. As there was little background information on the distribution of variables in the population, a feature of the study design was that it would permit the simultaneous investigation of different sources and mechanisms of exposure. Disadvantages of the study design were that 1) it was

not possible to ensure that equal numbers of exposed and unexposed children were present. This may have lessened the statistical efficiency, and 2) since exposures and outcomes were measured simultaneously, the directionality of the exposure-outcome relationship could not be confirmed.

In the environmental study, detailed studies of the distribution of lead in the environment were undertaken. The major sources of variation in environmental lead were determined through the establishment of a comprehensive air-monitoring network designed to examine spatial and temporal variations in lead levels. Indices of community-wide exposure to atmospheric lead were derived.

In stage two of the study, a subset of the children were selected for a case control study in order to determine whether cases (children with evidence of increased lead absorption) and controls (children with no evidence of excess lead absorption) differed in their exposure to identified sources of lead. Cases were defined as children with blood lead levels of 24 ug/dl or above. Controls were defined as children with blood lead levels of 14 ug/dl or below. Children with blood lead levels in the range 15 to 23 ug/dl were excluded from the study in order to differentiate between the two groups more clearly.

Detailed home investigations were conducted and information relating to sources for individual children obtained. Lead

concentrations in air, house dust, street dust, water and paint samples were obtained from the home environments of the children. Assessments of the condition and state of repair of the home were made, and behavioural characteristics of children relating to mechanisms of exposure were examined, with particular reference to the hand-mouth activities of children.

The aims and objectives of this work are described in the following section.

1.10.1 AIMS AND OBJECTIVES

1.10.1.1 (Cross Sectional Analytical Study)

1. To determine the blood lead distribution of first-grade school children living in the Woodstock area.

2. To identify risk factors associated with increased blood lead concentrations of children.

3. To determine mean concentration levels and sources of variation in environmental lead in the Woodstock area.

4. To identify children with high and low blood lead levels for in-depth case control study.

1.10.1.2 Case Control Study

1. To determine whether cases and controls differ in their exposure to identified sources of lead.

2. To determine possible mechanisms of exposure.

CHAPTER 2

PROFILE ON WOODSTOCK STUDY AREA

2.1 HISTORY OF WOODSTOCK

Woodstock is located in the 'twilight zone' of Cape Town's inner city. It comprises a variety of residential, commercial and light-industrial land uses, situtated some three to five kilometres East of the Cape Town Central Business District. The commercial areas illustrate a high degree of urbanity, with shops concentrated along the main roads, constituting a 'truly urban' space (Dewar and Uytenbogaardt 1978).

There is a wide range of housing types in existence; for instance, semi-detached houses, row houses, double storeys and flats. In many parts of Woodstock houses are considerably dilapidated and deteriorating. Woodstock is a racially mixed area comprising predominantly coloured (mixed race) and white groups. Whilst it is primarily a working class suburb, extremes of wealth exist and the community is diverse with respect to culture and religion.

In the following sections, an account is given of some of the major historical events which occurred in the area and which contributed to the present day character of Woodstock. This background information is necessary in order to understand the forces of urbanisation which have shaped Woodstock over time.

2.1.1 THE EARLY PERIOD (1652 - 1881)

Woodstock is the earliest and at one time most populous suburb of Cape Town. It is generally thought that Jan van Riebeeck set foot on Woodstock beach when he landed in South Africa in 1652. In 1780, the French line fortifications built to defend the Cape, ran through Woodstock. On the 10th January 1896, the signing of the Peace Treaty between the Dutch and the British took place in Woodstock. 'Treaty Tree', a 300 year old Milkwood, is today a national monument.

Woodstock was originally named Papendorp in 1788, after a Hollander Pieter van Papendorp, who arrived and settled at the Cape in the middle of the 18th century. In 1809 the name Papendorp was changed to Woodstock, after a favourite fisherman's inn in the area. Pieter van Papendorp registered a house and erf situated between the castle and Salt River (Pickard, 1969) and a line of houses soon spread next to his estate along what was then a dusty main road. This main road was probably one of the major attractions for the settlement in the area.

Although the history of Woodstock is very closely linked to the development of central Cape Town, in the early 1800s the area was predominantly rural and had a way of life distinct from that of the city. In 1845 Woodstock had an English Church and school; in 1876 there was a Dutch-Reformed Church and in 1877 a market was established. Woodstock was a favourite seaside resort in the 1870s.

A report in 1871 noted that Papendorp was "the refuge of the utterly destitute who eke out their means of livelihood by amassing together shells on the beach to sell to lime burners ... there is one small tannery here, a few people are engaged in fishing" (Badham, 1987).

In 1887, Bishop West Jones described Woodstock as

> "a very dreary suburb of Cape Town along the shore of Table Bay, with a population mainly of fishermen and other poor folk, many of them Malays, and nearly all living as heathens. There were a few, but a very few, of a better class"

(Ridd, 1981). The Malay population, according to Badham (1987), was actually relatively small, but large enough for provision to be made for the establishment of St Mary's Malay mission. The rector of St Mary's reported that "the moral tone of the village is fearfully low" (Badham, 1987).

Up until the 1880s, residents were concentrated along the coast and depended mainly on fishing for their livelihood. From the 1860s on, the construction of a railway line provided an important source of employment. According to Fagan (1986), movement through Woodstock between the original settlement in Cape Town and the hinterland, was a major influence on the development of the area, particularly after the building of the railway line. In 1881 there was widespread selling of property in the area, which was advertised in 1881 as a "rapidly rising hamlet" (Badham, 1987).

2.1.2 TURN OF THE CENTURY WOODSTOCK (1882 - 1913)

Woodstock was a popular residential town and by 1882, together with Salt River, became a separate municipality.

By 1897 Woodstock was the second largest municipality after Cape Town. Towards the end of the century, with the discovery of diamonds and gold, Cape Town became important as a commercial centre. Woodstock was developing and urbanising, as traffic to and from Cape Town passed through it. Two main lines of transportation were the railway line and the electric tram service which ran to and from the suburbs and Cape Town.

In the early 1900s, many people, local and immigrant, flocked to Cape Town in search of jobs. Woodstock, accessible and offering cheap accommodation, was the ideal place to settle. The poorest section of workers were attracted there, as it was the closest southern suburb to the city (Pickard, 1969). The growth in the population of Woodstock created a demand for goods and services in the immediate vicinity. Commercial enterprises started to develop in Woodstock which brought about a gradual change to the character of the area. The inhabitants were mainly artisans, involved in tannery, smithing and masonry (Badham, 1987).

By the turn of the century, nearly all the lower estates had been subdivided and developed, mainly with row housing or semi-detached houses, with typical commercial enterprises along the main roads (Fagan, 1986). In 1902, a journalist in the Cape described Woodstock as "a town of factories, of workmen's houses, of middle class shops and rival church spires ... [with] ugly and irreverent surroundings". He writes of Albert Road (one of the main through roads in the area) with "its uneven and shabby houses, its dusty little huckster shops and its occasional waste spaces strewn with rubbish" (in Badham, 1987).

The rapid urbanisation during this period is apparent in various maps of 1891 to 1905, and was stimulated also by the Anglo-Boer War (Fagan, 1986). According to Fagan, "the urbanisation of Woodstock as reflected by the growth of the road system over this short period is phenomenal" (Fagan, 1986).

There were many immigrants, mainly in the early part of the 20th century, and in 1904, a quarter of the Woodstock population originated from Britain (Ridd, 1981). During the 19th century, the population had had a larger Dutch-speaking sector. The Peninsula Commission of 1902 noted the very high percentage increase in population density in Woodstock. The total percentage increase was the highest of the suburbs, particularly in the European and coloured sectors (Badham, 1987; Ridd, 1981). Between 1891 and 1904, the population increased nearly six-fold from 4 974 in 1891 to 28 990 in 1904.

Woodstock's growth during this period and following years is apparent also from council records, which indicate that the growing municipality experienced all the problems of an increasingly urban area, vis. drainage problems, factory odours, noise and sewerage. In general at the turn of the century there was much overcrowding and unsanitary conditions prevailed. Housing was highlighted as a serious problem (Badham, 1987).

In 1903 several large factories were established in Woodstock during the building boom. In 1912 there were 19 industrial concerns (Whittingdale, 1973). These industrial activities contributed to the urban character of Woodstock.

In 1913, the municipal area of Woodstock joined with other municipal areas to form the Municipality of Greater Cape Town.

2.1.3 TWENTIETH CENTURY WOODSTOCK (1914 - 1979)

During the 1920s, the expansion of the railways and harbour transportation systems gradually reduced Woodstock's access to the beach. The last of the beach disappeared in the early 1970s with the construction of the extension to Table Bay harbour.

During the 1920s and 1930s, there was an influx of poor whites into Woodstock, who were mainly Afrikaans-speaking, from the rural areas. The 1932 Carnegie Commission of Inquiry into Conditions Amongst Poor Whites of South Africa, named Woodstock as an area to which people came, especially during the Depression years (Ridd, 1981).

The area became predominantly Afrikaans speaking, as English speakers moved out. During the 1930s, Greek and Italian immigrants moved in, who in turn were replaced by Portuguese in the 1960s, initially from Madeira and later from Mozambique and Angola. A part of Woodstock was nicknamed 'Little Madeira'. Both upper and lower Woodstock still house Portuguese families but many have subsequently left. Despite the arrival and departure of certain immigrant groups, much of Woodstock houses established white and coloured families who have been living there for many decades. Surveys in the area confirm this (Garside, 1987).

2.1.3.1 Group Areas Act

Woodstock has historically been a racially mixed area, comprising predominantly white and coloured (mixed race) groups. It is the largest so-called 'grey' area in Cape Town, which has managed, to a certain degree, to stay 'open' despite government pressure to implement the Group Areas Act. This Act, instigated in 1950, segregated residential areas in South Africa along racial and ethnic lines. In practice this meant that in Cape Town, most of the coloured population was relocated from the inner city areas of the city to residential areas on the periphery. Most of the removals occurred in the 1960s and 1970s and affected radically the social geography of Cape Town, initially one of South Africa's most integrated cities (Dewar and Uytenbogaardt, 1978).

One of the most extreme examples of the effects of the Group Areas Act was the case of District Six, a suburb bordering Woodstock on the perimeter of central Cape Town. It was declared white in 1966, at which time there was a community comprising approximately 31 000 coloured people, 15 000 Asians and 400 whites (Dewar and Uytenbogaardt, 1978). All of the properties, mainly white owned, were expropriated and demolished, and the residents rehoused on the periphery of the city.

Some ex-District Six residents moved into neighbouring Woodstock, a portion of which was proclaimed white in 1958, and another portion coloured in 1975, as, according to Ridd (1981), compensation to the coloureds for the loss of District Six. Woodstock was essentially divided into East for whites and West for coloureds. Pressure on accommodation in the coloured areas made rents appreciably higher than in the neighbouring white areas (Ridd, 1981). In 1979, part of lower Woodstock was reproclaimed as a coloured area, and the rest industrial or undetermined.

2.2 PRESENT DAY WOODSTOCK

2.2.1 SOCIAL CHARACTERISTICS

According to the 1985 census, the population of Woodstock comprised 15 165 persons in 1985. Eight percent of the population is aged four years or below, whilst 16% of the population is between the ages of five and 14 years. The majority of the population (60%) is coloured, the rest is predominantly white, with a low percentage of 'Asians' and blacks living there (less than five percent of the population).

Today, although segregationist measures have been put into practice, these have not been totally successful and some assert that in nearly all areas of Woodstock racial mixing takes place

(Cape Town Chamber of Commerce, 1986). Woodstock can be seen as a mixture of declared and controlled areas, some white, some coloured, some grey, and in this respect is almost unique in South Africa (Figure 2.1). In 1986, the government proposed that a white area of lower Woodstock, which is in reality mixed, be declared a coloured area. This proposal was vehemently opposed by the community of Woodstock, who organised a grassroots campaign to have the area declared open. So far, it appears that this campaign in defiance of the Group Areas Act has been successful (Newsweek, 1987; Time, 1987; Councillor Parkin, pers. comm.).

The racially mixed Woodstock community comprises extremes of wealth (although it is primarily a working class suburb), particularly with respect to the coloured population, among whom there are also many differing religions. The coloured people comprise a Cape coloured group which is predominantly Christian and a Cape Malay group which is predominantly Muslim. They are distinguished from Indian Muslims who live in separate group areas. The Muslim members of the community have moved into the area in increasing numbers in recent years. Today they comprise well over 50% of the coloured population there.

Social divisions in the community are based on colour, religion and economic status (Ridd, 1981). The exclusive and distinctive Muslim community distinguish themself from the more heterogeneous Cape coloured people on the basis of their religion and cultural

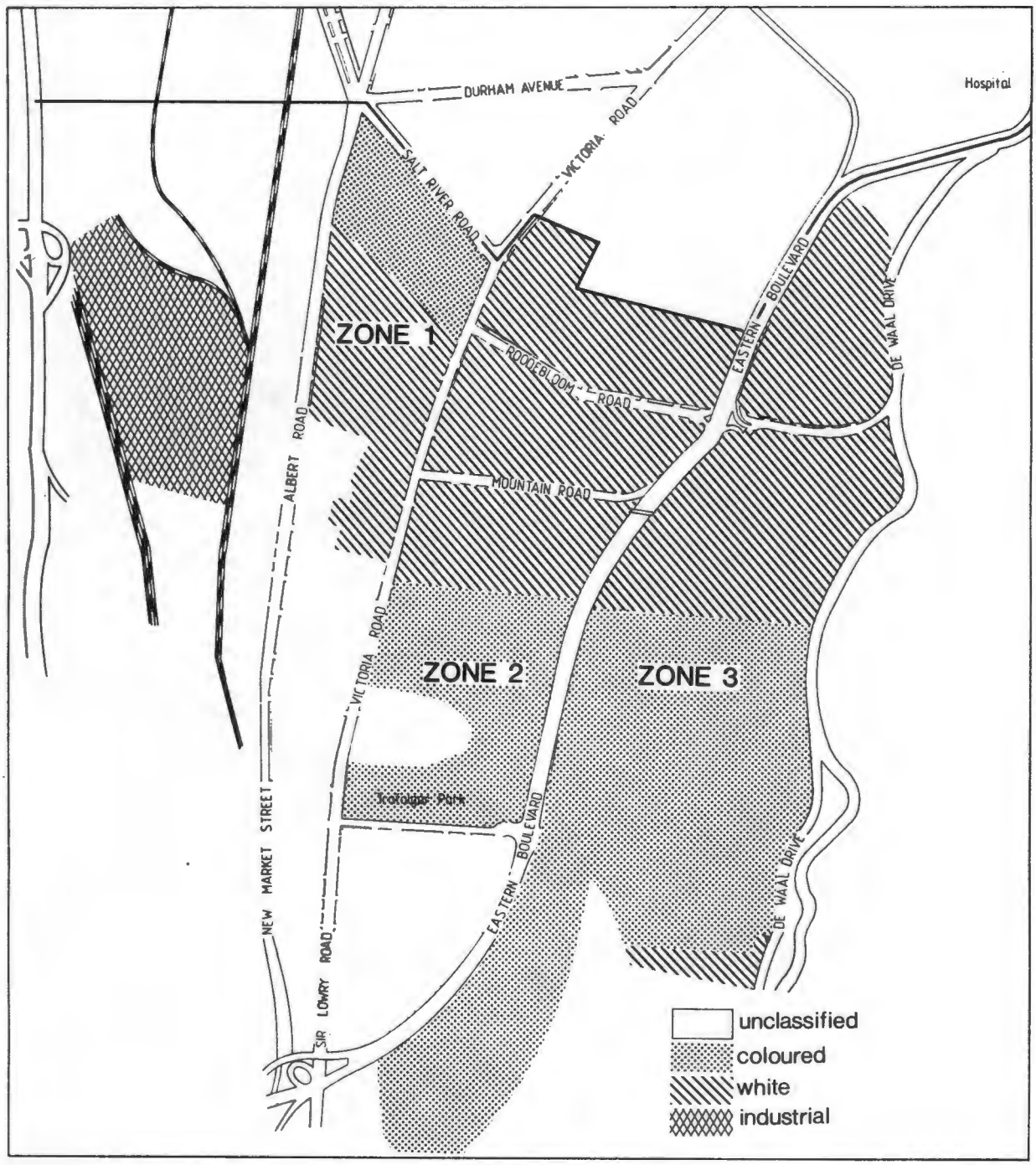


FIGURE 2.1

RESIDENTIAL BOUNDARIES FOR POPULATION GROUPS, WOODSTOCK

practices (Ridd, 1981). They have a strong sense of 'belonging' and community. In general, among the coloured people, socio-economic status is expressed in terms of religion and language. Afrikaans is more widely spoken but is accorded lower status than English. The Muslim community is of higher socio-economic standing than the Christian Cape coloured group (Ridd, 1981).

According to the 1985 census, few residents occupy professional positions and most are skilled or semi-skilled workers. About three percent of the economically active population is unskilled.

2.2.2 PHYSICAL CHARACTERISTICS

Woodstock accommodates a wide variety of land-uses, including residential, commercial and industrial (Figure 2.2). A computer search carried out by the Cape Town City Council (Technical Management Services) revealed that the industry in the area is mainly light industrial in nature, with a large proportion of textile and garment manufacturers being located in the area. There are no major lead-emitting factories in the area. Residential areas border the commercial strips, whilst the more space-intensive commercial and manufacturing concerns are located in other areas. In certain instances however, light industrial concerns intrude into residential areas.

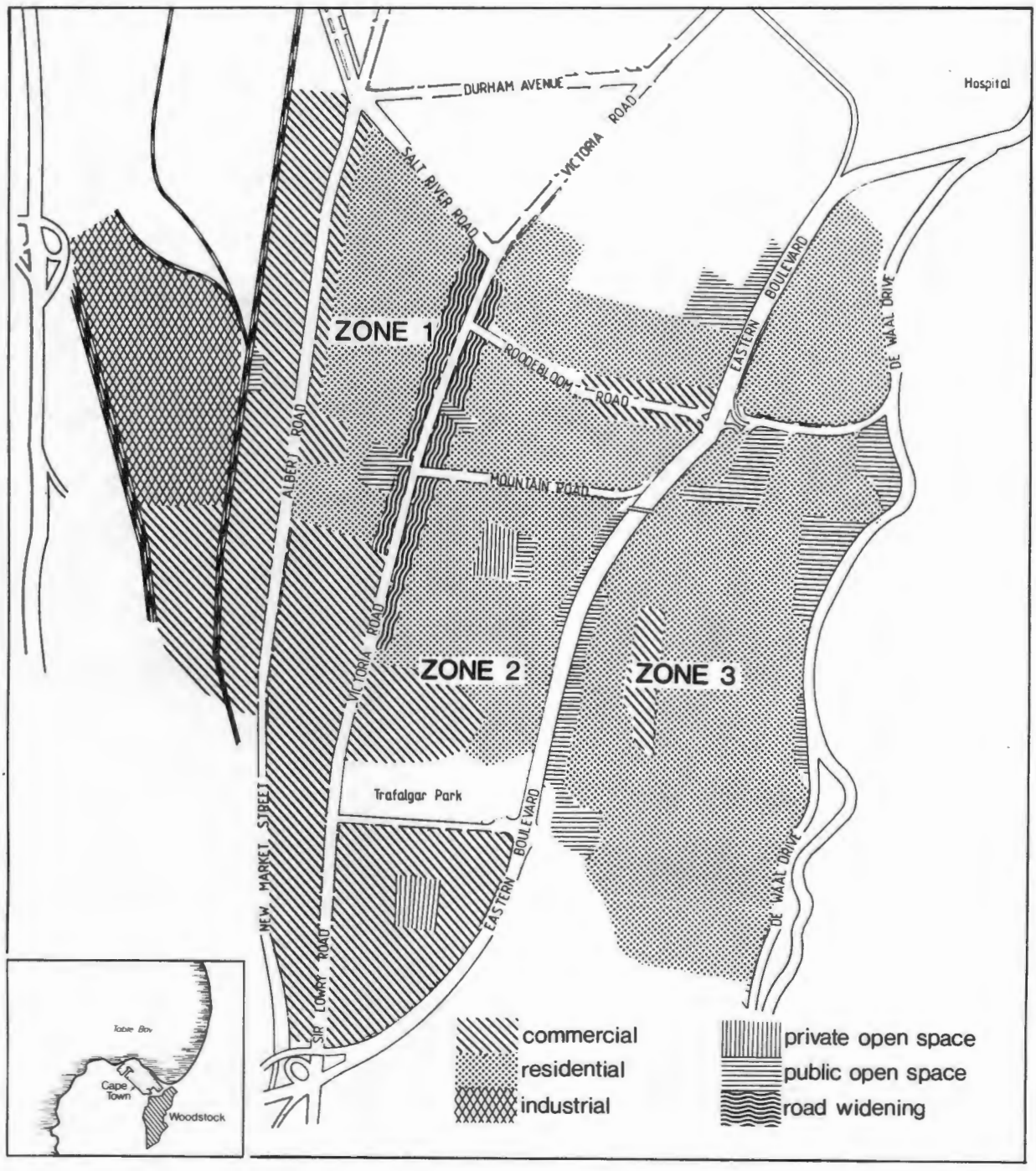


FIGURE 2.2

LAND-USE PATTERNS IN WOODSTOCK

2.2.2.1 Transport Corridors

Major through routes dominate the area, and carry heavy loads of traffic between the outlying areas and the city centre. They derive support from both local residential areas and passing traffic. Most traffic to and from the Central Business District passes through Woodstock. As most of the high intensity activity and traffic is on the major routes, pressure is released from other areas of the gridiron (Dewar and Uytenbogaardt, 1978).

The major through routes comprise two highways and two major main roads. The Eastern Boulevard highway acts as an element of division in the area, cutting Woodstock effectively in two. It carries a large percentage of traffic, whilst the De Waal Drive highway, an older route designed for slower speeds, acts as a supplement to the Eastern Boulevard. Before the construction of these highways, Main Road/Victoria Road was the main through route in the area. It is highly accessible and attracts activities of a local and metropolitan scale. Albert Road is similar, but less congested and has activities of a different nature; for instance, wholesaling-type commerce relating to the secondary sector.

2.2.2.2 Housing

"Woodstock is dying of neglect" wrote Pama in 1979 (in Badham, 1987). Almost ten years later, this is still largely the situation in Woodstock. The Group Areas Act has caused uncertainty and insecurity through the constant threat of forced removal. This could account in part for the apparent reluctance on the part of the developers and home-owners to upgrade buildings in the area (Cape Town Chamber of Commerce, 1986).

There is a wide range of housing types in existence; for instance, semi-detached houses, row houses, double-storeys and flats. As one moves up the mountain, residences are not as densely packed and increase in diversity and size. They are also not as dilapidated as working-class houses in lower Woodstock. However, pockets of poverty exist, such as the area just above the Eastern Boulevard highway, especially on the Western side, which is very dilapidated (Ridd, 1981).

Throughout Woodstock, private outside space is very limited. Back and front yards, if they exist at all, are very small, with back yards usually serving as storage areas. Family life spills out into public space and the street is used as a social place, where people meet and children play. This contributes to the character of Woodstock (Dewar and Uytenbogaardt, 1978).

2.3 SUMMARY

This chapter has outlined the history, social parameters and land-uses which characterise the inner city Cape Town suburb in which this study of childhood lead exposure took place. Important elements are the age of the suburb and its associated housing, the major transportation corridors which pass through it, the diversity of ethnic and cultural groups that continue to live in the suburb, and the currently diverse socio-economic status of its inhabitants. These elements are discussed in greater detail where appropriate, in the following chapters.

CHAPTER 3

METHODS

In this chapter the methods used in the study are described. Firstly the methods pertaining to the cross-sectional analytical study are discussed. These include the blood measurements, the anthropometric measurements, the questionnaire data, statistical analyses and the environmental lead measurements made in the environmental study. Next the methods used in the case control study are discussed. These include the dust, paint, water and air samples collected, the environmental assessments made during the walkabouts relating to conditions in the home, the home interviews, assessment of nutritional status, repeat blood samples and statistical analyses.

PART A :· CROSS SECTIONAL ANALYTICAL STUDY

3.1 STUDY POPULATION

Schools formed the primary sampling units, and selection of pupils within schools was restricted to one age group to eliminate age bias. As younger children are generally considered to be at greater risk than older children, it was decided that the study population be defined as first grade

pupils (aged six to seven years) living and attending schools in the greater Woodstock area. Preliminary investigations, based on lists of addresses of children obtained from principals of schools in the area, revealed that this target population consisted of approximately 200 children. Addresses of children from schools in the immediate surrounding areas, Salt River and central Cape Town, were also obtained; these revealed that an insignificant number of children from the study area (less than five percent) attended schools outside the greater Woodstock area. As the target population was small enough to be studied using available resources, the entire population was included in the study.

All principals of primary schools with pupils living in the area gave permission for children to be tested at school. Schools tested comprised five coloured and two white schools. A form telling parents about the study (translated into English and Afrikaans) and requesting written consent for their child to be tested, was sent home with all children. Parents were assured of the confidentiality of results and were given the option of knowing the test results. Provision was made for the address of the child to be written on the form (see Appendix A). All first grade children with parental permission, who lived and attended school in the study area, were eligible for inclusion into the study.

3.2 BLOOD MEASUREMENTS

Several biological indices of lead absorption exist, the most widely used index of current lead absorption being the blood lead measurement. This measurement reflects the previous one to two months exposure. Hair and tooth lead measurements are also used, but these reflect exposure over a longer period of a person's life-time. Other indices of a biological effect due to lead are also used. These include the detection of abnormalities of haem synthesis, such as measurement of the activity of the enzyme delta-aminolaevulinic acid dehydratase. It was decided to use the blood lead measurement as the primary test for lead absorption. This index is the most useful and authoritative index of lead toxicity, being the most reliable and clinically relevant (David et al., 1982). In addition, other haematological tests including haemoglobin and haematocrit determinations were performed on samples.

3.2.1 COLLECTION OF BLOOD SAMPLES

Following parental permission, given by 90% of parents of the study population, five ml of blood were obtained by venipuncture from each child who was present at school on the day of the study. Children not wishing to comply (less than five percent) were not sampled. To reduce the chance of lead contamination, all syringes, needles and tubes used for blood sampling were lead

free. A sufficient volume of blood was collected at the first visit to enable all the necessary analyses to be carried out at a later stage. Four ml of blood from each child were stored in tubes containing heparin anti-coagulant and kept on ice until such time as they could be delivered to the Red Cross Children's Hospital for storage under deep freeze conditions. Duplicate blood samples were obtained from 20% of the population in order to measure intra- and inter-laboratory variation. A further one ml blood sample was stored in tubes containing EDTA anti-coagulant and delivered to the Red Cross Children's Hospital for full blood count determinations. All blood samples were collected during the months May 1983 to June 1983.

3.2.2 PREPARATION AND ANALYSIS OF SAMPLES

3.2.2.1 Blood Lead Analyses

Precautions to avoid contamination were stringent. Every effort was made to avoid undue exposure to contaminated water, chemical reagents or by contacting or touching specimens. In this way it was hoped to reduce intra-laboratory variation. 0.7 ml Triton X-100 (10% v/v) was added to samples, and lead in blood samples, standards and blanks was concentrated by chelation with a solution of ammonium pyrrolidine dithiocarbamate (two percent w/v). The lead complex formed was then extracted into approximately one ml water saturated methyl isobutyl ketone

(MIBK). Following centrifugation, an atomic absorption spectrophotometer (Beckman 1272, M Model) was used to perform lead analyses. With each batch of samples a reagent blank and set of working standards were run simultaneously. The lowest accurately measurable concentration of lead in blood was found to be five ug/dl. This was determined by measuring known concentrations at these levels - lower concentrations were not readily differentiated from the blank.

QUALITY CONTROL

Routine blood lead measurements on samples were performed by the Institute of Child Health Laboratory, Red Cross Children's Hospital, Cape Town, and samples for quality control purposes were measured by the State Chemistry Laboratory, Cape Town. Both these laboratories participate in the national quality control programme which enables laboratories to assess the accuracy of blood lead determinations. The procedure involves the monthly collection of fresh ox blood to which known amounts of lead are added and mixed. The blood is then irradiated, sent to participating laboratories for analysis, and evaluated by the National Centre for Occupational Health (Röllin et al., 1988). Acceptable comparative measurements (within experimental error) are obtained (White et al., 1982). For both intra- and inter-laboratory variation, the mean difference, $\pm$ approximately two standard deviations, was within five percent of the mean value. In addition, there was no evidence of a systematic error; i.e.

the difference between duplicate values was not related to the mean of the duplicate values.

3.2.2.2 Other Haematological Tests

A Coulter Counter, Model S, was used to perform other haematological tests such as the determination of haemoglobin and haematocrit levels.

3.3 ANTHROPOMETRIC MEASUREMENTS

In order to obtain an objective measure of nutritional status, height and weight measurements were performed on all children who underwent blood lead tests. This took place at schools after the collection of blood samples.

3.3.1 WEIGHT

A standard bathroom scale, recommended by the World Health Organisation for measurements of children aged six to ten years (WHO, 1985), was used for all weight determinations. Readings were taken to the nearest 100 grams. In order to minimise the possibility of observer bias, all measurements were performed by one observer. The measuring scale was regularly calibrated and

the validity of measurements assessed using standard weights. To test for intra-observer reliability, a class of children at one school was weighed twice.

3.3.2 HEIGHT

All height measurements were performed in accordance with the recommended measures by the World Health Organisation. Measurements were obtained with children standing upright on a flat surface, shoes removed, with feet parallel and heels, buttocks, shoulders and back of head touching the measuring device. All measurements were obtained by one observer, to the nearest tenth of a centimeter. Repeat measurements were obtained on a class of children to assess intra-observer reliability.

3.3.3 REFERENCE POPULATION

The three measures, age, weight and height, formed the basis for the determination of the three indicators of nutritional status: weight for age (indicative of emaciation), height for age (indicative of stunting) and weight for height. These indicators were compared with the distribution of indicators in an international reference population of well-nourished healthy children, established by the United States National Centre for

Health Statistics (NCHS) and recommended by the World Health Organisation as a standard reference population (WHO, 1985). The weight and height of each child were expressed as a percentage of his/her 'expected' (age-standardised) weight or height, using the NCHS median weight or height as the reference standard.

3.4 QUESTIONNAIRE DATA

In order to identify particular risk factors which could have a bearing on the community's exposure to lead, a questionnaire covering a broad range of items relating to sources and mechanisms of exposure, socio-demographic factors, as well as medical factors was designed. Some items contained in the questionnaire were based on those contained in the questionnaire devised by Garnys et al. (1979).

3.4.1 STRUCTURE OF QUESTIONNAIRE

The initial draft for the questionnaire was drawn up after informal exploratory home interviews had been conducted on a sample of ten parents of children not included in the survey. The final questionnaire (Appendix C) was drawn up after pretests to ensure that the questionnaire was comprehensible and unambiguous. Colleagues, principals, teachers and experts in

questionnaire design were asked to determine how well the questionnaire achieved its objectives and to comment on potential biases.

In order to reduce the possibility of language bias, the questionnaire was translated into English, Afrikaans and Portuguese. Language bias was thought to be potentially significant at the white schools in particular: some children were from migrant families whose home language was Portuguese. Although face to face interviews with parents may have been preferable, resources were not available for this purpose and the questionnaire was sent home with the child to be completed by the parents (normally the mother) of the child. Teachers, in consultation with the children, decided on the most appropriate language medium for the questionnaire taken home.

For the purpose of answering and coding the questionnaire, a multiple choice format was adopted in which the appropriate square (or squares) could be marked. The majority of questions required simple "Yes" or "No" answers. In addition, some open-ended questions were included.

The questionnaire contained items of interest which fell into three main content areas: questions relating to sources and mechanisms of exposure to lead, questions relating to socio-demographic and economic factors, and questions relating to

potential ill-effects associated with exposure. These are briefly discussed below.

3.4.1.1 Sources and Mechanisms of Exposure

Questions relating to the nature and location of the General Activities of the child, which could have a bearing on exposure, included the following: the school attended (QA9), the nature of transport taken to and from school (walking or cycling vs transport by car, bus or train) (QA10), the nature of the traffic on the roads travelled by the child (QA14), the child's main play areas (inside, outside the home, busy streets, other homes) (QA15-A18).

Questions relating to Pica (the purposeful eating of non-food items) included the presence or absence of pica for items such as paint, cement, plaster, soil/earth, matchsticks and other individual items (QA28). Also included were questions that related to the age of onset and duration of pica (QA29-A32).

Questions relating to Housing included the age of the house (QC4), fuel source (QC7), ventilation (QC8-C9), smokers (QC10-C11), level of dustiness (QC12), type of water pipes (QC14-C17), presence of flaking paint inside and outside the house (QC18-C21), state of repair of the home (QC22), presence of factories, petrol garages, workshops near to the house (QC24-Q30).

Questions relating to the child's Dietary Composition included the frequency of various food items consumed such as tinned foods, meat, fish, vegetables, dairy products, cereals and other individual items (QB1-B2). Also included were questions relating to the type of pots used for cooking and storing food (QB6-B7).

3.4.1.2 Socio-Demographic Factors

Questions about Socio-Demographic, Cultural and Economic Factors included data that related to the child's home language (QA8), religion (QB3), ethnic group (indicated by the school attended by the child) (QA9), family composition (for example, the total number of people (QD1), children (QD2), siblings (QD3), parents (QD6) living in the home), home ownership (QC1), occupational density (defined as the number of people in relation to the number of people allowed for at optimum living density, taking into account the number of rooms in the house) (Batson, 1944) (QC6, D1-D2), mother's and father's jobs, education levels, incomes (QD7-D12), total family income (QD13), number of dependants (QD14-D15), rent (QD16) and others.

3.4.1.3 Medical History

Questions about Behavioural and Medical Factors associated with lead exposure included data relating to adverse behavioural characteristics such as hyperactivity (QA21-A27) and the nature of the child's school performance (QA11-A13). Questions relating to medical history potentially associated with excessive lead absorption included data referring to hearing, speech, eyesight problems, anaemia, fits, kidney problems and others (QA36). Questions relating to non-specific symptoms included items referring to clumsiness, tiredness, loss of appetite, loss of weight and others (QA37).

3.5 STATISTICAL ANALYSES

Univariate analyses were carried out to examine the relationship between individual variables and blood lead concentrations, haematological parameters or nutritional status. For the discrete variables, if the distribution of blood lead levels, haematological or nutritional parameters was approximately normal for the two or more categories of the variable, a t-test (for two categories) or a one way analysis of variance (for more than two categories) was performed. If the distribution of blood lead levels was skewed but the variances for the categories were not significantly different, a Mann-Whitney U test (for two categories) or a Kruskal-Wallis test (for more than two

categories) was performed. If the variances were significantly different, a median test was performed.

To test for correlations between continuous variables and the various parameters, Pearson and Spearman correlation coefficients (where normality could not be assumed) were determined. For questionnaire data, Spearman correlation coefficients were determined for continuous variables, to see if an association with blood lead levels existed.

Due to the large number of statistical tests which were carried out on the questionnaire data, it is recognised that some positive associations may have arisen by chance. For this reason the "p" value was used as a guide to detect important variables, with the magnitude of the difference also being considered.

In addition for coloured children, multivariate analyses on questionnaire data were carried out. A series of multi-way contingency tables were formed and log linear models used to analyse them (Dixon, 1981). Additional details relating to statistical tests carried out are given in the text.

BOX PLOTS

Box plots were used as summary displays of blood lead distributions and distributions of other continuous variables. In the box plot the upper and lower quartiles of the data are

displayed by the top and bottom of a rectangle (which represents the central 50% of the data). The median is exhibited by a horizontal line in the rectangle. The length of the vertical lines relative to the box show how spread out the tails of the distribution are (Chambers et al., 1983).

PART B : ENVIRONMENTAL STUDY

Whilst there is considerable information on long-term trends in atmospheric lead at several sites in South Africa, there is little information on the spatial distribution of lead in the local environment.

In order to obtain a reliable measure of community-wide air quality in the urban area of Woodstock (no routine environmental monitoring is carried out in this area), it was decided to establish a comprehensive air-monitoring network in the area, to take into account both the spatial and short-term temporal distribution of atmospheric lead. In addition, it was also decided to carry out dust sampling in the area. In this way it was hoped to obtain a better understanding of mean concentration levels and sources of variation in environmental lead.

The following issues were addressed:

1. The spatial variation in atmospheric lead levels.
2. The temporal variation in atmospheric lead levels.

3. The relationship of atmospheric lead levels to United States of America air quality standards.
4. The spatial variation in dust lead levels and the relationship between dust and atmospheric lead concentrations.
5. The role of potential influencing factors, such as traffic, on environmental lead levels.

3.6. ATMOSPHERIC LEAD MEASUREMENTS

Three factors governed the air-monitoring strategy devised for the study area: first, it was decided that sampling for lead in the area should be related to the population at risk, namely, young children. Secondly, it was necessary for sites to be both secure and accessible, as well as standardised in terms of position from traffic both horizontally and vertically. Finally, it was important that the air-monitoring strategy be suited to the particular circumstances under investigation but also be comparable with other local and international air-monitoring strategies.

3.6.1 SELECTION OF SITES

The Woodstock area is primarily an urban residential area: there are some small light industries and it is traversed by two

highways and two heavily travelled through-roads. Three zones bounded by major highways or through-roads were distinguished (see Figure 3.1). Zones one and two are characterised by a variety of different land-uses, including space intensive commercial and manufacturing concerns such as shops, factories, garages and service industries. There is no dominant source of industrial lead emissions in the area. The character of zones one and two contrasts with that of zone three, which is primarily residential, with few industrial or commercial activities and a less dense roads' network.

Two air-monitors were placed in each of zones one and two and one in zone three. Monitors were placed 200 to 300 metres from the boundary roads, with two additional monitors placed directly on major through-roads with high density traffic. The positions of the air-monitors in the study area are given in Figure 3.1. Contact meetings with members of the community were held in the area and few problems were encountered in obtaining suitable sites to house air-monitors. Private homes, schools and shops in the study area were used as bases. Sampling points were at a height of 1,5 to two metres from the curb. In certain instances where a monitor could not be maintained at a particular site for the entire study period, an alternative site close to the original and within 150 metres (maximum distance a few houses away) was used to continue monitoring the sector concerned. In addition, for a period of three weeks only, air pumps were

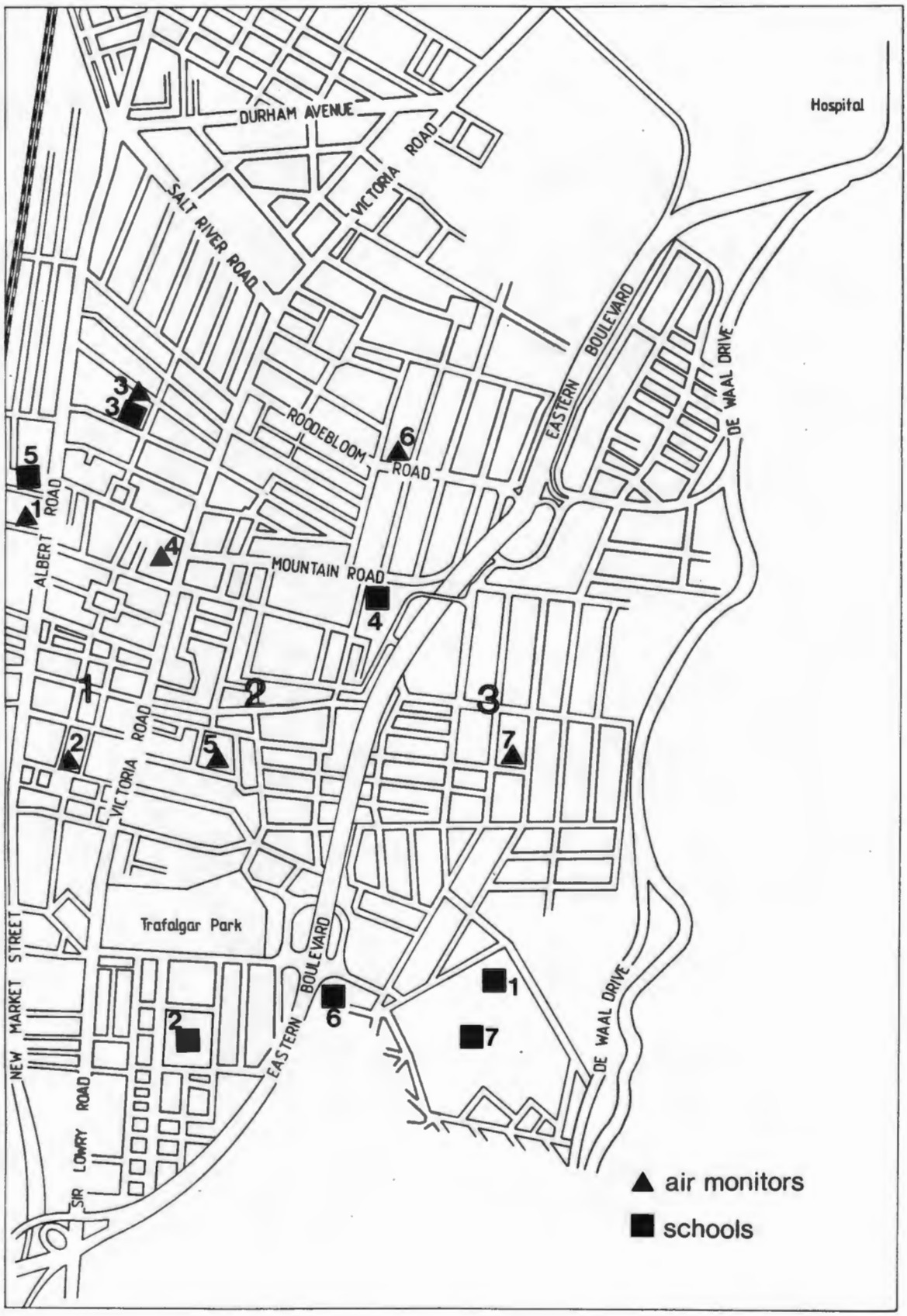


FIGURE 3.1

LOCATION OF AIR MONITORS AND SCHOOLS IN THE STUDY AREA

installed at coloured schools in the study area. Permission was not obtained in time to monitor air quality at white schools.

3.6.2 COLLECTION OF AIR SAMPLES

Air samples were collected through window-outlets or air vents at air-monitoring sites for a period of approximately one year. The sampling interval was generally 24 hours, but access to some sites on a regular 24 hour basis was not possible, for instance at sites with limited accessibility over weekends and holidays. Consequently, some observations are representative of longer intervals. In all, around 2 000 air lead samples were obtained from May 1983 to June 1984.

Suspended particulates were precipitated on cellulose membrane Millipore filters, 37 mm in diameter, with a pore size of 0.45 um. Filters were mounted on supporting pads in three-piece polystyrene filter holders. The exit end of the filter unit was connected to an air pump (Charles Austin Dymax 2A). The filtration unit was placed with the inlet facing downwards, in a funnel for protection from wind and rain. Samples were taken at a flow rate of 1.5 to two litres per minute yielding a total air volume of approximately two cubic metres per sample. Dry gas meters (Gallus and Remus) were utilised to measure daily air volumes. Air pumps were regularly checked for leaks using standard flow meters. Lead-free plastic tweezers were used for

the handling of filters and all samples were stored in tightly sealed Millipore disposable petri dishes.

3.6.3 PREPARATION AND ANALYSIS OF AIR SAMPLES

The method adopted for the preparation of air filter samples was a modified version of that used by the South African Council for Scientific and Industrial Research (C.S.I.R.) for the analysis of trace elements in airborne particulate matter. Samples were placed in clean 100 ml Pyrex beakers, and five ml concentrated nitric acid (Merck Reagent Grade) added to each. Beakers were then placed on a ceramic topped hotplate and gently heated until the volume was reduced to approximately 0.5 ml. Five ml Perhydrol (Merck Reagent Grade) were subsequently added to each sample and heated again until 0.5 ml of acid was left. This volume was then transferred to 10 ml volumetric flasks by repeated rinsing with 0.25 N nitric acid.

Between each use, glassware was soaked in a three percent `Contrad' solution for 18 hours, and in a 10% solution of nitric acid for 18 hours. Glassware was then repeatedly rinsed with double and triple distilled water.

A Perkin-Elmer model 5000 atomic absorption spectrophotometer, set at the radiation intensity maximum of 283 nm, was used to perform lead analyses. Samples were aspirated directly into the

flame and the absorbance recorded. With each batch of seven samples, a filter blank, reagent blank and set of working standards were run simultaneously. Three replicate readings (each an average of five readings) were obtained for each sample.

The detection limit for lead by this method was found to be 0.04 ug/ml. The precision of the method is five percent. For quality control purposes, a series of filter paper samples representing a range of different lead concentrations was cut into sections using a specially constructed lead-free cutting device; one section was sent to the C.S.I.R. Laboratories (Atmospheric Sciences Division) for analysis and the other was analysed in the University of Cape Town Spectroscopy Laboratory (Department of Analytical Science). Results revealed that differences between the two laboratories were within experimental error, i.e. the mean difference, $\pm$ approximately two standard deviations, was within five percent of the mean value.

The preparation and analyses of all samples were performed without the analyst having had prior knowledge of the nature of the site from where the sample was collected. Samples were analysed in a random order, without regard to site or day of collection.

3.6.4 DETERMINATION OF TRAFFIC VOLUMES

Traffic counts were taken over a period of two weeks (excluding public holidays) at each site (with one exception) using portable automated traffic counters. It was not possible to obtain counts from Site two due to problems which arose from interferences with equipment placed in this area.

3.7 DUST LEAD MEASUREMENTS

Dust lead measurements were obtained in the study area and spatial variations determined as well as the relationship of dust lead levels to traffic density and air lead levels.

3.7.1 SELECTION OF SITES

Dust samples were collected from each of the air-monitoring sites in the study area. The location of the sites is given in Figure 3.1.

3.7.2 COLLECTION OF DUST SAMPLES

There are no standardised procedures for the sampling of dust but samples are usually collected by sweeping, wiping or vacuuming a

standard area. Results are normally expressed in terms of concentration (ppm) but are sometimes also expressed in terms of mass-loading, for example, ug/unit area. This index represents the total lead per unit area. Samples were obtained at two-monthly intervals during May 1983 to June 1984, by vacuuming street pavements with a modified portable car vacuum cleaner. In addition, dust samples were collected on a weekly basis at each site over a period of five months in 1985 by sweeping a standard unit pavement area of two square feet (equal to 0.186 m^2). A plastic household dustpan and brush were used to collect samples. Sweeping procedures were pre-tested and standardised prior to the commencement of formal sampling.

In order to avoid cross-contamination of samples, at each site the collecting materials were changed or thoroughly cleaned using lead-free alcohol based swabs. Dust samples were stored in plastic bags and tightly sealed.

In order to avoid exposure to rain and redistribution by wind, dust sampling took place on relatively calm days after at least two days of dry weather. In all around 300 dust samples were collected.

3.7.3 PREPARATION AND ANALYSIS OF DUST SAMPLES

Samples were analysed by the Scientific Services Laboratory in Rondebosch, Cape Town.

There are no standardised procedures for the preparation and analysis of dust samples, particularly with respect to which particle size fractions are analysed. In order to compare the lead concentrations of the one millimetre fraction with the total sample, samples collected by the sweeping method were sieved using a one millimetre mesh and the two fractions analysed separately. Vacuum samples were not sieved prior to the chemical analyses.

All samples were dried, weighed to four decimal points and transferred to conical flasks. The samples were digested for two hours on a hot plate at 150 °C, with 10 ml concentrated nitric acid. After cooling, the samples were filtered and diluted to an appropriate volume.

A Varian AA5 atomic absorption spectrophotometer was used to perform lead analyses. With each batch of samples a reagent blank and set of working standards were run simultaneously.

3.8 STATISTICAL ANALYSES

As air lead levels were not normally distributed, log transformations of the variable air lead were performed to achieve normalisation. One and two way analysis of variance techniques were used to test for differences between sites, seasons, days of the week and interaction effects between site and season. Kendall and Spearman rank correlation coefficients were calculated to test for correlations between air lead concentrations and traffic density. Site two was excluded from some of the formal statistical analyses due to missing values at this site.

To test for differences between dust lead concentrations at the various sites, a Kruskal-Wallis test was performed. Spearman rank correlation coefficients were determined to examine the relationship between lead concentrations in the one millimetre fraction and total sample, the relationship between mass loading and concentration, the relationship between dust lead and air lead, and dust lead and traffic volume.

PART C : CASE CONTROL STUDY

3.9 HOME VISITS

White children were excluded from the case control study as it was decided to match children according to ethnic group. Coloured children, identified in the Woodstock cross-sectional analytical survey with blood lead concentrations greater than or less than a designated cut-off level, who at the time of the blood tests had been permanently resident in the study area for at least six months, were eligible for inclusion into the study. A case was defined as a coloured child having a blood lead level of 24 ug/dl or above. A control was defined as a coloured child having a blood lead level of 14 ug/dl or below. In order to increase the numbers of children, additional inner-city cases and controls identified subsequently by the author, in a lead screening study in a section of the urban community situated adjacent to the city centre of Cape Town, were included in the study (von Schirnding and Fuggle, 1986b). All in all 32 cases were identified, of whom five (16%) were lost to follow up. Cases lost to follow-up comprised those children whose families had moved out of the area and could not be traced. 43 controls were identified of whom seven (16%) were lost to follow-up. The final study population comprised 27 cases and 36 controls.

All home visits were completed during the six month period March to September 1985. At each home visited, an interview was

conducted with the parent or guardian of the child (this was normally the mother) (Appendix E2). Samples of water, paint, house dust, street dust, and air were obtained for chemical analyses. Soil samples were not obtained, as the majority of homes had no front garden. A 'walkabout' was undertaken through each home, during which the condition of flaking surfaces was assessed, the type of water piping determined and the overall state of home cleanliness and hygiene assessed (Appendix E1). All interviews and environmental evaluations were performed by the author with the assistance of an environmental technician on a double-blind basis - neither the interviewer, environmental technician nor interviewee had prior knowledge of the blood test results. Repeat blood samples were obtained from cases and controls.

3.10 ENVIRONMENTAL EVALUATIONS

3.10.1 INTERIOR SURFACE DUST SAMPLES

At least one dust sample (normally two or more) was obtained from the main living room and child's bedroom, as well as from other rooms or play areas which the child was said to frequent. Dust samples were obtained by systematically sweeping a standard area of two square feet so that results could be expressed in terms of mass-loading per unit area (ug/2 sq.ft) (equal to ug/0.186 m^2) as well as concentration (parts per million (ppm)). Sweeping

procedures were pre-tested and standardised prior to the commencement of formal sampling. A standard household plastic dust-pan and brush was used to collect samples. All samples were stored in plastic bags and tightly sealed.

The contents of each sample bag was weighed to four decimal points, transferred to a conical flask and digested with 10 ml concentrated nitric acid for two hours on a hot plate at 150 °C. After cooling, the samples were filtered and diluted to appropriate volumes. Samples were analysed on a Varian AA5 atomic absorption spectrophotometer.

3.10.2 PAINT SAMPLES

A sample of paint was obtained from flaking surfaces inside and outside the home. Surfaces sampled included walls, windowsills, doors and railings. Where possible, full-thickness (multilayer) samples were collected. Samples were prepared and analysed in the same manner as dust samples.

3.10.3 WATER SAMPLES

A sample of water was obtained from the kitchen tap. Plastic lead-free containers with screw-on tops were used to collect water samples.

The water samples were concentrated by successive evaporation of 25 ml portions, with the addition of 0.5 ml nitric acid at each step to ensure conversion of lead to the salt form. The dry residue after five steps was dissolved in five ml five percent nitric acid. Samples were analysed on a Varian AA5 atomic absorption spectrophotometer.

3.10.4 AIR SAMPLES

A maximum of six air pumps was available at any one time, therefore groups of six randomly chosen cases and controls (three cases and three controls) were selected for successive periods of air monitoring at individual homes. Air samples were obtained from 24 homes altogether. Where possible, filter units were placed 1.5 to two metres above the ground at the front entrance to the home. In certain cases alternative site positions in the home were found if suitable outlets at the front entrance were not available. For details relating to other aspects of the air sampling procedure and analytical methods, the reader is referred to Sections 3.6.1 to 3.6.3.

3.10.5 STREET DUST SAMPLES

A spot street-dust sample was obtained from the pavement at the entrance to each home by systematically sweeping a two square foot (0.186 m^2) area in the standardised way. Samples were analysed in the same way as interior surface dust samples (Section 3.10.1).

3.10.6 HOME WALKABOUTS (APPENDIX E1)

3.10.6.1 Flaking Paint Assessments

The condition of flaking surfaces was rated separately inside and outside the home. Homes were rated on a scale of one to three as being either dilapidated (nearly all the surfaces in a flaking condition), deteriorating (some surfaces to a significant degree in a flaking condition) or well maintained (nearly all the surfaces in a non-flaking condition).

3.10.6.2 Water Pipes

Both inlet and outlet water pipes which were accessible were examined and the construction material determined.

3.10.6.3 Home Cleanliness Assessments

The general state of home hygiene and cleanliness was assessed using a modified version of the rating scale devised by Smith and colleagues (Appendix E1). Answers to individual questions in a checklist were designated a 'zero' if a negative response was appropriate, and a 'one' if a positive response was indicated. Points were then summed. A score of zero to three was rated satisfactory, four to seven in need of attention and eight to ten unsatisfactory.

3.11 HOME INTERVIEWS (APPENDIX E2)

The home interviews focussed primarily on information relating to mechanisms of lead exposure and included a number of questions about the hand-mouth activities of the child (QD1-9). Information sought related to various items such as evidence of pica for certain non-food items (QD1-5), mouthing activity (sucking of fingers (QD6), mouthing of hands and objects (QD7)) and hand-washing activity (QD8, D9). Information about the child's main play areas was also obtained (QD10, 11, 12, 15) and a broad range of questions concerned with the nature and quality of supervision and stimulation which the child received were included. Information sought related to items such as who the child's main caretaker was (QD14), whether or not the child had attended a creche (QD13), whether the child had access to toys,

books (QD16), went on family outings (QD21) and other similar concerns. The interview also contained questions which were concerned with the child's dietary composition (QC1-4), housing (age of the house, fuel source, renovation, painting or decorating activities over the previous year) (QF1-8) and included also a very brief medical history (QB1-4) and behavioural history.

Items of interest which related to socio-demographic, cultural and economic factors included details about the child's family composition (number of people, number of siblings in the home) (QE1-6), religion (QA4), home language (QA3) and economic standing (mother and father's schooling, job, income) (QG1-10).

3.12 NUTRITIONAL STATUS

Height and weight measurements were performed on children at school after the blood lead screening surveys (see Section 3.3 for details of the methods used) and the three standard indicators of nutritional status, weight for age, height for age and weight for height, were determined. These indicators were compared with the distribution of indicators in an international reference population (Section 3.3).

3.13 REPEAT BLOOD SAMPLES

During the home visits repeat blood samples were obtained following parental permission, from case and control children (95% of the study population). Duplicate samples of three to four ml venous blood were obtained where possible (see Section 3.2.2.1 for a description of the analytical methods used). It was found that the blood lead levels of the cases and controls had decreased by five ug/dl and three ug/dl respectively upon repeat measurement. The decline in both groups may have reflected a change in blood lead level due to age, seasonal variation or measurement error. The magnitude of the difference in the blood lead distributions of the cases and controls upon repeat measurement however, remained stable.

3.14 STATISTICAL ANALYSES

Univariate analyses were performed to test for differences among the two groups (cases and controls) with respect to the individual variables. For the discrete variables, the Fishers Exact or Maximum Likelihood Chi Square Tests were performed. For continuous variables, due to the relatively low numbers and skewed distributions, the Mann-Whitney U test was used. In addition, a series of multi-way contingency tables were formed and log linear models used to analyse them (Dixon, 1981).

ODDS RATIOS

Odds ratios were used to quantify the strength of association between selected factors (for example, mouthing activity) and case-control status.

In a 2 x 2 table the following notation is used:

		Case	Control
Selected factor	Present	a	b
	Absent	c	d

The exposure odds ratio is the ratio of the odds in favour of exposure among the cases (a/b) to the odds in favour of exposure of controls (c/d), ie, ad/bc. Approximate 95% confidence intervals for the odds ratios were calculated (Schlesselman, 1982).

CHAPTER 4

RESULTS AND DISCUSSION : CROSS SECTIONAL ANALYTICAL STUDY (PART A)

In the previous chapter the methods utilised in the cross sectional analytical study, the environmental study and the case control study were described. In the following four chapters the results of these respective works are described and discussed. Aspects of this work have been published elsewhere or presented at conferences (von Schirnding and Fuggle, 1984b; von Schirnding and Fuggle, 1986b,c; von Schirnding, 1987a,b).

The present chapter deals with some of the results of the cross-sectional analytical study. First, blood lead concentrations are described in relation to the factors ethnic group, gender, age, school and residential area. Next, haematological factors such as haemoglobin and haematocrit levels in the population, and the nutritional status of the childen, are described. The relationship between these factors and blood lead levels is also described. It should be noted that there are missing values for several variables relating to haematological and nutritional parameters. These are not due to any systematic bias but rather to logistical factors. Missing values for variables in the questionnaire survey are dealt with in the text.

4.1 BLOOD LEAD CONCENTRATIONS

For the purpose of comparison, descriptive statistics (mean, median and standard deviation) are used to describe distributions. Tables 4.1 to 4.4 give the parameters of the blood lead distribution for the Woodstock population, which are illustrated graphically in Figures 4.1 to 4.3. The mean blood lead level for all children living in the study area was 15 ug/dl and the median was 16 ug/dl. The standard deviation was six ug/dl, illustrating considerable variation (Table 4.1). Overall, eight percent of children had blood lead concentrations $\geq$ 25 ug/dl, the current United States of America action level for lead in blood in young children.

TABLE 4.1

BLOOD LEAD CONCENTRATIONS (ug/dl)

No.	169
Mean (ug/dl)	15
Std. Dev.	6
Median	16
% $\geq$ 25	8

4.1.1 BLOOD LEAD CONCENTRATIONS BY ETHNIC GROUP

From Figure 4.1 it can be seen that the shape of the distribution curve approximated that of a bi-modal distribution with peaks at

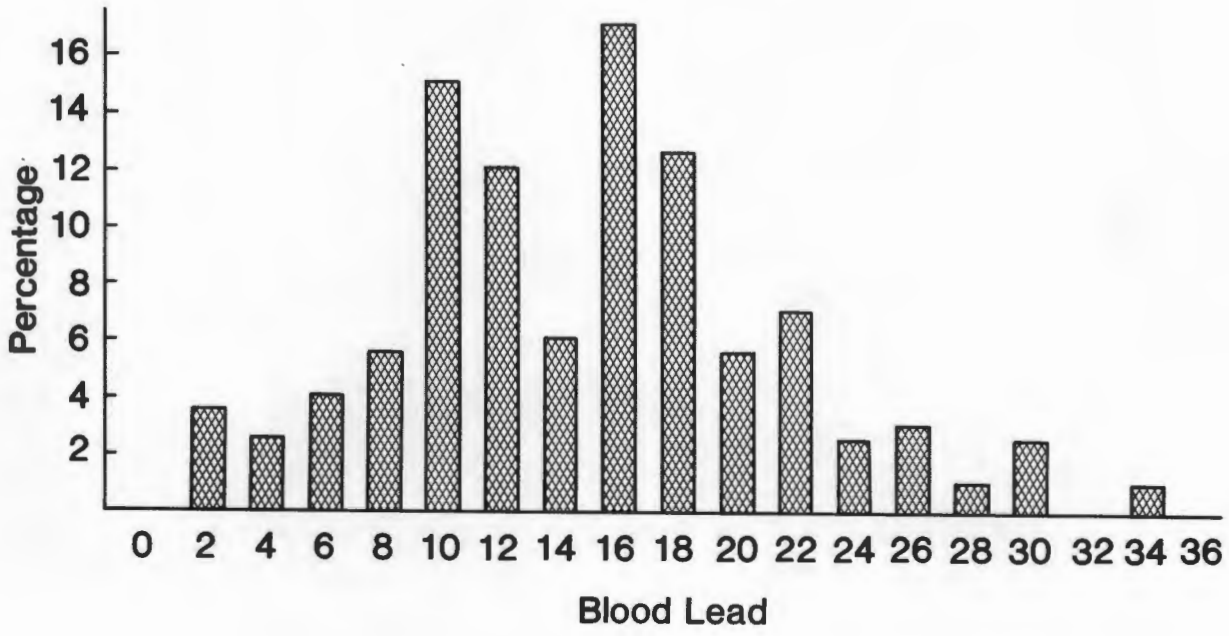


FIGURE 4.1

BLOOD LEAD CONCENTRATIONS (ug/dl) (ALL PUPILS)

10 and 16 ug/dl. It was decided to see whether any difference with respect to ethnic group occurred. From the analysis of the distribution according to white and coloured ethnic groups (Table 4.2), it was evident that a marked difference existed in the blood lead concentrations of the two groups. Each group was approximately normally distributed, with the white population having a mean of 12 ug/dl and the coloured population having a mean of 18 ug/dl. The difference in blood lead concentrations was statistically highly significant (t-test performed, $p = 0.0001$) (Figure 4.2). Among the coloured pupils, 13% had blood lead levels $\geq$ 25 ug/dl, the United States action level. No white pupils had blood lead levels in this range (Figure 4.3). Due to the finding of these ethnic differences, (discussed in Section 5.1.3), further analyses were stratified by ethnic group.

TABLE 4.2

BLOOD LEAD CONCENTRATIONS (ug/dl) BY ETHNIC GROUP

	Coloured Pupils	White Pupils
No.	104	65
Mean (ug/dl)	18	12
Std. Dev.	6	5
Median	17	11
% $\geq$ 25	13	0

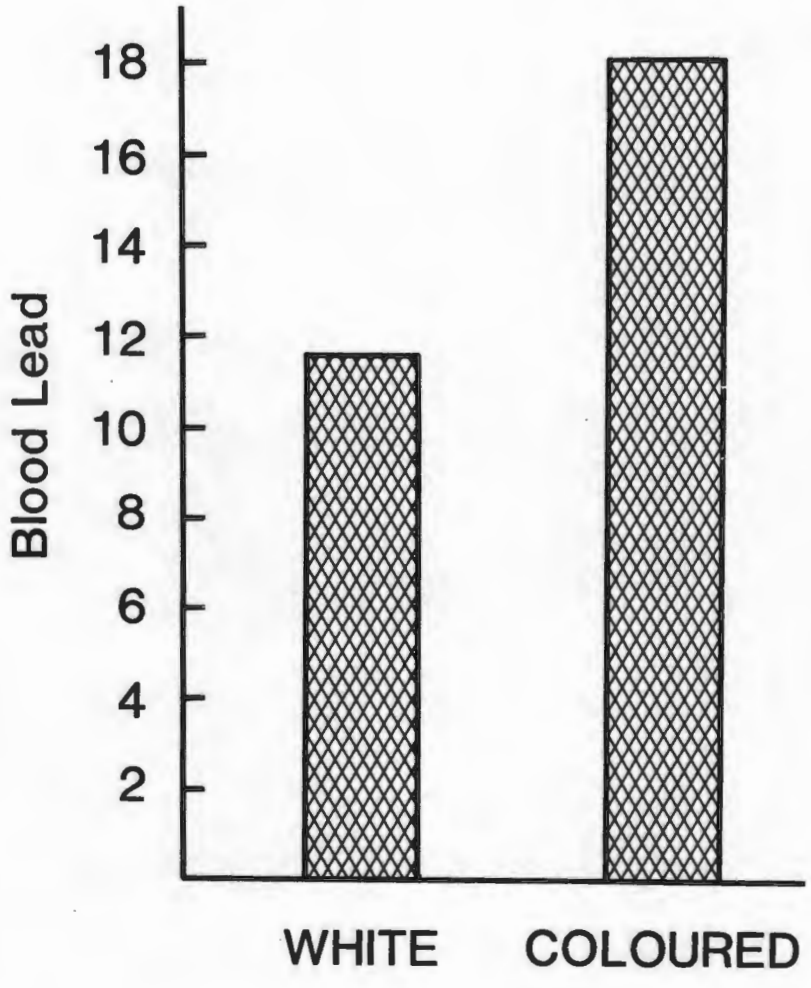


FIGURE 4.2

BLOOD LEAD CONCENTRATIONS (ug/dl) BY ETHNIC GROUP

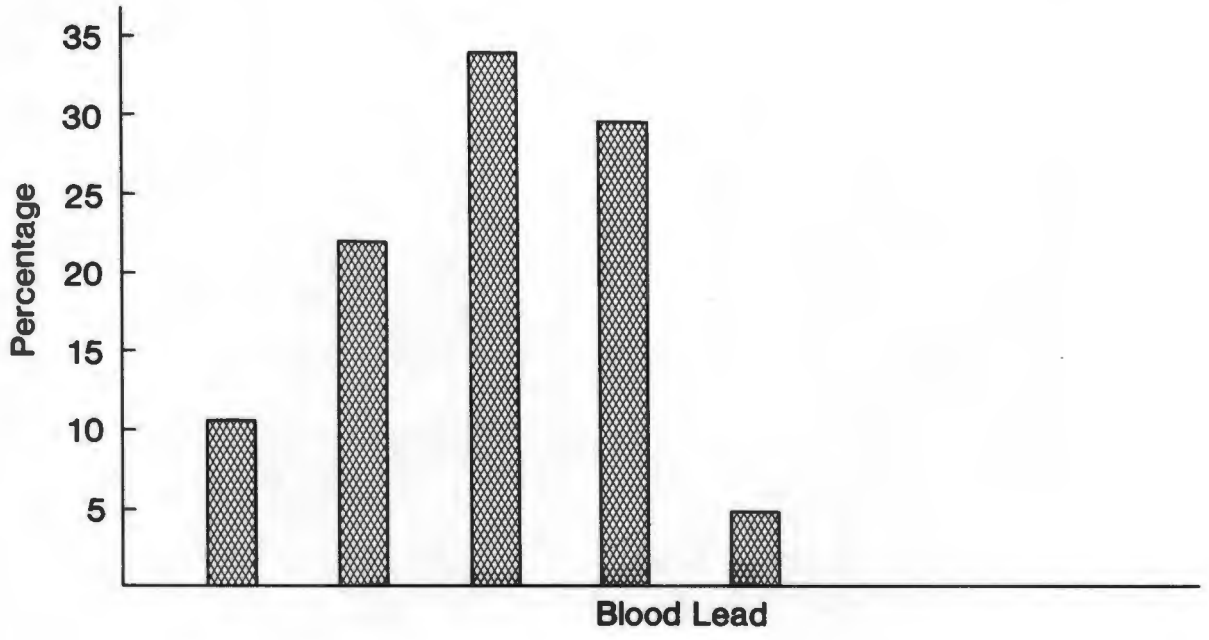


FIGURE 4.3a

BLOOD LEAD CONCENTRATIONS (ug/dl) (WHITE PUPILS)

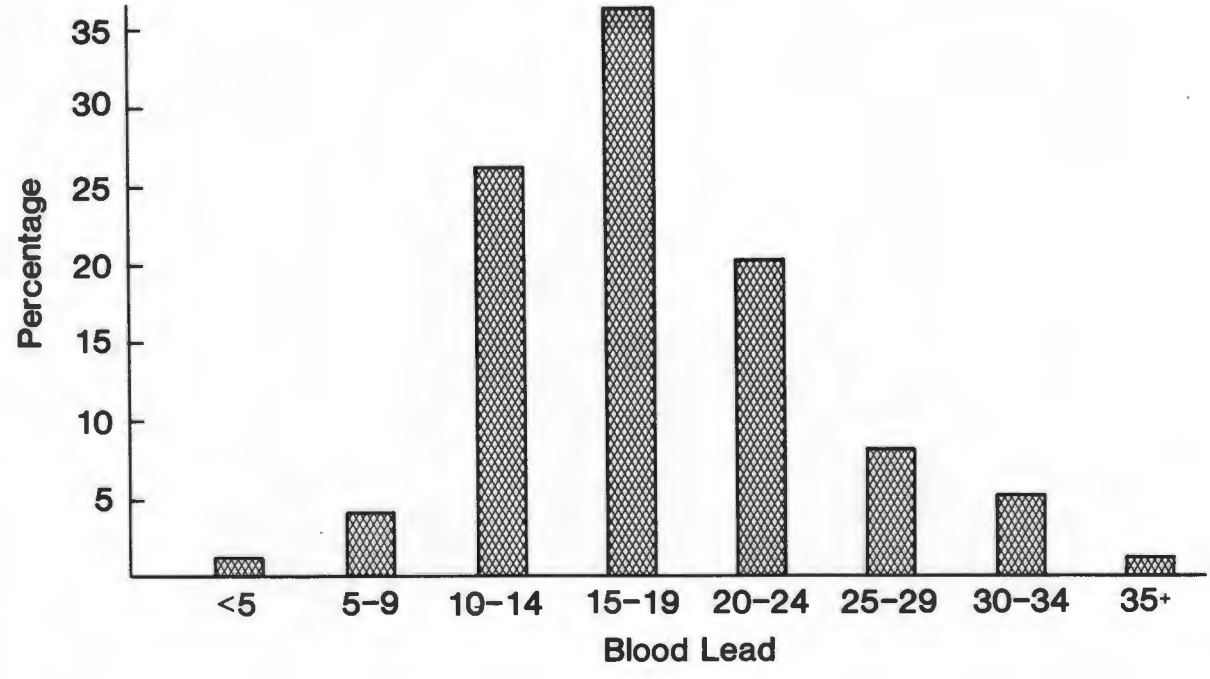


FIGURE 4.3b

BLOOD LEAD CONCENTRATIONS (ug/dl) (COLOURED PUPILS)

4.1.2 BLOOD LEAD CONCENTRATIONS BY GENDER AND AGE

It was found that there was no difference in blood lead concentration with respect to gender, among either the white or coloured groups (t-tests performed, $p = 0.7737$, 0.8275 respectively) (Table 4.3).

TABLE 4.3

BLOOD LEAD CONCENTRATIONS (ug/dl) BY ETHNIC GROUP AND GENDER

	Coloured Pupils		White Pupils	
	Females	Males	Females	Males
No.	49	45	26	34
Mean (ug/dl)	18	18	11	11
Std. Dev.	6	7	6	4
Median	18	17	10	11
% $\geq$ 25	12	15	0	0

Although all children were in the first grade, it was nevertheless decided to determine whether any difference in blood lead concentration occurred between children less than seven years of age and children of seven years or older. There were no statistically significant differences in blood lead levels between the two age groups among the white or coloured pupils (t-tests performed, $p > 0.05$ respectively) (Table 4.4).

TABLE 4.4

BLOOD LEAD CONCENTRATIONS (ug/dl) BY ETHNIC GROUP AND AGE

	Coloured Pupils		White Pupils	
	<7 years	≥7 years	<7 years	≥7 years
No.	52	46	21	38
Mean (ug/dl)	18	17	12	11
Std. Dev.	6	6	5	5

4.1.3 DISCUSSION

4.1.3.1 Overall Blood Lead Concentrations

In the preceding section, the blood lead distribution of children living in the study area was determined.

Caution should be exercised in comparing the blood lead results of this part of the study with those carried out in different parts of the world. Differences occur in various studies with respect to the nature of the population sampled (for example with respect to age distributions, national versus local population samples), the methods of blood sampling and analysis used, and the quality control procedures utilised. With these reservations in mind, an attempt is made here to draw some preliminary comparisons between various studies.

With respect to research carried out in South Africa, the blood lead levels of this population, average age six to seven years,

were found to be slightly higher than those of a group of 89 inner city children (average age six years) attending a dental clinic in Cape Town, which were 12.7 ug/dl (White et al., 1982). The blood lead levels of both these groups of inner city children are above those of children living in other parts of the Cape Peninsula. For instance, von Schirnding (1982) found that blood lead levels of a group of children aged six to eight years from a semi-rural area in Hout Bay, Cape Province, averaged 11 ug/dl.

White et al. (1982) found that blood lead levels of 114 hospitalised children (mean age five years) living in and around the Cape Peninsula averaged 8.2 ug/dl.

Grobler et al. (1985) studied 30 children aged 14 to 16 years living in a remote area of the Cape Province. They found that blood lead levels averaged 3.4 ug/dl, ranging from 0.5 to 7.5 ug/dl. No information on the ethnic composition or sex distribution of the populations sampled by White et al. (1982) or Grobler et al. (1985) was given.

Studies in other parts of the world have confirmed that blood lead levels of populations living in remote areas are considerably below those of urban populations.

In a study of blood lead levels in 100 Papua New Guinea children aged seven to ten years, living in a remote area, it was found that the mean blood lead level was 5.2 ug/dl, ranging from one to

thirteen ug/dl (Poole et al., 1980). In a study of blood lead levels in a remote Himalayan population, it was found that blood lead levels in 47 children aged three to twelve years old averaged around 3.5 ug/dl (Piomelli et al., 1980). A group of 90 Yanomama Indians from a remote part of Venezuela have been reported to have blood lead levels averaging 0.83 ug/dl, which are among the lowest recorded in the world (Hecker et al., 1974).

From the NHANES II survey (National Health and Nutrition Examination Survey) conducted during 1976 to 1980 on a representative sample of the United States population aged six months to 74 years, it was found that the mean blood lead level for the six to eight year old age group was 13.9 (median 13 ug/dl) (Mahaffey et al., 1982). Among children under six years living in central cities however, lead levels were markedly higher, averaging 20 ug/dl (Mahaffey et al., 1982). This is considerably above the blood lead levels of inner city first grade children in the present study, but the United States population sample included younger children who are at higher risk.

Blood lead levels in the United Kingdom are considerably below those of the United States of America and it is relatively rare to find children with blood lead levels $\geq$ 20 ug/dl (Prof. Barltrop, pers. comm.). From the EEC surveys carried out 1979 to 1980, 3.1% of the inner city population were found to have blood lead levels greater than 25 ug/dl (Southwood, 1983).

In Australia, blood lead surveys have been carried out in various states. In two surveys of Sydney school children, 12% and 24% of children respectively had blood lead levels greater than 25 ug/dl (Garnys et al., 1979). The mean blood lead levels of children in the two surveys were 17 ug/dl and 21 ug/dl respectively. These values are somewhat higher than those reported in the present study, but it has been suggested that contamination of capillary blood samples might have occurred (de Silva and Donnan, 1980).

In another survey of primary school children in Sydney, mean blood lead levels of 324 children at four schools were found to vary between 13 ug/dl and 18 ug/dl. Seven children were found to have blood lead levels $\geq$ 30 ug/dl (Nolan, 1982). These results are comparable to those found in the present study.

In a study of children from an inner suburban area of Melbourne, it was found that 10% of 62 pre-school children had blood lead levels over 25 ug/dl, the highest being 37 ug/dl. No school age children, however, had blood lead levels over 25 ug/dl (de Silva and Donnan, 1980). These values would thus appear to be lower than the blood lead levels found in the present study. In a study of 513 pre-school children from an industrial working class region of Adelaide, the geometric mean blood lead level was found to be 16.3 ug/dl, suggesting that blood lead levels in this pre-school population are of a similar magnitude to that of the school-age population studied here (Calder et al., 1986).

Blood lead levels of children from Scandinavia and Finland are considerably below those of the Australian and American children discussed above. In a study of blood lead levels in a group of 286 Finnish pre-school children, blood lead levels were found to vary between six and 6.7 ug/dl (Taskinen et al., 1981). In a study of blood lead levels of 1 395 Swedish children, it was found that the average blood lead level was 5.5 ug/dl (Skerfving et al., 1986). Whilst these studies sampled also non-urban children, it is evident that mean blood lead levels in these countries are extremely low.

From the respective works quoted above, it is apparent that much variation in blood lead levels occurs in various parts of the world. As already indicated, it is difficult to make direct comparisons between various studies, but the data suggests that blood lead levels of this inner city Woodstock population are more comparable to those of American and Australian children than they are to Scandinavian children, which are close to blood lead levels reported in remote unpolluted areas of the world.

4.1.3.2 Blood Lead Concentrations by Age, Gender, and Ethnic Group

Considerable variation in children's blood lead levels was found

within the study area. The NHANES II study identified age, gender and race as major demographic covariates in the United States population (Mahaffey et al., 1982).

AGE

No significant differences in the blood lead levels of children above or below seven years of age were found. This was expected, as the age range in the population was restricted to first grade pupils.

The NHANES II survey found that for children under six years of age, there was no significant association between age and blood lead levels, but for children six to 17 years, there was a statistically significant association (Mahaffey et al., 1982). Mean blood lead levels decline with increasing age until late adolescence, when they gradually increase (Mahaffey et al., 1982).

GENDER

No significant difference in the blood lead levels of boys and girls was found. In general, differences in blood lead levels between the sexes are not significant for children below the age of six years. For older children between the ages six to 17 years, the difference in mean blood lead levels increases with age, with boys having higher blood lead levels than girls. The

United Kingdom EEC survey found only a small difference in the blood lead levels of boys and girls (0.5 ug/dl higher in boys) up to the age of 12 years (Quinn, 1985). More pronounced and significant differences occur in adolescents and adults.

ETHNIC GROUP

In the present study, blood lead levels were found to vary significantly between ethnic groups. The average blood lead concentration of the coloured population was 18 ug/dl. This was six ug/dl higher than that for the white population, which was 12 ug/dl. 13% of coloured pupils had blood lead levels greater than or equal to 25 ug/dl, the United States action level. No white pupils had blood lead levels in this range.

In a study of blood lead levels in 293 Cape urban coloured preschoolers by Deveaux et al. (1986), the mean blood lead level was found to be 16.2 ug/dl. This is slightly lower than that found for the Woodstock first grade coloured children in this study. Both the present study and that by Deveaux et al. found that approximately 13% of coloured children had blood lead levels greater than or equal to 25 ug/dl.

Kilroe-Smith (1987) has recently reported the blood lead levels of groups of school children aged 11 to 13 years and older, living in Johannesburg. No significant differences between

ethnic groups were found in this study, but relatively few "non-white" pupils were sampled.

In the United States, average blood lead levels of blacks are consistently higher than whites. These differences hold across both sexes and all age groups, income groups and residential areas. For children aged six months to five years, it was found that blood lead levels of black children were on average six ug/dl higher than for white children (Mahaffey et al., 1982). This difference is of the same order of magnitude as that found between coloured children and white children in this study.

Billick et al. (1979) analysed data from New York city blood lead screening programmes and found that in 1976, among five to six year olds the geometric mean was 18.2 ug/dl for black children, 16.7 for Hispanic children and 15.9 for white children in New York city. From this study of New York children it would appear that the difference in blood lead levels between the races is considerably smaller than that reported in the NHANES II study which included children of a younger age. The differences reported between the ethnic groups in the present study are also considerably higher than those for the New York children.

The United Kingdom EEC study showed little evidence of racial differences, but included only a very small proportion of non-whites (< 10%). The results should thus be viewed with caution (Quinn, 1985). It was found that the average blood lead

concentration in the Indian/Pakistani/Bangladeshi group was higher (by 1.6 ug/dl) than the average level for whites (Quinn, 1985). A large study of pre-school children in Birmingham reported that elevated levels of lead occurred in Asian children (Archer et al., 1980).

In a study by Sherlock et al. (1985), it was found that blood lead concentrations in children were not related to ethnic origin when the effect of other factors was allowed for. Three groups of 35 children aged two and a half to five years, matched for gender and age, were studied. One group consisted of Caucasian children, and the other two groups of children were vegetarians and non-vegetarians respectively. Asian children had blood lead levels averaging 8.1 ug/dl, whilst Caucasian children had blood lead levels averaging 9.7 ug/dl. These differences were statistically significant.

In general, this part of the analysis of blood lead concentrations showed that there were no significant differences in blood lead concentrations between the sexes or age groups, which was expected. Variation in blood lead concentrations between the ethnic groups was considerable and highly significant. It was decided to see if differences in blood lead concentrations occurred with respect to the school attended by the child and the residential zone where children lived.

4.1.4 BLOOD LEAD CONCENTRATIONS BY SCHOOL AND RESIDENTIAL ZONE

As can be seen from Table 4.5a and Figure 4.4, considerable variation in the blood lead concentrations of pupils existed between schools.

TABLE 4.5a

BLOOD LEAD CONCENTRATIONS (ug/dl) BY SCHOOL

	SCHOOL						
	1	2	3	4	5	6	7
No.	15	21	30	35	27	22	19
Mean (ug/dl)	13	18	9	14	20	22	13
Std. Dev.	4	5	5	4	4	7	4
Median	13	18	8	13	21	20	13

A one-way analysis of variance was performed which indicated that the variation in blood lead levels between schools was statistically highly significant ($p = 0.0001$). Significant differences in blood lead concentration between pairs of schools are given in Table 4.6.

Among the coloured schools the median blood lead levels at schools two, five and six, with children living in residential zones one and two (see Figure 3.1) were 18 ug/dl, 21 ug/dl and 20 ug/dl respectively; these were higher than at schools one and seven, with children living in residential zone three, which had median blood lead levels of 13 ug/dl. The mean blood lead

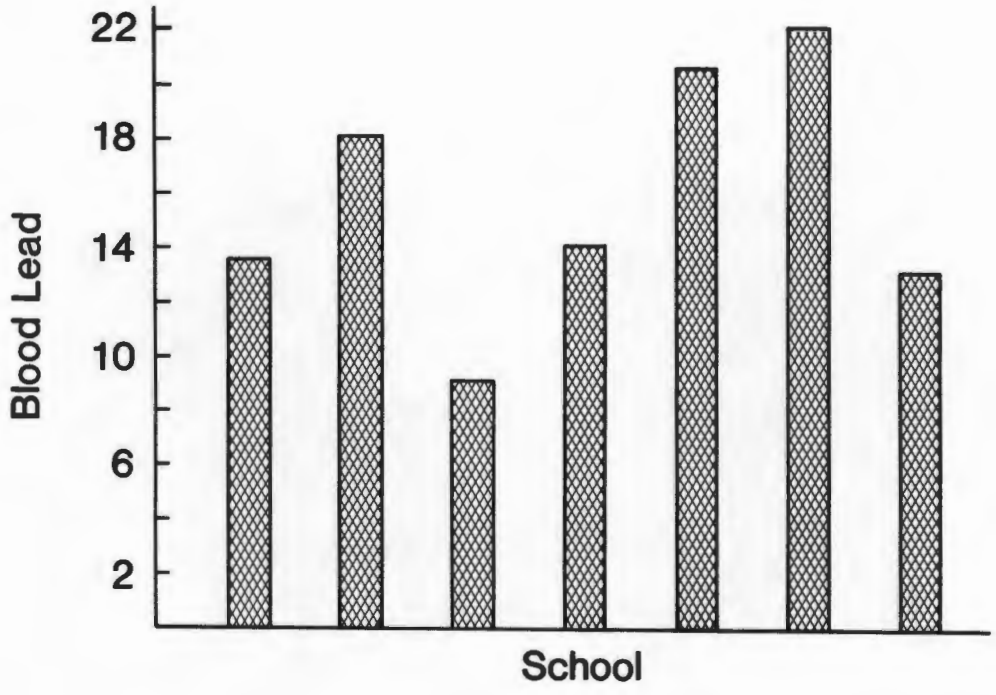


FIGURE 4.4a

BLOOD LEAD CONCENTRATIONS (ug/dl) BY SCHOOL

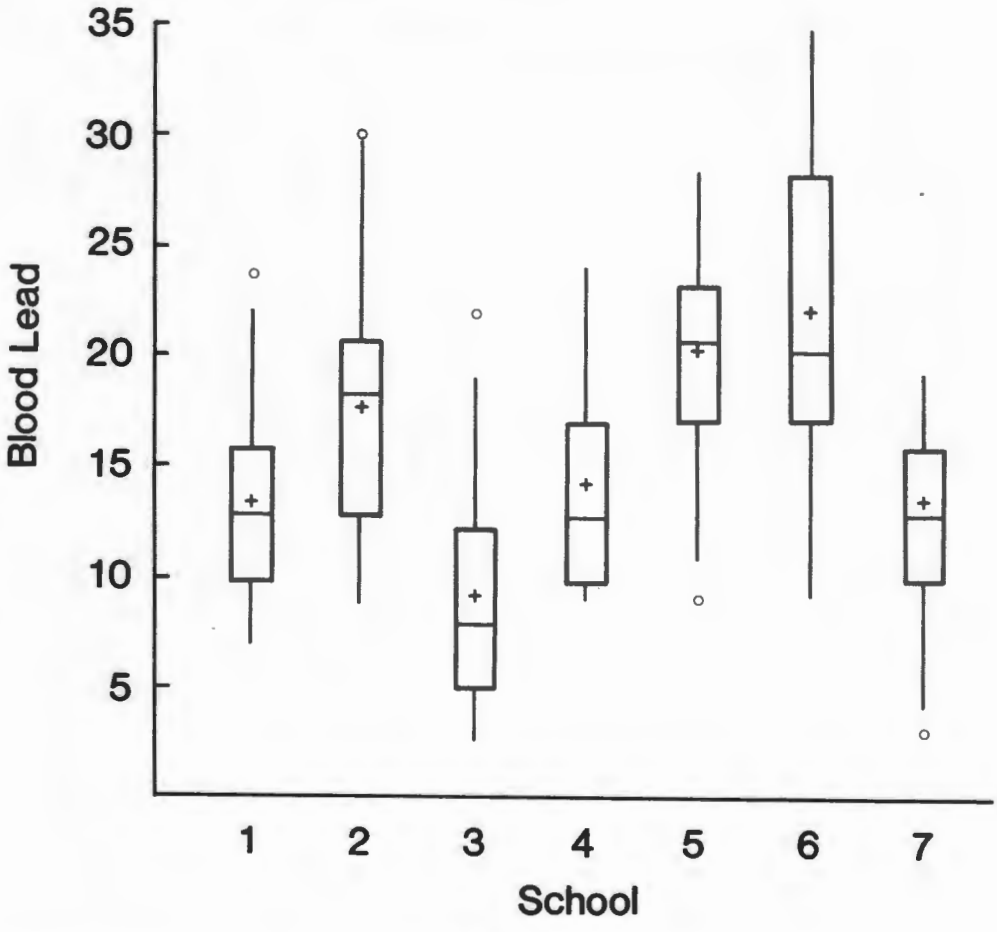


FIGURE 4.4b

BLOOD LEAD CONCENTRATIONS (ug/dl) BY SCHOOL (BOX PLOTS)

level for the former group of three schools considered together was 20 ug/dl; this was statistically different from that of the latter group of two schools considered together, which was 13 ug/dl (t-test performed, p = 0.0001).

TABLE 4.6

DIFFERENCE IN BLOOD LEAD CONCENTRATIONS BETWEEN SCHOOLS
* DENOTES STATISTICALLY SIGNIFICANT DIFFERENCE AT 5% SIGNIFICANCE LEVEL

	S C H O O L						
School	1	2	3	4	5	6	7
1					*	*	
2			*				*
3		*		*	*	*	
4			*		*	*	
5	*		*	*			*
6	*		*	*			*
7		*			*	*	

TABLE 4.5b

BLOOD LEAD CONCENTRATIONS (ug/dl) BY SCHOOL GROUP (COLOURED PUPILS)

	Group 1 (Schools 2, 5, 6)	Group 2 (Schools 1, 7)
No.	70	34
Mean (ug/dl)	20	13
Std. Dev.	6	4
Median	19	13
% $\geq$ 25	20	0

Among the white population, the median blood lead level at school three was eight ug/dl; this was considerably lower than that at school four, which was 13 ug/dl. This difference was statistically significant ($p = 0.0004$). Children from both schools three and four lived in residential zones one and two.

Figure 4.5 gives the distribution of blood lead levels in the study area according to ethnic group and residential address. It was decided to determine whether the blood lead levels of coloured children resident in zone three differed from those of children resident in zones one and two. From Table 4.7, it was evident that there was a statistically highly significant difference in the blood lead levels of the children living in the respective zones (Mann-Whitney U test performed, $p = 0.0006$). The median blood lead level of children resident in zone three was 13 ug/dl; children resident in zones one and two had a median blood lead level of 18 ug/dl.

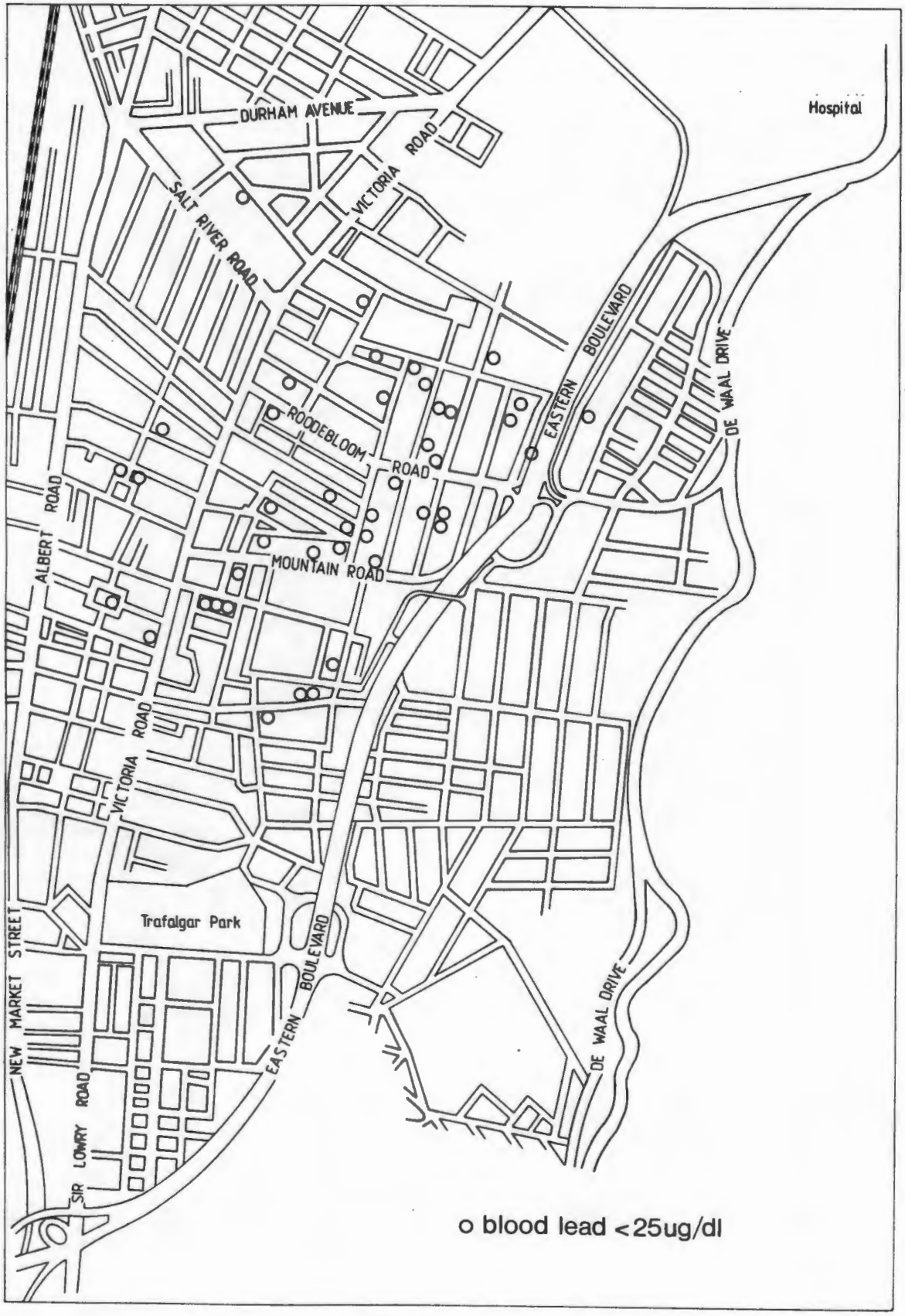


FIGURE 4.5a

DISTRIBUTION OF BLOOD LEAD LEVELS (ug/dl) BY RESIDENTIAL ADDRESS (WHITE PUPILS)

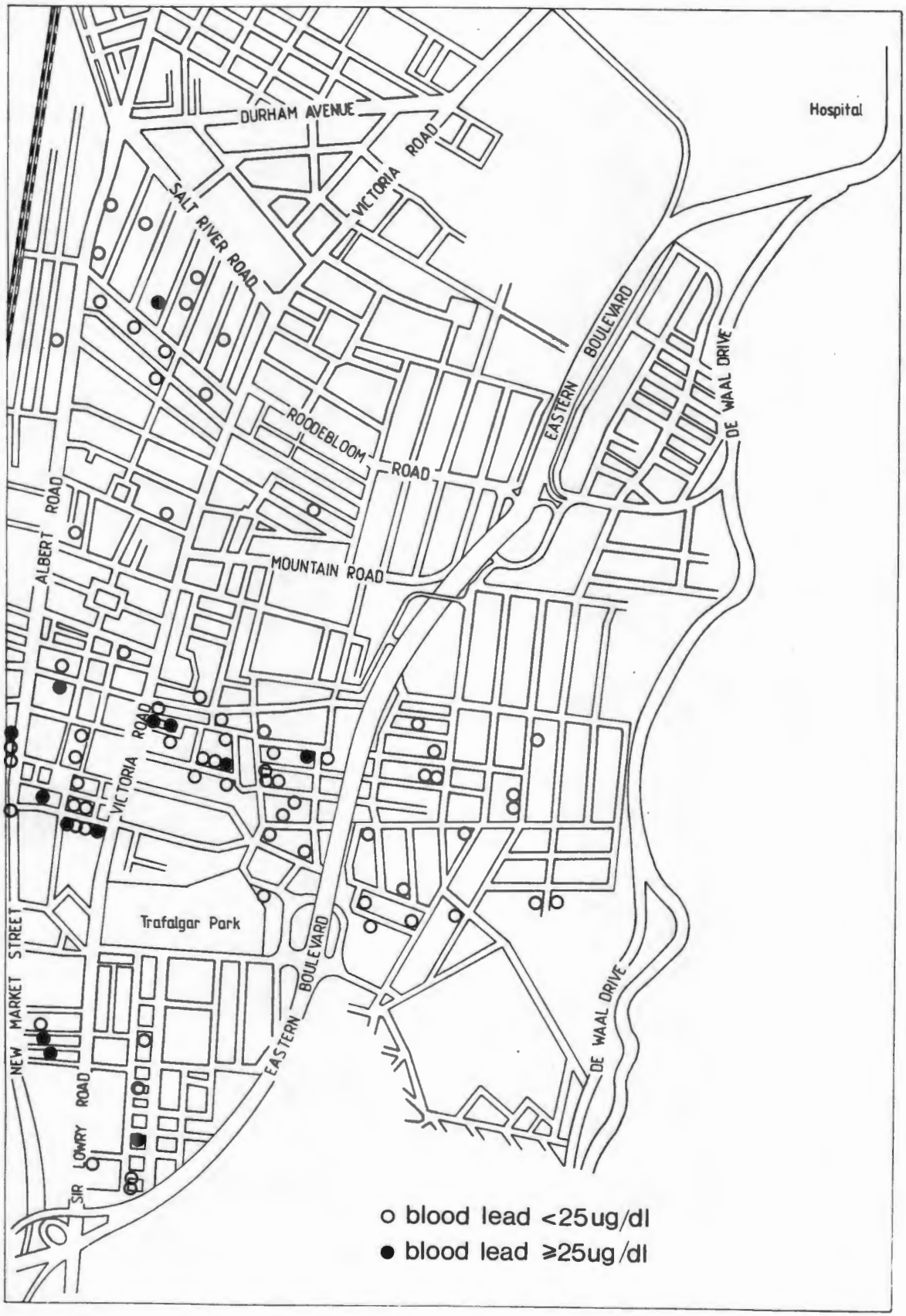


FIGURE 4.5b

DISTRIBUTION OF BLOOD LEAD LEVELS (ug/dl) BY RESIDENTIAL ADDRESS (COLOURED PUPILS)

TABLE 4.7

BLOOD LEAD CONCENTRATIONS (ug/dl) BY RESIDENTIAL ZONE (COLOURED PUPILS)

	Zones 1,2	Zone 3
No.	61	15
Mean	19	13
Std. Dev.	6	5
Median	18	13

The finding that highly significant differences in blood lead concentrations occurred with respect to the school attended by the child (for both coloured and white children respectively) and the residential zone where coloured children lived, was of considerable interest.

These large variations occurred within a relatively small inner city area. It was evident that confounding between the factors residential zone and school existed. (Confounding of factors is addressed in the multivariate analysis, Section 5.1.5.2). Factors such as variations in socio-demographic, cultural and economic indicators, nutritional status and environmental exposure could account for variations in blood lead concentrations between the ethnic groups, and for variations between schools and residential areas. The effect of some of these individual factors on blood lead distributions is discussed in the following sections.

4.2 OTHER HAEMATOLOGICAL PARAMETERS

The descriptive parameters (mean, standard deviation, median) of the distributions for other blood measurements which were performed, including haemoglobin, haematocrit and red blood cell levels, are given in Tables 4.8 to 4.10.

Table 4.8 gives a breakdown of the distribution according to ethnic group, Table 4.9 gives a breakdown according to ethnic group and gender, and an analysis of the distributions by school is given in Table 4.10.

TABLE 4.8a

RED BLOOD CELL LEVELS ($X10^{12}/1$) BY ETHNIC GROUP

	All	Coloured Pupils	White Pupils
No.	154	91	63
Mean	4.75	4.75	4.75
Std. Dev.	0.39	0.42	0.35
Median	4.69	4.69	4.70

TABLE 4.8b

HAEMOGLOBIN LEVELS (g/dl) BY ETHNIC GROUP

	All	Coloured Pupils	White Pupils
No.	154	91	63
Mean	13.16	12.96	13.45
Std. Dev.	1.03	1.12	0.81
Median	13.10	13.00	13.20

TABLE 4.8c

HAEMATOCRIT LEVELS (1/1) BY ETHNIC GROUP

	All	Coloured Pupils	White Pupils
No.	154	91	63
Mean	0.39	0.38	0.39
Std. Dev.	0.03	0.03	0.03
Median	0.39	0.38	0.39

TABLE 4.9a

RED BLOOD CELL LEVELS ($X10^{12}/1$) BY ETHNIC GROUP AND GENDER

	Coloured Females	Pupils Males	White Females	Pupils Males
No.	45	38	25	33
Mean	4.67	4.80	4.75	4.70
Std.Dev.	0.36	0.42	0.30	0.33
Median	4.60	4.75	4.71	4.62

TABLE 4.9b

HAEMOGLOBIN LEVELS (g/dl) BY ETHNIC GROUP AND GENDER

	Coloured Females	Pupils Males	White Females	Pupils Males
No.	45	38	25	33
Mean	13.11	12.90	13.40	13.67
Std.Dev	0.98	1.28	0.79	0.74
Median	13.00	13.10	13.20	13.10

TABLE 4.9c

HAEMATOCRIT LEVELS (1/1) BY ETHNIC GROUP AND GENDER

	Coloured Pupils		White Pupils	
	Females	Males	Females	Males
No.	45	38	25	33
Mean	0.38	0.39	0.39	0.39
Std.Dev	0.03	0.04	0.02	0.02
Median	0.38	0.39	0.39	0.39

TABLE 4.10a

RED BLOOD CELL LEVELS ($x10^{12}/l$) BY SCHOOL

School	No.	Mean	Std. Dev.	Median
1	14	4.64	0.37	4.71
2	20	4.75	0.40	4.89
3	30	4.81	0.43	4.76
4	33	4.69	0.26	4.63
5	20	4.65	0.28	4.62
6	22	4.74	0.41	4.66
7	15	5.01	0.56	5.01

TABLE 4.10b

HAEMOGLOBIN LEVELS (g/dl) BY SCHOOL

School	No.	Mean	Std. Dev.	Median
1	14	12.98	0.92	12.8
2	20	12.90	1.12	13.1
3	30	13.44	0.85	13.1
4	33	13.45	0.79	13.4
5	20	12.41	1.46	12.3
6	22	13.35	0.93	13.1
7	15	13.18	0.77	13.1

TABLE 4.10c

HAEMATOCRIT LEVELS (1/1) BY SCHOOL

School	No.	Mean	Std. Dev.	Median
1	14	0.38	0.03	0.37
2	20	0.39	0.03	0.40
3	30	0.39	0.03	0.39
4	33	0.39	0.02	0.39
5	20	0.37	0.03	0.37
6	22	0.39	0.03	0.39
7	15	0.39	0.03	0.39

With regard to haemoglobin and haematocrit levels, it was evident that white children had slightly higher levels than coloured children. The differences in haemoglobin concentrations were statistically significant (t-test performed, p = 0.0020), but are not of clinical significance, as values were within normal limits. In general the population was not suffering from abnormalities in haemoglobin, haematocrit or red blood cell levels.

4.2.1 RELATIONSHIP OF BLOOD LEAD LEVELS TO OTHER HAEMATOLOGICAL PARAMETERS

Results of correlation analyses are given in Table 4.11 for white and coloured pupils respectively. Appendices B1 and B2 give the scatter plots for the relationship between blood lead and

haemoglobin, haematocrit and red blood cell levels respectively for white and coloured pupils.

TABLE 4.11a

ASSOCIATION BETWEEN RED BLOOD CELL ($X10^{12}/1$) AND BLOOD LEAD LEVELS

	White Pupils	Coloured Pupils	All Pupils
No.	63	91	154
Corr. Coeff.(r)	-.04218	.05176	.02901
"p" Value	.7427	.6261	.7210

TABLE 4.11b

ASSOCIATION BETWEEN HAEMOGLOBIN (g/dl) AND BLOOD LEAD LEVELS (ug/dl)

	White Pupils	Coloured Pupils	All Pupils
No.	63	91	154
Corr. Coeff.(r)	.12950	-.08408	-.10307
"p" Value	.3117	.4282	.2033

TABLE 4.11c

ASSOCIATION BETWEEN HAEMATOCRIT (1/1) AND BLOOD LEAD LEVELS (ug/dl)

	White Pupils	Coloured Pupils	All Pupils
No.	63	91	154
Corr. Coeff.(r)	.05808	.02400	-.00586
"p" Value	.6512	.8214	.9425

Correlation analyses performed revealed that there was no statistically significant relationship between blood lead levels and any of the haematological parameters measured, among either white or coloured pupils.

4.3 NUTRITIONAL STATUS

In order to assess intra-observer variation, repeat height and weight measurements were performed on a class of 35 children at a randomly selected school, and the degree of agreement between the measurements determined. The mean and standard deviation of the differences of the measurements between the two sets of observations were calculated. Table 4.12 shows that the mean difference $\pm$ two standard deviations was less than one cm for height and one kg for weight, indicating satisfactory agreement between the measurements. In addition, there was no relationship between the differences and the average measurements.

TABLE 4.12

REPEAT HEIGHT AND WEIGHT MEASUREMENTS (DIFFERENCE BETWEEN 1ST AND 2ND MEASUREMENTS)

	Height (cms)	Weight (kgs)
No.	35	35
Mean	.08	.33
Std.Dev.	.40	.30
Median	0	.30

Tables 4.13 and 4.14 give an indication of the distribution of the height and weight measurements expressed as a percentage of the NCHS reference standards, of the whole study population, as well as according to ethnic group, and school.

Coloured pupils were on average 98% of their expected height for age, 94% of their expected weight for age and 97% of their expected weight for height. White pupils were on average 100% of their expected height for age, 102% of their expected weight for age and 101% of their expected weight for height. The observed differences between the ethnic groups were statistically significant (but not clinically important) with respect to weight for age and weight for height (t-tests performed, p = 0.0073, 0.0250). Among coloured pupils, 11% had weights less than 80% of their expected weight for age. Among white pupils four percent had weights less than 80% of the expected.

TABLE 4.13a

PERCENT EXPECTED HEIGHT FOR AGE BY ETHNIC GROUP

	All Pupils	Coloured Pupils	White Pupils
No.	147	93	54
Mean	99	98	100
Std. Dev.	5	6	4.5
Median	98	98	99
% < 90	3	3	2

TABLE 4.13b

PERCENT EXPECTED WEIGHT FOR AGE BY ETHNIC GROUP

	All Pupils	Coloured Pupils	White Pupils
No.	148	94	54
Mean	97	94	102
Std. Dev.	16	14	18
Median	95	92	98
% < 80	8	11	4

TABLE 4.13c

PERCENT EXPECTED WEIGHT FOR HEIGHT BY ETHNIC GROUP

	All Pupils	Coloured Pupils	White Pupils
No.	146	92	54
Mean	98	97	101
Std. Dev.	10	9	11
Median	97	96	99

Differences were also observed between the sexes, but these were not statistically significant, although coloured girls were on average 91% of their expected weight for age compared to boys who were on average 96% of their expected weight for age. In general the nutritional status of the two population groups was satisfactory, although the nutritional status of the coloured population was slightly lower than that of the white population.

From the standardised heights and weights of pupils at individual schools, it was apparent that there was relatively little

variation in nutritional status. There were no statistically significant differences among the schools, with the exception of schools two and five, and one and two, where statistically significant differences at the 5% significance level existed with respect to age-standardised height. These differences are not of clinical importance however (Figure 4.6).

TABLE 4.14a

PERCENT EXPECTED HEIGHT FOR AGE BY SCHOOL

	SCHOOL						
	1	2	3	4	5	6	7
No.	15	20	25	29	21	20	17
Mean	96	103	100	99	97	98	98
Std. Dev.	4	8	4	5	5	4	2
Median	97	100	99	98	97	97	99

TABLE 4.14b

PERCENT EXPECTED WEIGHT FOR AGE BY SCHOOL

	SCHOOL						
	1	2	3	4	5	6	7
No.	15	20	25	29	22	20	17
Mean	88	99	101	102	91	95	95
Std. Dev.	12	16	14	22	14	13	12
Median	89	98	98	97	90	92	93

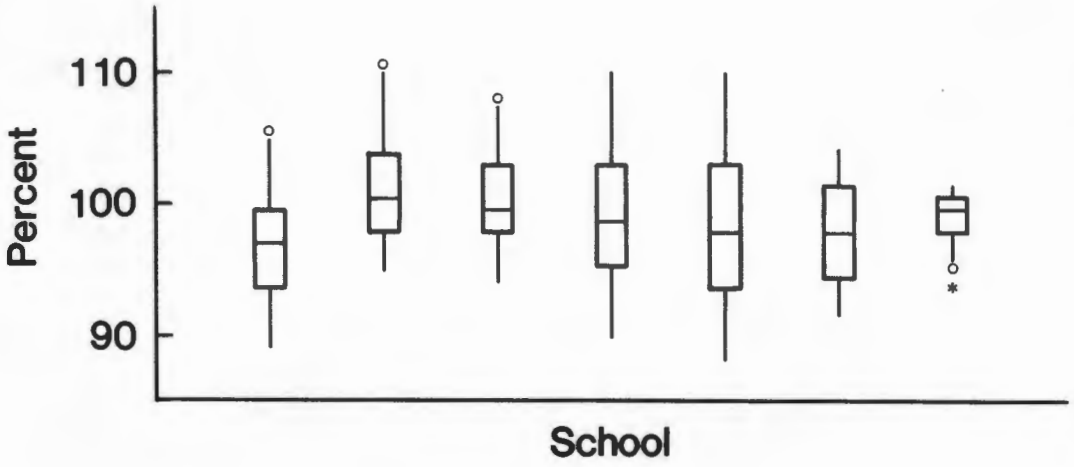


FIGURE 4.6a

PERCENT EXPECTED HEIGHT FOR AGE BY SCHOOL (BOX PLOTS)

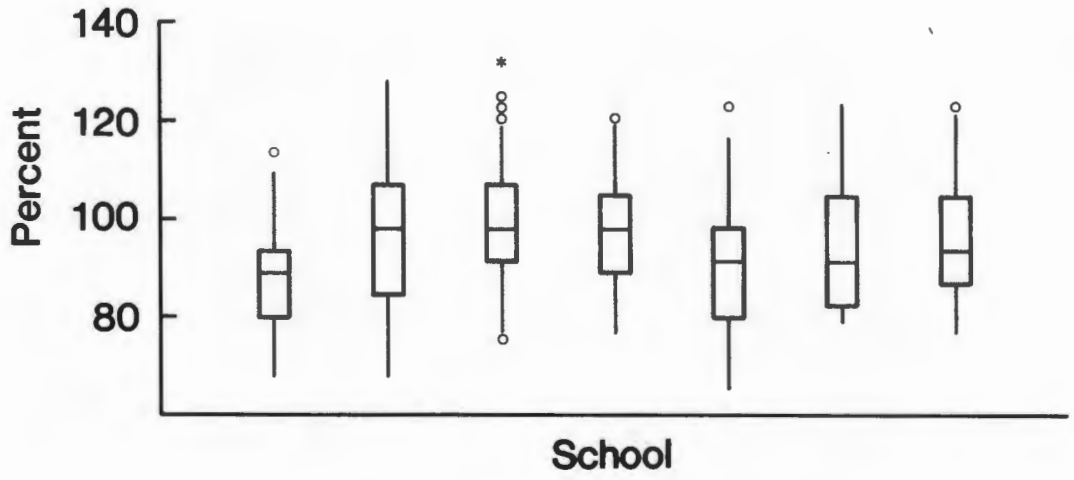


FIGURE 4.6b

PERCENT EXPECTED WEIGHT FOR AGE BY SCHOOL (BOX PLOTS)

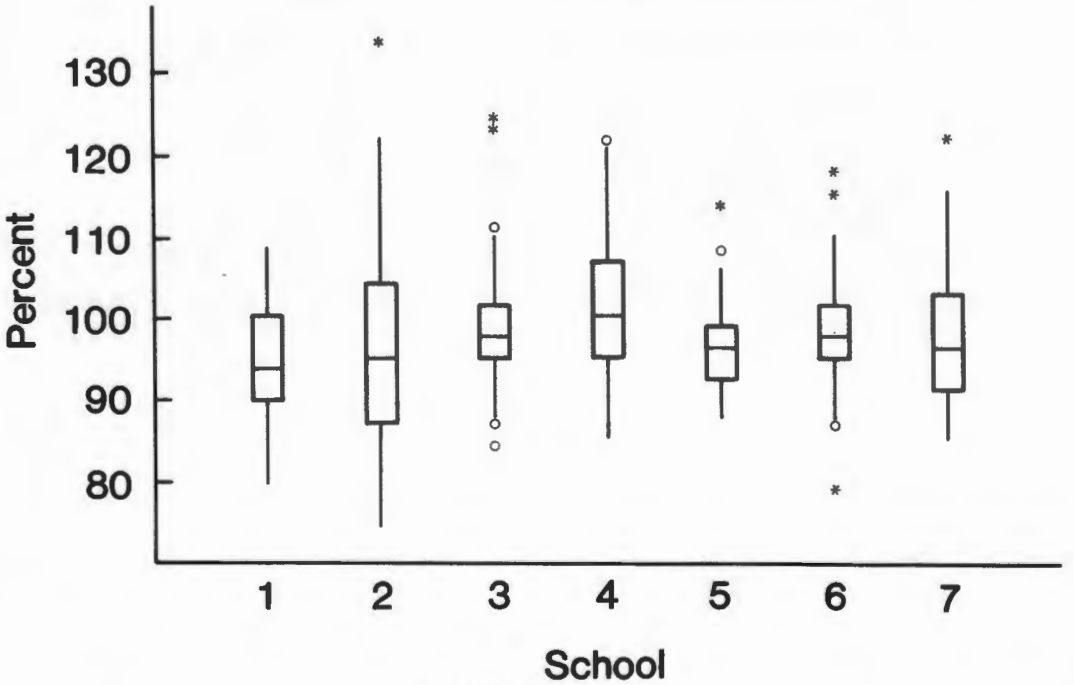


FIGURE 4.6c

PERCENT EXPECTED STANDARDISED WEIGHT BY SCHOOL (BOX PLOTS)

TABLE 4.14c

PERCENT EXPECTED WEIGHT FOR HEIGHT BY SCHOOL

	SCHOOL						
School	1	2	3	4	5	6	7
No.	15	19	25	29	21	20	17
Mean	94	96	100	102	97	99	98
Std. Dev.	8	13	9	12	7	9	9
Median	93	94	98	100	96	98	97

When schools were divided into two groups on the basis of the residential zones where children lived (see Section 4.1.4), no significant differences in nutritional status between the groups were found.

TABLE 4.14d

PERCENT EXPECTED HEIGHT FOR AGE BY SCHOOL GROUP (COLOURED PUPILS)

	Group 1 (Schools 2, 5, 6)	Group 2 (Schools 1, 7)
No.	61	32
Mean	99	97
Std. Dev.	6	3
Median	98	98
% < 90	3	3

TABLE 4.14e

PERCENT EXPECTED WEIGHT FOR AGE BY SCHOOL GROUP (COLOURED PUPILS)

	Group 1 (Schools 2, 5, 6)	Group 2 (Schools 1, 7)
No.	62	32
Mean	95	92
Std. Dev.	14	12
Median	94	90
% < 80	10	12

TABLE 4.14f

PERCENT EXPECTED WEIGHT FOR HEIGHT BY SCHOOL GROUP (COLOURED PUPILS)

	Group 1 (Schools 2, 5, 6)	Group 2 (Schools 1, 7)
No.	60	32
Mean	97	96
Std. Dev.	10	9
Median	96	94

4.3.1 RELATIONSHIP OF BLOOD LEAD LEVELS TO NUTRITIONAL STATUS

Correlations performed between the three nutritional parameters, age-standardised height, weight, weight for height and blood lead measurements were not statistically significant among either the white or coloured group (Table 4.15). The scatter plots of these relationships are given in Appendices B3 and B4.

TABLE 4.15a

THE RELATIONSHIP BETWEEN STANDARDISED HEIGHT AND BLOOD LEAD CONCENTRATIONS (ug/dl)

	All Pupils	Coloured Pupils	White Pupils
No.	147	93	54
Corr. Coeff(r)	-.10863	.02152	.19114
"p" Value	.1903	.8378	.1662

TABLE 4.15b

THE RELATIONSHIP BETWEEN STANDARDISED WEIGHT AND BLOOD LEAD CONCENTRATIONS (ug/dl)

	All Pupils	Coloured Pupils	White Pupils
No.	148	94	54
Corr. Coeff(r)	-.14415	-.03341	-.14210
"p" Value	.0805	.7492	.3054

TABLE 4.15c

THE RELATIONSHIP BETWEEN STANDARDISED WEIGHT FOR HEIGHT AND BLOOD LEAD CONCENTRATIONS (ug/dl)

	All Pupils	Coloured Pupils	White Pupils
No.	146	92	54
Corr. Coeff(r)	-.02991	.01067	.12404
"p" Value	.7201	.9196	.3715

Overall therefore, the nutritional status of both white and coloured children was satisfactory and was not related to blood lead levels. Similar findings relating to children's nutritional

status and blood lead levels were made with respect to a group of Cape pre-school coloured children studied by Deveaux et al. (1986).

4.4 SUMMARY

This part of the study has shown that significant variations in blood lead concentrations occurred between the white and coloured ethnic groups, and between schools and residential zones. There were no significant abnormalities in nutritional status or haematological parameters between the groups and these parameters were not related to blood lead concentrations. Descriptive profiles of the ethnic groups are compared and contrasted in the first part of the next chapter, which deals with the results of the questionnaire analyses.

CHAPTER 5

RESULTS AND DISCUSSION: CROSS SECTIONAL ANALYTICAL STUDY (PART B)

5.1 QUESTIONNAIRE DATA

The overall response rate for the questionnaire was 73% (80% for the coloured group and 63% for the white group). There was no statistically significant difference in the blood lead concentrations of the respondents versus the non-respondents, among both the white and coloured population, indicating that a significant selection bias was unlikely (see Sections 5.1.4 and 5.1.5).

Due to the large and highly significant difference in blood lead concentrations between the two population groups, it was decided to analyse the questionnaire separately for the two ethnic groups.

5.1.1 DESCRIPTIVE PROFILE OF WHITE PUPILS

(Appendix D2)

NATURE AND LOCATION OF THE CHILD'S GENERAL ACTIVITIES

21% of children went by car to school, whilst the majority (76%) walked. 50% of the respondents reported that their children walked to school along busy streets. 29% of children were reported to play mainly inside the house, 29% played mainly in the back yard and 26% played mainly in the front yard. Two children (five percent) played mainly in the street. Four children (10%) played often in busy streets.

PICA

Approximately a quarter of the children were reported to have been observed eating non-food items such as paint, cement, plaster, soil, sticks, matchsticks or cigarette ends. The majority were reported to have eaten non-food items occasionally (less frequently than once a week).

STATE OF HOUSE REPAIR

The average age of the homes of the children was 54 years. 28% of children lived in houses with a large amount of flaking paint outside and 18% lived in houses with a large amount of flaking paint inside. 38% of respondents reported that their homes were

in need of major repairs. 23% lived in homes with an outside toilet.

HOUSE DUST

13% of the respondents described their homes as being often dusty, 61% said their homes were often slightly dusty, whilst 26% said their homes were not dusty.

WATER PIPES

53% of respondents reported that their homes had copper pipes, nine percent said their homes had galvanised iron pipes, whilst 29% of respondents did not know what type of water piping was present in their homes. 38% of respondents reported that their pipes had been replaced and 38% did not know whether or not they had been replaced. Most pipes had been replaced within the last two years.

ELECTRICITY, SMOKERS

All of the respondents used electricity in the home, whilst 23% in addition used gas and eight percent wood. The median number of smokers in the home was two.

FACTORIES

41% of the respondents reported that they lived within five minutes walking distance from a factory. 32% and 26% of respondents said they lived within two minutes walking distance of petrol garages and workshops respectively.

DIETARY COMPOSITION

The majority of respondents (74%) reported that their children ate tinned food only occasionally or never. 79% of children ate fresh meat between five and seven days a week, whilst 70% ate fresh vegetables between five and seven days a week. 66% of children ate fresh fish once a week. 55% ate fresh fruit every day, whilst 48% ate sweets every day. 78% of respondents used aluminium pots for cooking and 38% used steel pots. 11% used pottery dishes for storing and cooking food.

SOCIO-DEMOGRAPHIC, CULTURAL AND ECONOMIC FACTORS

69% of children spoke English at home, 13% spoke English and Afrikaans at home, whilst 15% spoke Portuguese and English at home. All children were of the Christian faith.

The median total family income was R800-00 per month. 90% of the fathers and 82% of the mothers had completed six or more

years of schooling. The median number of people per household was five. The median number of rooms per household was four. 27% of respondents owned their homes, and 21% of children lived in families without their father.

The descriptive profile of white children is contrasted with that of coloured children in the next section, and the significance of the results is discussed in Section 5.13.

5.1.2 DESCRIPTIVE PROFILE OF COLOURED PUPILS

(Appendix D3)

Tables of results pertaining to this section are included in Appendix D3. A brief account of findings is given in the following sections.

NATURE AND LOCATION OF THE CHILD'S GENERAL ACTIVITIES

30% of children went by car to school, five percent went by bus or train, and the majority of children (65%) walked to school. 61% of respondents reported that their children walked to school along busy roads. 46% of the children were reported to play mostly indoors, 20% played mostly in the back yard, 19% mainly in the front yard, and 11% in the street. 23% of the respondents reported that their children often played in busy streets.

PICA

Seven children (nine percent) were reported to have been observed eating non-food items such as paint, cement, plaster, soil, sticks, matchsticks or cigarette ends. Most were reported to have eaten non-food items only occasionally (less frequently than once a week).

STATE OF HOUSE REPAIR

The average age of the children's homes was 52 years. 32% of the houses were reported to have a large amount of flaking paint outside, while 23% had a large amount of paint flaking inside. 32% of respondents reported that their houses needed major repairs. 57% lived in homes with an outside toilet.

HOUSE DUST

29% of the respondents reported that their homes were often very dusty, 35% said their homes were slightly dusty and 36% said their homes were not dusty.

WATER PIPES

64% of children lived in homes which had internal copper water pipes, 15% lived in homes which had galvanised iron pipes, and

three percent (two children) lived in homes which had lead water pipes. 25% of respondents reported that their water pipes had been replaced (the majority within the last three years) and no respondents reported that the original pipes were lead, although some parents did not know.

ELECTRICITY, SMOKERS

The majority of respondents (93%) had electricity in the home, but 21% used paraffin regularly, 47% used gas, and 11% used coal. The average number of smokers in the home was two.

FACTORIES

60% of the respondents reported that they lived within five minutes walking distance from a factory. 39% and 42% of respondents lived within two minutes walking distance of petrol garages and workshops respectively.

DIETARY COMPOSITION

The majority of the respondents (67%) reported that their children ate tinned food only occasionally or never. Six children (eight percent) ate tinned food every day. The majority of children (71%) ate fresh meat between five and seven days a week. 48% ate fresh vegetables between five and seven

days a week. The majority of the children (74%) ate fresh fish once a week, fresh fruit every day and, similarly, sweets every day. 54% of the respondents used steel pots for cooking, 59% used aluminium pots. Seven respondents (nine percent) used pottery dishes for storing or cooking food.

SOCIO-DEMOGRAPHIC, CULTURAL AND ECONOMIC FACTORS

48% of children spoke English at home. 24% were Afrikaans speaking and 27% spoke both English and Afrikaans at home. 37% of children were of the Christian faith; the rest were of the Islamic faith.

The median total family income of the population was R520-00 per month. 71% of fathers and 74% of mothers had completed six or more years at school. The median number of people per household was six and the median number of rooms per household was three. 26% of respondents owned their homes, and 30% of the children lived in single parent families without their father.

5.1.3 SUMMARY AND DISCUSSION: POPULATION CHARACTERISTICS (Table 5.1)

There were no major differences between white and coloured pupils with respect to the nature and location of the child's general activities, apart from the fact that among white pupils,

approximately the same numbers of children played inside the house as played in the back/front yards. Among coloured children on the other hand, the largest proportion played inside the house.

With regard to pica, more white than coloured children were reported to have been observed eating non-food items, but in the majority of cases, the frequency was less than once a week. Due to the poor response rate in questions relating to pica however, results should be treated with caution. It should be noted that the assessment of pica is difficult, especially by questionnaire. The difference in rates of pica reported by whites and coloureds may reflect the fact that in the one population group, parents were more likely to have observed this condition than in the other group. Evidence from other work suggests that the prevalence of pica among white children is lower than that of other population groups (Barltrop, 1966). In this study, in most cases, children were not regularly observed eating non-food items, indicating that whilst ingestion of non-food items occurred, pica in the clinical sense of the term may not have been present. In a study of the prevalence of pica by Barltrop (1966), it was estimated that at the age of six years, approximately 15 to 20% of children have pica. Rates among younger children are much higher.

There were no major differences with respect to the reported state of housing and prevalence of flaking paint among coloured

and white pupils: approximately the same percentage (32 vs 38%) of coloureds and whites reported that their homes were in need of major repairs. The interpretation of the term "in need of major repairs" is a relative and subjective one however, and possibly was interpreted differently by the two groups. Almost twice as many coloureds as whites , lived in homes with outside toilets, indicating a relatively low standard of housing among certain sectors of the coloured population. The homes of coloureds were also dustier than whites : 29% vs 13% said their homes were often very dusty. Most homes had copper pipes in both groups, but in many cases respondents were uncertain as to what kind of pipes they had. More coloureds than whites lived close to factories.

There were minor differences in dietary composition among coloureds and whites; the majority in both cases reported that their children ate tinned food only occasionally. White children ate more fresh vegetables and coloured children more fruit. Significantly more coloured children ate sweets regularly. It should be emphasised that the assessment of dietary habits by questionnaire is difficult; the results should therefore be interpreted with caution.

With regard to socio-demographic, cultural and economic factors, more whites than coloureds spoke English and a small percentage spoke Portuguese. All whites were of the Christian faith whereas the majority of coloureds were of the Islamic faith. Income levels differed significantly between the two groups, although

both population groups were primarily working class. The median total family income/month among whites and coloureds was R800 and R520 respectively. The level of schooling was better among whites than coloureds. Whites also lived in larger homes with fewer inhabitants, and just over a quarter of both groups owned their homes.

In general therefore, it is evident from the descriptive profiles of the population groups that whites differed from coloureds with respect to socio-demographic, cultural and economic factors (Table 5.1). These differences could have influenced the nature of responses to certain questions. Interpretation of different responses between population groups should therefore be treated with caution.

Differences in overall socio-economic status are likely to be important factors differentiating the blood lead distributions of the two population groups. These may in turn be related to environmental factors. Whilst differences in nutritional status and other haematological factors were noted among the groups (Chapter 4), these were minor and unlikely to be of overriding significance in influencing blood lead concentrations. Other unmeasured biological parameters may however, be significant. Base-line exposure to lead in air and dust in the environment at large is not thought to be an important factor as there were no major differences in environmental lead in the zones where

coloured and white children lived. (Lead levels in the environment are discussed in Chapter Six).

TABLE 5.1

POPULATION CHARACTERISTICS

Variable	Percentage of Population Coloureds	Whites
Play-Site		
Inside	46	29
Back yard	20	29
Front yard	19	26
Street	11	5
Play Busy Streets	23	10
Outside Toilet	57	23
House Dust		
Very Dusty	29	13
Factories		
(5 mins. walking distance)	60	41
Home Language		
English	48	69
Religion		
Christian	37	100
Father's Schooling		
(6 or more yrs completed)	71	90
Mother's Schooling		
(6 or more yrs completed)	74	82
Total Family Income		
(Rands/Month)	520	800
Single Parent Family	30	21

The risk factors for lead exposure, as assessed by the questionnaire, are discussed in the next section for the white and coloured children respectively.

5.1.4 RISK FACTORS FOR LEAD EXPOSURE AMONG WHITE PUPILS

In this section, blood lead levels are examined in relation to the various items contained in the questionnaire.

As previously stated, there was a response rate of 63% for the questionnaire. The mean blood lead levels of the respondents and the non-respondents are given in Table 5.2. There was no significant difference in the blood lead levels of the two groups. It is therefore unlikely that the non-response would have resulted in a significant selection bias.

TABLE 5.2

BLOOD LEAD CONCENTRATIONS (ug/dl)
(RESPONDENTS VS NON-RESPONDENTS)
(WHITE PUPILS)

	Respondents	Non-Respondents
No.	41	24
Mean	11	12
Std. Dev.	5	5
Median	10	11

Due to the relatively low response rate overall, and the small absolute number of respondents, statistical analyses were only performed on those variables with a sufficient number of responses per category. These variables and results are given in Appendix D4.

Significant differences in blood lead concentration were found with respect to the nature of the child's play site. For instance, those children who played mainly inside the house or in the back yard had lower blood lead levels than children who played in the front yard or elsewhere (eight, nine ug/dl vs 13 ug/dl respectively, p = 0.0350).

TABLE 5.3

PLAY-SITE (Q A15)

	Inside House	Back Yard	Front Yard	Garage/ Carport
No	11	11	9	6
Mean (ug/dl)	8	9	13	13
Std. Dev.	4	5	4	4
Median	9	7	12	14
Stat. Test.	Kruskal-Wallis			
"p" Value	.0350			

Significant differences in blood lead concentration were also found with respect to the frequency of consumption of the items fresh fish and cool drinks. Children who ate fresh fish more

often or who drank cooldrinks more regularly had lower blood lead levels than others (p = 0.0389, 0.0143 respectively).

TABLE 5.4

FRESH FISH (DAYS/WEEK) (Q B2)

	0-1	2-7
No	20	8
Mean (ug/dl)	12	7
Std. Dev.	5	6
Median	11	4
Stat. Test.	Mann-Whitney	
"p" Value	.0389	

TABLE 5.5

COOLDRINK (DAYS/WEEK) (Q B2)

	0-6	7
No	15	11
Mean (ug/dl)	12	7
Std. Dev.	5	4
Median	11	7
Stat. Test.	Mann-Whitney	
"p" Value	.0143	

With respect to behavioural factors, children who were described as being more active than other children had higher blood lead levels than others (19 vs 15 ug/dl, p = 0.0059). There were no significant differences in blood lead concentration with respect to the other items tested in the questionnaire.

TABLE 5.6

MORE ACTIVE THAN OTHERS (Q A22)

	Yes	No
No	9	22
Mean (ug/dl)	15	9
Std. Dev.	5	4
Median	13	9
Stat. Test.	Mann-Whitney	
"p" Value	.0059	

The results indicate that certain behavioural patterns of children relating to the nature of their play-site, may be of significance in influencing blood lead levels. Certain dietary items were also found to be of significance, but these may relate to other social factors rather than to exposure. They may also be an artefact of the multiple testing carried out.

The finding that children who were more active than others had higher blood lead levels was of interest, but it is not possible to comment on the temporal direction of the association. It should also be noted that this response by parents is subjective and is not a clinical finding.

5.1.5 RISK FACTORS FOR LEAD EXPOSURE AMONG COLOURED PUPILS

The questionnaire data in this section of the survey was analysed in two ways. First, each variable in the questionnaire was

analysed in relation to blood lead concentrations. Secondly, a multivariate analysis of certain significant variables was carried out.

5.1.5.1 Univariate Analysis of Questionnaire Data

As previously stated, there was an 80% response rate for the questionnaire among the coloured population. There was no statistically significant difference in the blood lead levels of the non-respondents to the questionnaire versus the respondents, as is shown in Table 5.7.

TABLE 5.7

BLOOD LEAD CONCENTRATIONS (ug/dl)
(RESPONDENTS VS NON-RESPONDENTS)
(COLOURED PUPILS)

Respondents		Non-Respondents
No.	83	21
Mean	18	19
Std.Dev.	6	5
Median	17	19

Individual questions which had a response rate between 20% and 80% were used to test for differences in blood lead levels between the respondents (to individual questions) and the non-respondents. Appendix D5 lists the variables (and results) for which statistically significant differences existed at the 5%

significance level between the blood lead concentrations of the two groups. In most cases the non-response group had higher blood lead levels than the response group. This may have decreased the chance of finding real associations. It was felt that the low number and diverse nature of the items for which differences existed did not represent a significant bias or necessitate special action.

Certain variables were not considered for further analysis as the majority of responses were in one direction, or the number of responses was too small. Those variables excluded from the analysis are indicated in Appendix D6.

The results of cross tabulations and correlation analyses carried out on the questionnaire are given in Appendix D7 and in the text. Table 5.8 lists those discrete and continuous variables for which statistically significant differences in blood lead levels existed for the different categories of the respective variables.

TABLE 5.8

LISTING OF SIGNIFICANT VARIABLES
(UNIVARIATE ANALYSES)

VARIABLE	"p" VALUE	
Occ. Density	.0001	
Father's Income (Q D9)	.0001	
Tot. Fam. Inc./No. Dependants	.0001	
Father's Schooling (Q D8)	.0011	
Home Language (Q A8)	.0011	
Factories (Q C24)	.0011	Significant at
Total Family Income (Q13)	.0015	1% Sig. Level
Religion (Q B3)	.0018	
Weight Loss (Q A37)	.0018	
Tinned Veg (Q B1)	.0036	
Mother's Schooling (Q D11)	.0062	
Home Owned (Q C1)	.0090	
Aluminium Pots (Q B6)	.0197	
Parental Care (Q D6)	.0255	
Health Effects (Q B36)	.0263	
House-Dust (Q C12)	.0280	
Tinned Food (Q B1)	.0290	
Major Repairs Needed (Q C22)	.0305	Significant at
No. of Siblings (Q D3)	.0319	5% Sig. Level
No. of Sleeping Rooms (Q C6)	.0321	
No. of People (Q D1)	.0347	
Creche Attendance (Q A20)	.0358	
Fresh Meat (Q B2)	.0393	
Sunlight (Q C8)	.0408	
Mealie meal (Q B2)	.0459	
Play-Site (Q A15)	.0704	
Loss of Appetite (Q A37)	.0736	
Petrol Garages (Q C28)	.0761	(Significant
No. of Rooms (Q C5)	.0784	at 10% Level)
Mother's Income (Q D12)	.0794	
Cheese (Q B2)	.0807	
Overactive (Q A23)	.0884	

i) NATURE AND LOCATION OF THE CHILD'S GENERAL ACTIVITIES

As was earlier indicated (Section 4.14), there was a highly

significant difference in the blood lead levels of children attending different schools, with average (median) blood lead levels at schools ranging from 13 to 21 ug/dl.

There was no significant difference in the mean blood lead levels of children who walked to school compared to children who went by car, bus or train ($p = 0.2312$). Children who often played in busy streets had higher blood lead levels than those who did not (19 vs 17 ug/dl), but the difference was not statistically significant ($p = 0.2496$). Children playing inside or in the front yard had lower blood lead levels than children playing in the back yard or in the street (16 and 17 ug/dl vs 20 and 21 ug/dl). There was nevertheless no significant difference in the blood lead concentrations at the 5% significance level ($p = 0.0704$).

ii) PICA

The mean blood lead levels of children who were observed at some stage to have eaten one or more non-food items (in most cases reportedly less frequently than once a week) had higher blood lead levels than other children (21 vs 17 ug/dl), but this difference did not reach statistical significance ($p = 0.1025$).

iii) HOUSING

There was no statistically significant correlation between the

age of children's homes and blood lead levels (p = 0.6137). Children from homes which had a significant degree of flaking paint on the exterior surfaces had higher blood lead levels than other children (20 vs 16 ug/dl); this difference however was not statistically significant (p = 0.1495). Similarly blood lead levels of children from homes with significant levels of flaking paint indoors had higher blood levels than others (19 vs 17 ug/dl) but this difference was also not statistically significant (p = 0.8053). Children from homes which were in need of major repairs had higher blood lead levels than children from homes in a satisfactory state of repair (20 vs 16 ug/dl); this difference was statistically significant (p = 0.0305).

TABLE 5.9

MAJOR REPAIRS NEEDED (Q C22)

	Yes	No
No.	24	43
Mean (ug/dl)	20	16
Std. Dev.	7	5
Median	18	16
Stat. Test	Median	
"p" value	.0305	

Children from homes with an outside toilet had on average higher blood lead levels than those with inside toilets (19 vs 16 ug/dl); this difference did not reach statistical significance (p = 0.1120).

There was a statistically significant difference in the mean blood lead levels of children whose homes were described as 'very dusty' compared with those whose homes were not very dusty. The mean blood lead levels in the two groups were 21 and 16 ug/dl respectively (p = 0.0280).

TABLE 5.10

HOUSE DUST (Q C12)

	Very Dusty	Slightly Dusty	Not Dusty
No.	24	29	30
Mean (ug/dl)	21	16	16
Std. Dev.	7	5	5
Median	19	17	15
Stat. Test	Kruskal-Wallis		
"p" value	.0280		

Blood lead levels of children living in the vicinity of factories were higher than other children (19 vs 15 ug/dl), as were the blood lead levels of children living in the vicinity of petrol garages compared with others (20 vs 16 ug/dl). The difference in mean blood lead levels in the first case was statistically significant (p = 0.0011), but not in the second case (p = 0.0761). The blood lead levels of children living close to workshops were also higher than other children (19 vs 17 ug/dl), but this difference was not statistically significant (p = 0.5189).

TABLE 5.11

FACTORIES (Q C24)

	Yes	No
No.	50	32
Mean (ug/dl)	19	15
Std. Dev.	7	5
Median	18	15
Stat. Test	Median	
"p" value	.0011	

With respect to fuel sources in the home, children from homes where paraffin was used had higher blood lead levels than others (21 vs 17 ug/dl) but this difference was not statistically significant (p = 0.1306). Children from homes with a lot of sunlight entering the house had lower blood lead levels than those with little sunlight (17 vs 21 ug/dl). This difference was statistically significant (p = 0.0408). There was no significant difference in the mean blood lead levels of children from homes with copper piping, compared to children from homes with other types of piping (p = 0.7564).

iv) DIETARY COMPOSITION

Children who ate tinned food at least once a week had higher blood lead levels than children who ate tinned food occasionally or never (20 vs 16 ug/dl). This difference was statistically significant (p = 0.0290).

TABLE 5.12

TINNED FOOD (Q B1)

	1 Or More Days/Week	Occasionally/ Never
No.	24	50
Mean (ug/dl)	20	16
Std. Dev.	7	6
Median	18	17
Stat. Test	Mann-Whitney	
"p" value	.0290	

There were also significant differences with respect to other components of the diet, such as the frequency of fresh meat, tinned vegetables and mealie meal consumed.

TABLE 5.13

FRESH MEAT (Q B2)

	1-6 Days/Week	7 Days/Week
No.	33	32
Mean (ug/dl)	16	18
Std. Dev.	6	5
Median	14	17
Stat. Test	Mann-Whitney	
"p" value	.0393	

TABLE 5.14

TINNED VEGETABLES (Q B2)

	0-1 Day/Week	2-7 Days/Week
No.	25	11
Mean (ug/dl)	15	22
Std. Dev.	5	5
Median	16	22
Stat. Test	Mann-Whitney	
"p" value	.0036	

TABLE 5.15

MEALIE-MEAL (Q B2)

	Never	1-7 Days/Week
No.	20	11
Mean (ug/dl)	15	20
Std. Dev.	5	5
Median	15	17
Stat. Test	Mann-Whitney	
"p" value	.0459	

With respect to the type of pots used in cooking, those children from homes where aluminium pots were used had significantly higher blood lead levels than children from homes where other pots were used (19 vs 16 ug/dl, p = 0.0197).

TABLE 5.16

ALUMINIUM POTS (Q B6)

	Yes	No
No.	46	32
Mean (ug/dl)	19	16
Std. Dev.	7	5
Median	18	16
Stat. Test	Mann-Whitney	
"p" value	.0197	

v) SOCIO-DEMOGRAPHIC, CULTURAL AND ECONOMIC FACTORS

There was a statistically significant difference in the blood lead levels between English and Afrikaans speakers, with mean blood lead levels being lower among the English speakers (16 vs 22 ug/dl, p = 0.0011).

TABLE 5.17

HOME LANGUAGE (Q A8)

	English	Afrikaans	Eng & Afr
No.	40	20	23
Mean (ug/dl)	16	22	16
Std Dev.	6	6	5
Median	16	21	16
Stat. Test	Kruskal-Wallis		
"p" Value	.0011		

There was also a statistically significant difference in the blood lead levels among children of different religions: Muslim children had mean blood lead levels between 13 and 16 ug/dl, and children of the Christian faith had mean blood lead levels of 20 ug/dl (p = 0.0018).

TABLE 5.18

RELIGION (Q B3)

	Islam	"Malay" (Muslim)	Christian
No.	16	28	26
Mean (ug.dl)	13	16	20
Std. Dev.	6	5	6
Median	12	16	18
Stat. Test	Kruskal-Wallis		
"p" value	.0018		

There was a statistically significant inverse relationship between blood lead and the variables; number of rooms used for sleeping (r = -0.23984, p = 0.0321); father's income (r = -0.56448, p = 0.0001); total family income (r = -0.48046, p = 0.0015) and income (taking into account the number of dependants) (r = -0.58321, p = 0.0001). A statistically significant positive association was found between blood lead and the variables; number of people living in the home (r = 0.23953, p = 0.0347); number of siblings (r = 0.24012, p = 0.0319); occupational density (r = 0.45786, p = 0.0001).

Children whose homes were owned by their parents had significantly lower blood lead levels than children whose homes were rented (14 vs 19 ug/dl, p = 0.0090).

TABLE 5.19

HOME OWNED(Q C1)

	Yes	No
No.	22	62
Mean (ug/dl)	14	19
Std. Dev.	6	6
Median	13	18
Stat. Test	Mann-Whitney	
"p" value	.0090	

Children who lived with both parents had lower blood lead levels than children from single parent families who did not live with their father (16 vs 21 ug/dl, p = 0.0255).

TABLE 5.20

PARENTAL CARE (Q D6)

	Two Parents	One Parent
No.	56	24
Mean (ug/dl)	16	21
Std. Dev.	6	7
Median	16	21
Stat. Test	Median	
"p" value	.0255	

Children whose fathers or mothers had completed seven years of schooling or more had lower blood lead levels than children whose parents had up to six years' schooling (20, 21 ug/dl vs 15 ug/dl for the respective groups).

TABLE 5.21

FATHER'S SCHOOLING (Q D8)

	Up To 6 Years	More Than 6 Years
No.	36	33
Mean (ug/dl)	20	15
Std. Dev.	6	5
Median	19	14
Stat. Test	Mann-Whitney	
"p" value	.0011	

TABLE 5.22

MOTHER'S SCHOOLING (Q D11)

	Up To 6 Years	More Than 6 Years
No.	40	36
Mean (ug/dl)	21	15
Std. Dev.	7	5
Median	20	16
Stat. Test	Median	
"p" value	.0062	

These differences were in both cases statistically significant ($p = 0.0011$, $p = 0.0062$). Children who had attended creches had significantly lower blood lead levels than those who had not attended creches (16 vs 19 ug/dl, $p = 0.0358$).

TABLE 5.23

CRECHE ATTENDANCE(Q A20)

	Yes	No
No.	41	43
Mean (ug/dl)	16	19
Std. Dev.	6	6
Median	16	18
Stat. Test	Mann-Whitney	
"p" value	.0358	

vi) BEHAVIOURAL AND MEDICAL TRAITS

Children who were described by respondents as being very active prior to school age, or who were more active than others (84% and 29% of the population respectively) had higher blood lead levels than other children (18, 19 ug/dl vs 16 ug/dl) but these differences were not statistically significant (p = 0.2254, 0.2097 respectively). Children who were described by their parents as being overactive (18% of the population) similarly had higher blood lead levels than others (20 vs 16 ug/dl). This difference was not statistically significant at the 5% level however (p = 0.0884). There was no significant difference in the blood lead levels among children who were described as being particularly active at present (p = 0.4872) or among children who had difficulty concentrating (p = 0.7003) or were easily

distracted (p = 0.5486), compared with other children. Children repeating first grade had higher blood lead levels than other children (20 vs 17 ug/dl) but this difference was not statistically significant (p = 0.1609).

Children who had at some time suffered from poor speech or who tired easily had higher blood lead levels than those who did not (19 vs 17 ug/dl respectively for the two groups), but these differences were not statistically significant (p = 0.1683, 0.2824). Children who had exhibited a recent loss of appetite had higher blood lead levels than other children (19 vs 16 ug/dl, p = 0.0736) and children who had had a recent loss of weight had higher blood lead levels than those who had not (22 vs 16 ug/dl). This difference was statistically significant (p = 0.0018).

TABLE 5.24

WEIGHT LOSS (Q A37)

	Yes	No
No.	15	48
Mean (ug/dl)	22	16
Std. Dev.	6	6
Median	22	16
Stat. Test	Mann-Whitney	
"p" value	.0018	

There was also a significant difference between blood lead levels in children who exhibited one or more ill-health effect

potentially indicative of excess lead absorption, compared to children who did not (20 vs 17 ug/dl, p = 0.0263).

TABLE 5.25
HEALTH EFFECTS (Q A36)

	1 or More Health Effects	No Health Effects
No.	29	57
Mean (ug/dl)	20	17
Std. Dev.	7	5
Median	19	17
Stat. Test	Mann-Whitney	
"p" value	.0263	

vii) SUMMARY

This part of the statistical analysis for coloured children showed that there were several environmental factors (state of repair of the home, dustiness of the home), socio-demographic factors (home language, religion, crowding, income) as well as certain dietary factors (tinned foods and other individual items) that were of importance in influencing blood lead levels. Due to the possibility of confounding, further multivariate analyses were carried out on the data. These are discussed below. With respect to certain medical factors which were found to be related to blood lead levels, it is not possible to comment on the temporal direction of the associations. Also, confounding of factors could have resulted in spurious associations.

5.1.5.2 Multivariate Analyses

In order to obtain further clarification of the data, the associations and relationships between variables significant at the one percent level were tested. Associations and relationships which were significant at the one percent level are listed in Appendix D8.

To obtain a clearer picture, multivariate analysis was considered. Those statistically significant variables potentially indicative of exposure were selected for further analysis adjusting for the effect of socio-economic/demographic factors. Due to missing values, logistic regression was not considered appropriate. This would have provided estimates of the contributions that particular variables would have made to the overall variability in blood lead. Multiway tables were used, analysing the relationship between environmental factors against blood lead, having adjusted for the effects of socio-economic/demographic factors. The variable occupational density was selected as being the strongest socio-demographic indicator among the continuous variables measured (this value was categorised as values less than or equal to the median and those above the median). In addition the variable father's schooling

(significant at the one percent level) was selected from the discrete variables to indicate socio-economic standing.

Variables potentially indicative of exposure to lead which were selected for entry into the multi-way tables, were those relating to the level of dust in the home, the proximity of factories to the home, the nature of repairs needed in the home, the frequency of tinned vegetables consumed, residential zone (as defined previously on the basis of geographic location) and school group (Section 4.1.4).

In each multi-way table, one of the above six variables potentially indicative of exposure to lead was selected, as well as the two variables indicative of socio-demographic and economic standing, in order to determine whether the 'exposure' variables were of significance taking into account socio-economic standing. The partial association was obtained between blood lead level and each of the respective three factors in the multi-way table, adjusting in the case of each factor tested for the effects of the associations between blood lead and the other two factors, and the associations between all three factors.

The results of the series of multi-way contingency table analyses carried out are given in Table 5.26.

TABLE 5.26a

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(Blood Lead (A), Father's Schooling (B), Occup. Density (C), Interior Dust (D))

EFFECT	CHISQUARE	"P" VALUE
AB	5.13	0.0236
AC	1.08	0.2996
AD	1.95	0.3767

TABLE 5.26b

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(Blood Lead (A), Father's Schooling (B), Occup.Density (C), Factories (D))

EFFECT	CHISQUARE	"P" VALUE
AB	4.79	0.0286
AC	0.56	0.4552
AD	6.36	0.0117

TABLE 5.26c

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(Blood Lead (A), Father's Schooling (B), Occup. Density (C), Home Repairs (D))

EFFECT	CHISQUARE	"P" VALUE
AB	4.95	0.0261
AC	0.64	0.4246
AD	2.76	0.0964

TABLE 5.26d

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(Blood Lead (A), Father's Schooling (B), Occup. Density (C), Residential Zone (D))

EFFECT	CHISQUARE	"P" VALUE
AB	3.20	0.0738
AC	1.34	0.2475
AD	7.79	0.0053

TABLE 5.26e

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(Blood Lead (A), School, (B), Occup. Density (C), Father's Schooling (D))

EFFECT	CHISQUARE	"P" VALUE
AB	14.72	0.0001
AC	0.27	0.6000
AD	3.44	0.0636

TABLE 5.26f

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(Blood Lead (A), Father's Schooling (B), Residential Zone (C), School (D))

EFFECT	CHISQUARE	"P" VALUE
AB	2.43	0.1194
AC	0.88	0.3487
AD	9.95	0.0016

(For the variable 'tinned vegetables' there were too many empty cells for a reliable analysis to be carried out). There were no significant three way interactions. There were significant partial associations between blood lead levels and the respective exposure variables factory, residential area and school, when controlling for socio-economic standing.

The two exposure variables with the smallest 'p' values, school and residential area, were entered into a multi-way contingency table with the variable father's schooling, to examine the partial associations between these variables (Table 5.26f). The partial association between blood lead level and school was significant, taking into account the effects of residential area and father's schooling. This finding is discussed in Section 5.1.6.5 and in Chapter Six.

5.1.6 DISCUSSION

RISK FACTORS

From the analysis of the questionnaire, risk factors related to certain environmental and socio-demographic / economic / cultural characteristics were determined. These differed for the two population groups studied. For white children, few associations

were found due to the relatively small population size and the smaller variation in blood lead as compared to coloured children.

The findings indicate that for coloured children living in the area social factors are significant predictors of blood lead. Due to the poor response rate for questions relating to social factors among whites, it is not possible to comment on their significance for this population group. As whites were more homogenous with respect to socio-economic standing however, it is likely that social factors were of less importance for this group.

5.1.6.1 Socio-economic and Demographic Factors

Social factors which were important for coloureds included socio-economic status and factors related to family structure. For instance, among coloured pupils, children from low socio-economic backgrounds had higher blood lead levels than other children. This was indicated by the significant correlations in blood lead with father's income and total family income, father's schooling and mother's schooling. Blood lead levels were inversely related to the relative level of affluence of the family and the educational level of the child's parents. Several other variables indicative of socio-economic status which did not reach statistical significance, were nevertheless in the same general direction.

Demographic factors related to the nature of the child's family also emerged as being of importance. For instance, a positive relationship was found between blood lead levels and occupational density of the household. Children who came from overcrowded homes had higher blood lead levels than other children. Other factors related to family structure such as single parent families and the number of siblings were also associated with raised blood lead levels.

There have been relatively few studies which have focussed on the social factors associated with childhood lead exposure (O'Hara, 1982). Whilst certain social factors have been known for a long time to be associated with blood lead levels, Stark et al., in an article published in 1978, drew attention to them. They looked at socio-demographic and economic factors in relation to blood lead levels in 8 334 New Haven children aged six years and below. In subsequent studies, social factors were found to be of more significance than environmental factors for some children (Stark et al., 1982 a,b). A factor such as having a single unemployed parent was not found to be significant for black or Hispanic children, but was highly significant for whites. Similarly, the number of siblings a child had was significant for whites but not for others. Creche attendance had a protective effect on blood lead level among whites but not for blacks. (In the present study, creche attendance was associated with lower blood lead levels among coloureds).

Stark et al. suggested that among American blacks, patterns of child care were not severely affected by factors such as economic level and family size; in contrast for whites supervision of the young child appeared to be modified by the nature of the structure of the child's family. It may have been the case, however, that because the blacks in Stark et al.'s study were in all likelihood from lower socio-economic backgrounds, there may not have been a sufficient range in social conditions within which to examine the effect of social factors. Of interest is the fact that there are similar findings with respect to coloureds in this study, vis a vis whites in Stark et al.'s study, despite the fact that blood lead levels were considerably higher in their study.

Many other studies have found blood lead levels to be correlated with, for instance, socio-economic status. In the United States for example, low socio-economic groups have been found to have higher blood lead levels than higher socio-economic groups. In the NHANES II study there was a very strong relationship between social class and blood lead levels across all age groups in the population. This relationship was most marked in children under six years of age. Family income was the variable chosen to represent socio-economic status in the NHANES II study. The mean blood lead concentration increased as family incomes decreased. For instance, the mean blood lead concentration (all races) was 20 ug/dl for families with an annual income < 6 000 dollars, 16.2

ug/dl for families with an income between 6 000 and 15 000 dollars, and 14 ug/dl for families with an annual income greater than 15 000 dollars.

The situation in the United Kingdom with regard to the relationship of blood lead levels with socio-economic status is less clear. In the EEC blood lead surveys, there was no consistent trend in blood lead concentrations among adults in the United Kingdom, after controlling for various factors. Among children, the majority studied were children of leadworkers or children living around leadworks, whose parents were manual workers. Their average blood lead concentrations were about one ug/dl higher than those of children of non-manual workers. Due to the fact that most of the children studied were of low socio-economic status, however, no firm conclusions can be drawn from the United Kingdom data.

In the light of the relationship between blood lead levels and socio/demographic and economic factors found in this study, it is interesting to note that Deveaux et al. (1986), in their study of 323 pre-school Cape children found no relationship between socio-economic status and blood lead levels. This may be due to the fact that the children studied were those attending nursery schools, who are likely to be of higher socio-economic status than non-nursery school attenders.

It is often assumed that children of lower socio-economic status are at higher risk due to the fact that they are more likely to reside in inner city areas where there is old housing with leaded paint, high density traffic, or lead water pipes. Whilst environmental factors are explored in more detail in the second part of the study, this part of the study has shown that demographic factors such as overcrowding are likely to play a role. This was indicated in the multivariate analysis, where among the continuous variables, occupational density was shown to be important. Overcrowding is associated with other negative social and economic factors, which in turn may impact on the parents ability to supervise children. It is suggested that social factors assume importance in predisposing children to lead in the environment. Should significant quantities of lead be present in the environment this part of the study shows that coloured children from low socio-economic backgrounds are at highest risk. This aspect is explored further in the case control study, Chapter Seven.

5.1.6.2 Cultural Factors

The child's religion and home language were also important determinants of blood lead levels. In this coloured community, both these factors are known to be related to socio-economic status (O'Toole, 1973; Ridd 1981). For instance, being English-speaking and/or of the Islamic faith are accorded higher socio-

economic status than being Afrikaans-speaking or of the Christian faith.

In this study, Afrikaans speakers had significantly higher blood lead levels than English speakers and Muslim children had lower blood lead levels than Christians. It is thought that cultural factors associated with religion may play a role in determining blood lead levels. In Woodstock, religion is the strongest social institution after the family (O'Toole, 1973). In the Muslim religion (Islam), personal bodily cleanliness is emphasised. This pertains to 'purity' while engaged in ritual practises such as prayer, as well as at other times (Denny, 1985). Personal hygiene may be of importance in influencing blood lead levels.

There is also a strong sense of closeness in the Muslim home, the family being the cornerstone and mainstay of the community (Farah, 1968). According to Denny (1985), the family acts as a bulwark against instability and chaos, providing protection and support and a setting for the proper care of children.

It is not thought that Muslim customs regarding the preparation and eating of food play a major role in influencing blood lead levels. All 'Halal' food that has been slaughtered properly is permitted. Only certain types of substances are forbidden, for example, carrion, blood, pork and 'anything sacrificed or dedicated to other than God' (Denny, 1985).

5.1.6.3 Home Environment

Sources of lead in the home environment were not quantitatively determined in this part of the study. Nevertheless, qualitative information indicated that certain factors related to the home environment were predictors of blood lead levels among the coloured population. Factors related to housing which were important included the state of repair of the home, the level of dust in the home and the proximity of factories to the home. Proximity of factories is not thought to be a major factor relating to exposure, as none of the factories in the area were major sources of lead emissions to the ambient air. The proximity of factories was in turn related to other factors.

Children from homes in a poor state of repair and children from dusty homes had higher blood lead levels than others. The level of dust in the home may be influenced by the state of repair of the home. Houses in a deteriorating state may have greater amounts of dust than in well-maintained homes. Lead concentration levels in the dust could derive from old flaking lead-based paint and/or from atmospheric fallout. In this part of the study the relationship between the amount of flaking paint and blood lead levels was not statistically significant, although children from houses with flaking paint had higher blood lead

levels than others. This aspect is explored in more detail in the case control study.

It was also found that children who came from homes which were owned by their parents had lower blood lead levels than children whose homes were rented. It is likely that homes which are owned are in a better state of repair than homes which are rented. Often there is a greater incentive and finances are more readily available to home owners, for the upkeep of their homes. Rented homes, on the other hand, are often owned by absentee landlords, many of whom have allowed the houses in this area (particularly the exteriors) to deteriorate.

Environmental factors such as the level of dust in the home and the state of repair of the home were not significant when socio-economic factors were taken into account. There is thus a high degree of confounding between social and environmental factors.

Water pipes were not a significant factor in the exposure of children to lead - the majority of homes had copper pipes, whilst in two homes only the presence of lead pipes was reported. There was nevertheless the possibility that the water pipes had lead soldered joints - this is not thought to be of much significance however, as the water supply in Cape Town is not plumbosolvent.

There was a relationship between the nature of the child's main play-site and blood lead levels, although this did not reach

statistical significance among coloured children. Among coloured children, playing mainly in the back yard or on the street was associated with higher blood lead levels, and playing inside or in the front yard was associated with low blood lead levels. For white children, playing mainly inside the house was protective, but playing mainly in the front yard was not. This relationship was statistically significant. The fact that playing inside the house was associated with lower blood lead levels in both population groups suggests that exposure to lead is of more significance for this age group outside the home environment than inside. It is likely that children playing inside the home are better supervised than children playing outside and that therefore fewer opportunities arise for exposure to lead sources in the home.

5.1.6.4 Diet

Specific dietary items were found to be associated with blood lead levels and significant differences in blood lead concentrations occurred with respect to the frequency of consumption of certain food items. The specific food items of importance differed for the white and coloured groups. Whereas among whites, blood lead levels were higher among those who regularly ate fresh fish and drank cooldrinks, among the coloureds the items of importance with respect to increased blood lead levels were mealie-meal, fresh meat and tinned foods. With

respect to tinned foods, it is known that lead is present in the solder in South African manufactured tins and that elevated levels of lead may occur in certain food items. It is likely that the significance of the other food items including perhaps, tinned foods, is in their relationship to socio-economic factors. There was also a relationship between the types of pots used for cooking and blood lead levels. This too, is likely to be the result of socio-economic factors, rather than of exposure.

5.1.6.5 School

In this study a significant difference in blood lead levels of children attending different schools was found. In order to determine whether there were factors such as differences in nutritional status at the schools, which could explain the observed phenomena, blood lead levels of coloured pupils at the schools were examined in relation to these factors.

No significant differences in overall nutritional status were found between schools grouped on the basis of their catchment areas.

There was also a relationship between the residential zone where children lived, the proximity of factories to the home and blood lead levels. For instance, children living in zone three had lower blood lead levels than children living in zones one and

two. This relationship held irrespective of socio-economic factors which were taken into account, although socio-economic factors were of more significance in influencing the blood lead levels of children in these zones. The zone where children lived, however, was not found to be significant when the school attended by the child was taken into account.

The factor 'school' remained significant irrespective of the socio-economic status of the children and residential zone when a contingency table analysis was carried out. There was no difference at schools with respect to flaking paint (all classrooms were in a state of good repair) but there was a strong association between blood lead levels and the geographic position of schools with respect to traffic density. In order to assess whether or not levels of lead in the environment were elevated, and could account for this association, it was necessary to undertake a detailed environmental study. The issues addressed in the environmental study are given in Section 3.6 of Chapter Three. Air and dust lead levels in the study area are discussed in the next part of the study and their relevance to children's exposure is discussed in Section 6.3.3.

CHAPTER 6

RESULTS AND DISCUSSION: ENVIRONMENTAL STUDY

In this chapter results pertaining to the environmental study are described and discussed : these include the air lead concentrations measured and the dust lead measurements made in the study area.

6.1 AIR LEAD CONCENTRATIONS

6.1.1 SPATIAL DISTRIBUTION OF AIR LEAD CONCENTRATIONS

In order to compare ambient lead levels among the sites, the average lead concentrations were determined for each site over the entire sampling period May 1983 to June 1984. The mean, standard deviation and median lead concentration levels at the sites in the area are given in Table 6.1. Figure 6.1 gives a graphical representation of the average lead levels at the sites.

It can be seen that considerable differences in lead concentration occur between sites. Annual means at sites varied threefold from less than 0.5 ug/m^3 to above two ug/m^3. The

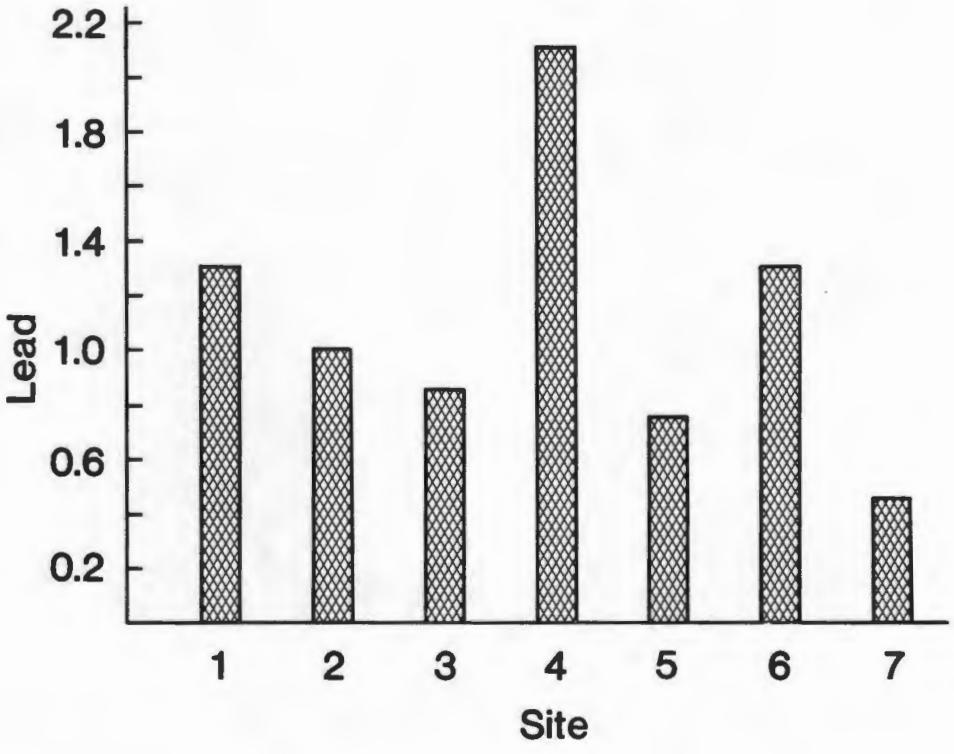


FIGURE 6.1

ANNUAL AIR LEAD CONCENTRATION (ug/m^3) BY SITE

average yearly lead concentration (all sites together) for the study area as a whole was 1.1 ug/m^3.

TABLE 6.1

ANNUAL AIR LEAD CONCENTRATION (ug/m^3) BY SITE

SITE	MEAN	STD. DEV.	MEDIAN
1	1.3	1.1	0.9
2	1.0	0.9	0.7
3	0.9	0.8	0.6
4	2.1	1.6	1.7
5	0.7	0.8	0.5
6	1.3	1.1	0.9
7	0.4	0.4	0.3
All	1.1	1.1	0.7

A one way analysis of variance was performed, which revealed that the variation in lead concentrations between sites was statistically significant (Table 6.2).

TABLE 6.2

VARIATION IN AIR LEAD CONCENTRATION (ug/m^3) BETWEEN SITES (ANALYSIS OF VARIANCE)

SOURCE	S.S	D.F	M.S	F-VALUE	TAIL PROBABILITY
Between groups	10.9955	5	2.1991	82.34	0.0000
Within groups	35.5471	1331	0.0267		
TOTAL	46.5426	1336			
Levene's Test for equal var.		5, 1331		6.95	0.0000
Welch		5, 585		90.00	0.0000
Brown-Forsythe		5, 1149		82.44	0.0000

Pairwise multi-comparisons were conducted (Table 6.3), and the Bonferroni probabilities for pairs of sites indicated that differences between all pairs were statistically significant at the five percent significance level, with the exception of sites one and six, and three and five.

TABLE 6.3

DIFFERENCES IN ANNUAL AIR LEAD CONCENTRATION (ug/m^3) BETWEEN SITES (BONFERRONI PROBABILITIES)

SITE					
3	0.0000*				
4	0.0000*	0.0000*			
7	0.0000*	0.0000*	0.0000*		
1	0.8502	0.0000*	0.0000*		
5	0.0000*	0.0216	0.0000*	0.0000*	0.0000*
SITE	6	3	4	7	1

(Anova results : F:82.34, D.F.:5, 1331, P:0.0000)
*significant difference between sites at 5% significance level

The traffic volumes measured at each site are given in Table 6.4.

TABLE 6.4

TRAFFIC VOLUMES AT SITES

SITE	TRAFFIC (cars/day)
1	17675
3	907
4	>20000
5	241
6	7752
7	260

Air lead concentrations at sites directly on major roads were significantly higher than at other sites. For example, average lead concentrations were approximately twofold higher at sites one, four and six (situated on major roads) than at sites two, three, five and seven (situated 200 metres to 300 metres away from major roads). The highest annual mean lead concentration (2.1 ug/m^3) was measured at site four which had the highest traffic volume. This site also registered the maximum daily air lead concentration which was 10 ug/m^3. Sites two, three, five and seven, with low traffic flows, had considerably lower lead concentrations (Table 6.5).

TABLE 6.5

ANNUAL AIR LEAD CONCENTRATION (ug/m^3) AT SITES ON AND AWAY FROM MAJOR ROADS

	Sites On Major Roads (1, 4, 6)	Sites Away From Major Roads (2, 3, 5, 7)
Mean	1.5	0.8
Std. Dev.	1.3	0.8
Median	1.2	0.5

A correlation analysis was performed which revealed a statistically significant relationship at the five percent level between air lead levels and traffic volumes (r=0.9429) (Table 6.6).

TABLE 6.6

RELATIONSHIP BETWEEN ANNUAL AIR LEAD CONCENTRATIONS (ug/m^3) AND TRAFFIC

SITE	AIR LEAD (ug/m^3)	TRAFFIC (cars/day)
1	1.3	17675
3	0.9	907
4	2.1	>20000
5	0.7	241
6	1.3	7752
7	0.4	260

Results of the three week monitoring periods at the coloured schools revealed that air lead levels at schools five and six,

situated in the immediate proximity of high density traffic (Figure 3.1) were higher than at schools situated away from traffic (Table 6.7).

TABLE 6.7

MEAN AIR LEAD CONCENTRATION (ug/m^3) AT COLOURED SCHOOLS

School				
1	2	5	6	7
0.25	0.45	0.80	0.80	0.30

6.1.2 TEMPORAL DISTRIBUTION OF AIR LEAD CONCENTRATIONS

6.1.2.1 Seasonal Variations and Air Quality Standards

Investigation of the temporal variation in the level of atmospheric lead shows the typical annual air pollution pattern, with lead concentrations falling to a minimum during the summer months and increasing considerably during the winter months (Figure 6.2). This trend occurred at all sites. An examination of differences in lead concentrations between seasons was performed, using an analysis of variance. (Summer months were taken as December to February, autumn as March to May, winter as

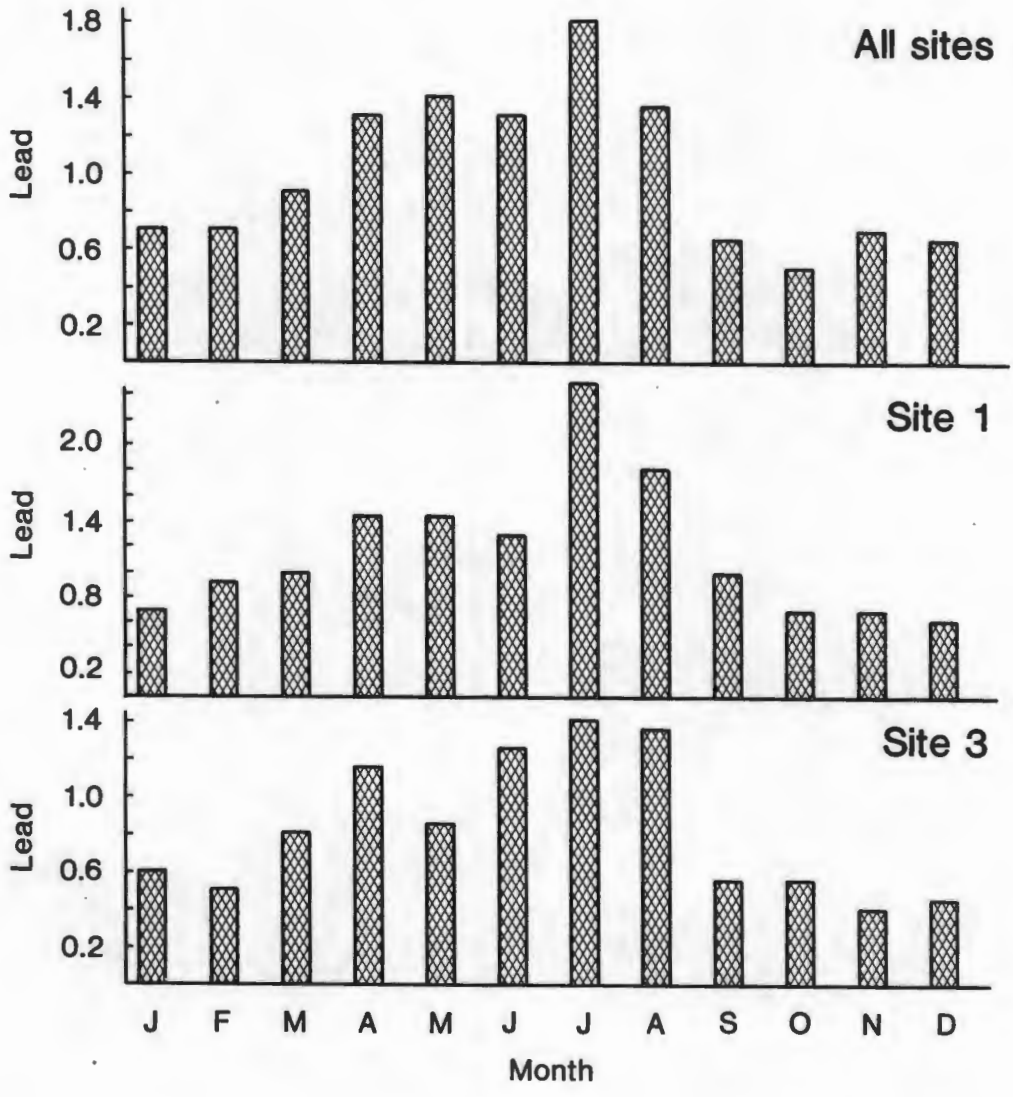


FIGURE 6.2a,b,c

MONTHLY AIR LEAD CONCENTRATION (ug/m^3) AT SITES

June to August and spring as September to November). It was found that there were no statistically significant differences in air lead concentrations between summer and spring, nor between winter and autumn, but the differences between the other seasons were statistically significant at the 5% level (Tables 6.8 and 6.9).

TABLE 6.8

VARIATION IN AIR LEAD CONCENTRATION (ug/m^3) BETWEEN SEASONS (ANALYSIS OF VARIANCE)

SOURCE	S.S	D.F	M.S	F-VALUE	TAIL PROBABILITY
Between groups	4.1683	3	1.3894	47.41	0.0000
Within groups	34.4966	1177	0.0293		
TOTAL	38.6649	1180			
Levene's Test for equal var.		3, 1177		51.58	0.0000
Welch		3, 594		48.05	0.0000
Brown-Forsythe		3, 834		47.02	0.0000

TABLE 6.9

DIFFERENCES IN AIR LEAD CONCENTRATION (ug/m^3) BETWEEN SEASONS (BONFERRONI PROBABILITIES)

	WINTER	SPRING	SUMMER
Spring	0.000*		
Summer	0.0000*	.1092*	
Autumn	0.865	0.0000*	0.0000*

(Anova results : F:47.41. d.f.:3, 1177. p: 0.0000)
* significant difference between seasons at 5% significance level

Grouping summer and spring months together, and autumn and winter months together, it is evident that the lead levels during the winter/autumn months were on average two-fold higher than during summer/spring months, and that the variation in air lead concentrations was nearly three-fold higher in the winter and autumn (Table 6.10, Figure 6.3). During winter months an average air lead concentration of 1.3 ug/m^3 can be expected.

TABLE 6.10

SIX-MONTHLY AIR LEAD CONCENTRATION (ug/m^3) BY SITE

		SITE 1	2	3	4	5	6	7	All sites tog.
Mar-Aug	Mean	1.6	1.2	1.1	2.5	1.0	1.5	0.6	1.3
	Std.Dev.	1.2	1.0	1.0	2.0	1.0	1.2	0.5	1.3
	Median	1.2	1.0	0.7	1.9	0.7	1.1	0.3	1.0
Sep-Feb	Mean	0.8	0.6	0.5	1.5	0.5	0.8	0.3	0.7
	Std.Dev.	0.4	0.6	0.3	0.6	0.4	0.5	0.3	0.5
	Median	0.7	0.5	0.5	1.6	0.4	0.7	0.3	0.5

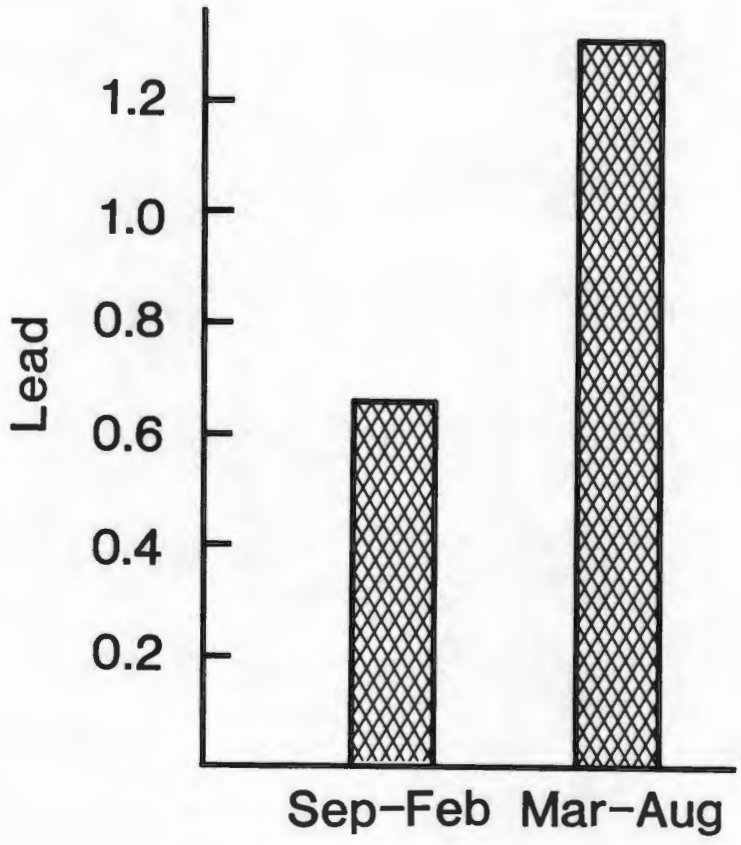


FIGURE 6.3

SIX MONTHLY AIR LEAD CONCENTRATION (ug/m^3) (ALL SITES TOGETHER)

A two way analysis of variance was performed to see if any interaction between the two factors, site and season, occurred. No interactions between site and season were significant at the 5% significance level (Table 6.11).

TABLE 6.11

VARIATION IN AIR LEAD CONCENTRATION (ug/m^3) BETWEEN SITES AND SEASONS (ANALYSIS OF VARIANCE)

SOURCE	S.S	D.F	M.S	F-VALUE	TAIL PROBABILITY
Site	9.3555	5	1.8711	78.86	0.0000
Season	3.6701	1	3.6701	154.68	0.0000
Interaction	0.2237	5	0.0447	1.89	0.0940
Error	31.4374	1325	0.0237		
Levene's test for equal var.		11, 1325		22.72	0.0000
Welch		11, 478		66.60	0.0000
Brown-Forsythe		11, 1008		62.13	0.0000

Bonferroni probabilities revealed that the difference between summer and winter at all sites was statistically significant, with the exception of site seven. Thus the effect of season was independent of site, with sites individually reporting levels twice as high in winter and autumn as in summer and spring.

RELATIONSHIP TO SEASONAL AIR QUALITY STANDARDS

Quarterly air lead levels at the sites (Table 6.12, Figure 6.4) were compared with the internationally recognised air lead standard recommended by the United States Environmental Protection Agency, which is 1.5 ug/m^3 (averaged over a three month period). It should be noted however, that standards and air monitoring strategies differ between and within various countries; therefore caution should be exercised in making direct comparisons.

TABLE 6.12

QUARTERLY AIR LEAD CONCENTRATION (ug/m^3) BY SITE

		SITE							
		1	2	3	4	5	6	7	All sites tog.
JUNE-AUG	Mean	1.9	1.4	1.3	2.7		1.5	0.5	1.5
	Std.Dev.	1.5	1.1	1.1	2.1		1.3	0.5	1.4
	Median	1.4	1.0	0.8	2.5		1.2	0.3	1.1
SEP-NOV	Mean	0.8	0.6	0.5	1.3	0.3	0.9	0.5	0.6
	Std.Dev.	0.5	0.5	0.3	1.4	0.3	0.5	0.3	0.5
	Median	0.8	0.5	0.5	0.5	0.2	0.8	0.4	0.5
DEC-FEB	Mean	0.8	0.7	0.5	1.6	0.5	0.8	0.3	0.7
	Std.Dev.	0.4	0.6	0.2	0.6	0.4	0.5	0.3	0.5
	Median	0.7	0.5	0.5	1.7	0.5	0.7	0.2	0.5
MAR-MAY	Mean	1.3	1.0	0.9	2.3	1.0	1.5	0.7	1.2
	Std.Dev.	0.9	0.9	0.8	1.9	1.0	1.2	0.5	1.2
	Median	1.1	0.7	0.7	1.8	0.7	1.1	0.6	0.9

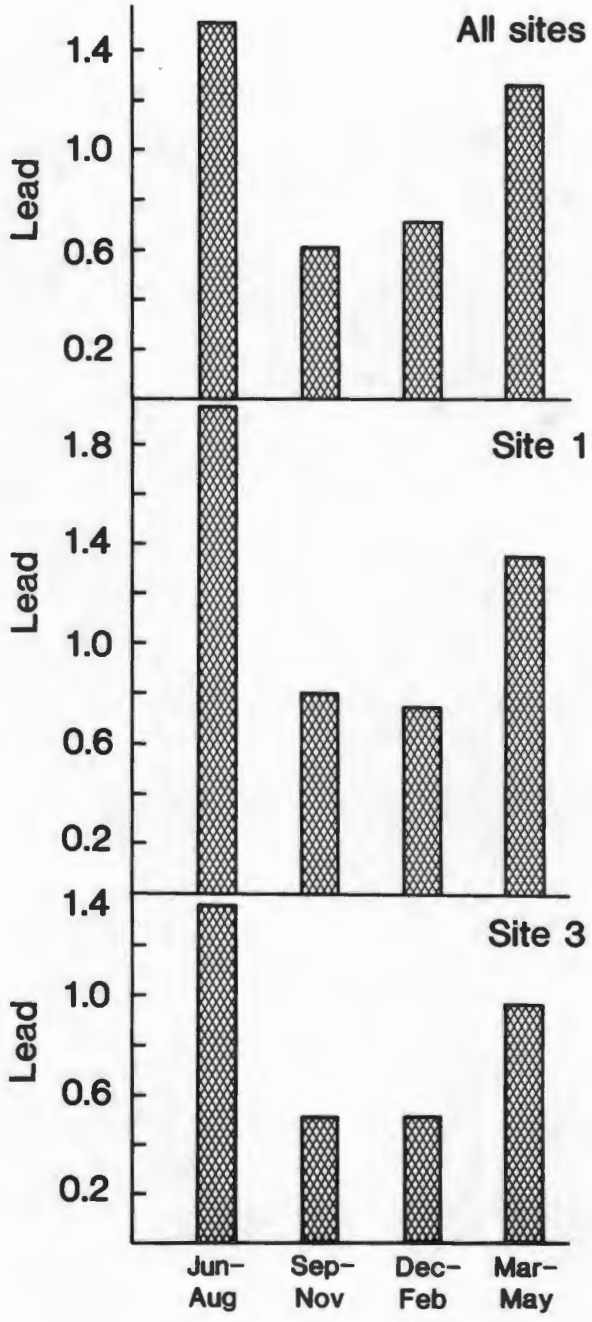


FIGURE 6.4a,b,c

QUARTERLY AIR LEAD CONCENTRATION (ug/m^3) AT SITES

From Table 6.12 it can be seen that at some sites the 1.5 ug/m^3 standard was surpassed. For instance, at site four the standard was surpassed during most of the year. At site one the standard was surpassed during the winter months, whilst at site six lead concentrations were close to the standard. Lead concentrations measured at sites two, three, five and seven were below the United States standard. It would thus appear that on most occasions during the summer, lead levels in Woodstock fall within the United States standard, whereas in winter at sites on busy roads, the standard is exceeded.

6.1.2.2 Daily Variations

Considerable daily variation in lead concentration was noted at all sites (Figure 6.5). It was decided to determine whether any difference in lead concentration occurred between weekdays and weekends. (Holidays were regarded as belonging to the weekend group.) A two-way analysis of variance indicated statistically significant differences at the 5% significance level between weekdays and weekends and no statistically significant interactions between site and weekday/weekend. A one way analysis of variance was subsequently performed, grouping data irrespective of site with respect to individual days of the week. Data recorded over periods of two days or more were excluded from

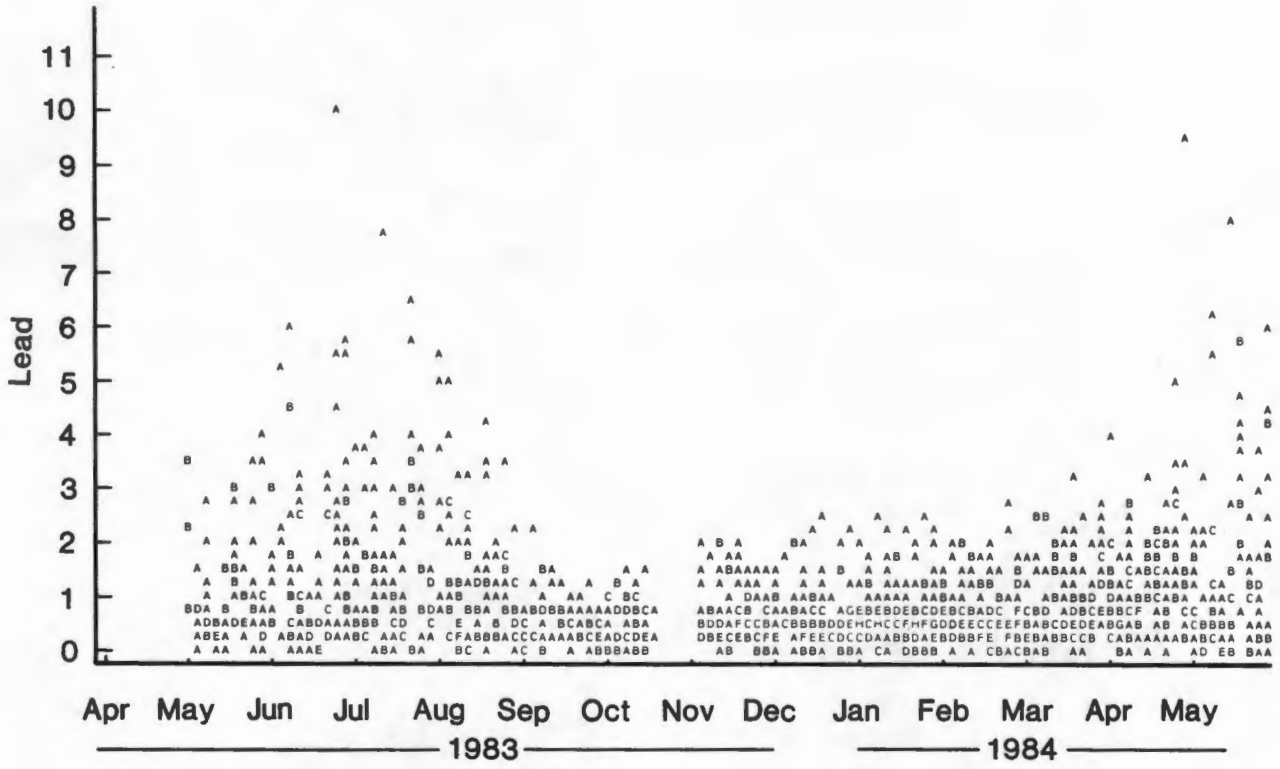


FIGURE 6.5a

DAILY AIR LEAD CONCENTRATIONS (ug/m^3) (ALL SITES TOGETHER)

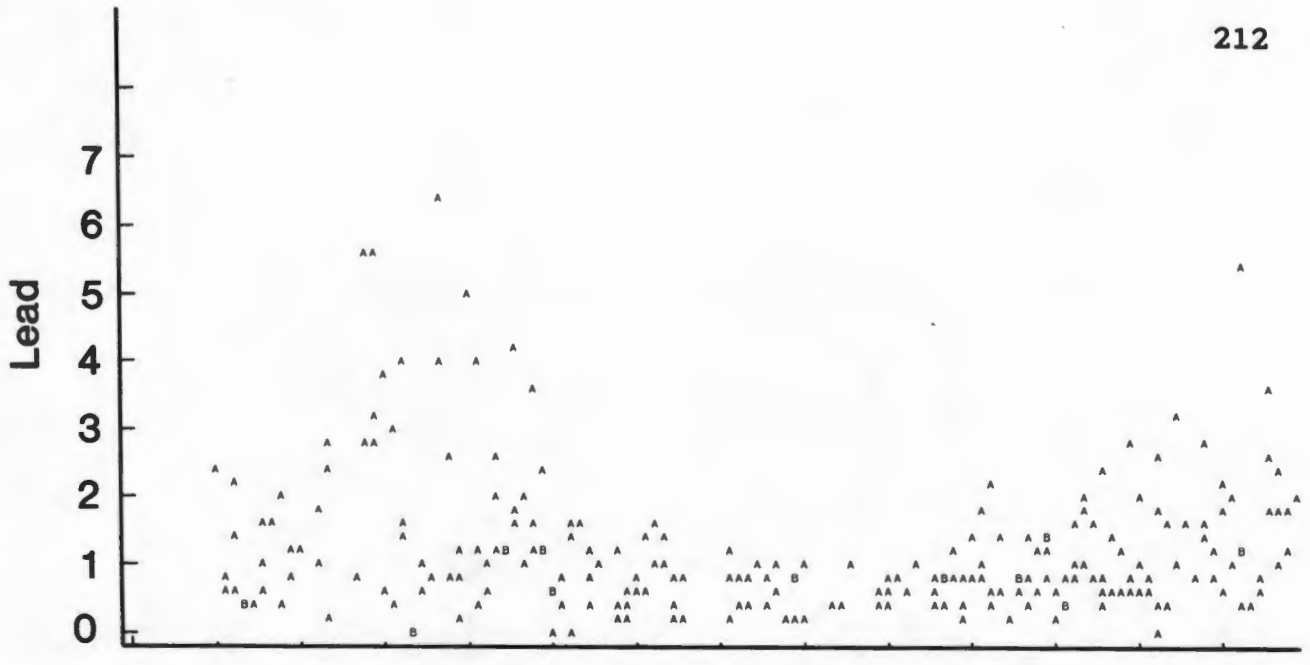


FIGURE 6.5b

DAILY AIR LEAD CONCENTRATIONS (ug/m^3) (SITE 1)

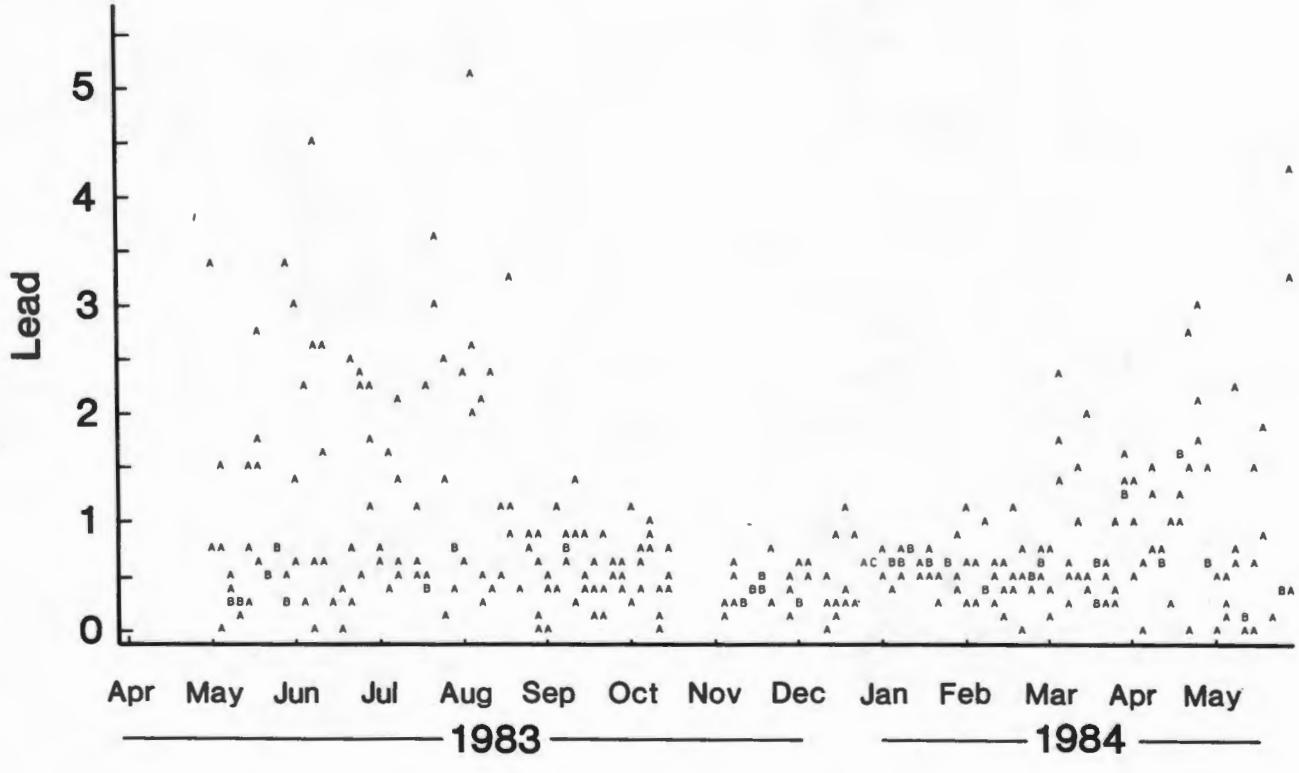


FIGURE 6.5c

DAILY AIR LEAD CONCENTRATIONS (ug/m^3) (SITE 3)

the analysis. Significant variations in the lead levels between the days of the week were found (Table 6.13).

Further statistical analyses revealed that Sundays were significantly different from other days of the week and that Fridays were different from most days except Mondays (Table 6.14).

TABLE 6.13

VARIATION IN AIR LEAD CONCENTRATION (ug/m^3)
BETWEEN DAYS OF THE WEEK (ANALYSIS OF VARIANCE)

SOURCE	S.S	D.F	M.S	F-VALUE	TAIL PROBABILITY
Between groups	2.3213	6	0.3869	11.57	0.0000
Within groups	36.8287	1101	0.0335		
TOTAL	39.1500	1107			
Levene's test for equal var.		6, 1101		6.26	0.0000
Welch		6, 475		16.98	0.0000
Brown-Forsythe		6, 1042		11.84	0.0000

TABLE 6.14

DIFFERENCES IN AIR LEAD CONCENTRATION (ug/m^3) BETWEEN DAYS OF THE WEEK (BONFERRONI PROBABILITIES)

	MON	TUE	WED	THU	FRI	SAT
TUE	0.5116					
WED	0.3646	0.8094				
THU	0.2167	0.5625	0.7283			
FRI	0.0155	0.0017*	0.0006*	0.0002*		
SAT	0.0287	0.1055	0.1512	0.2597	0.0000*	
SUN	0.0000*	0.0000*	0.0000*	0.0000*	0.0000*	0.0000*

(Anova results : F:11.57, d.f.:6, 1101, p: 0.0000)
* significant difference between days

On average, lead concentrations on the peak traffic day Friday were considerably higher than on other days of the week and Sundays were lower, suggesting the importance of traffic in influencing daily concentrations (Table 6.15).

TABLE 6.15

ANNUAL AIR LEAD CONCENTRATION (ug/m^3) BY DAY OF WEEK

DAY OF WEEK	MEAN	STD. DEV.
MONDAY	1.2	1.3
TUESDAY	1.1	1.2
WEDNESDAY	1.1	1.2
THURSDAY	1.0	1.0
FRIDAY	1.5	1.5
SATURDAY	0.9	0.9
SUNDAY	0.6	0.5

The same trend was exhibited during summer and winter, but was more marked during winter months (Table 6.16).

TABLE 6.16

ANNUAL AIR LEAD CONCENTRATION (ug/m^3)
BY DAY OF WEEK AND SEASON

	WINTER/ AUTUMN	SUMMER/ SPRING
MONDAY	1.6	0.7
TUESDAY	1.6	0.7
WEDNESDAY	1.5	0.7
THURSDAY	1.1	0.7
FRIDAY	1.9	0.9
SATURDAY	1.1	0.5
SUNDAY	0.7	0.5

6.2 DUST LEAD CONCENTRATIONS

The non-parametric Kruskal-Wallis test was used to determine whether differences in lead concentrations between sites were statistically significant. Kendall and Spearman rank correlation coefficients were also calculated to test for correlations between air lead levels, dust lead levels and traffic volumes.

To compare dust lead concentrations among sites, average lead concentrations were determined for each site. These are given in Table 6.17.

TABLE 6.17

MEAN DUST LEAD, AIR LEAD & TRAFFIC LEVELS AT SITES

SITE	DUST LEAD (ppm)	AIRLEAD (ug m^{-3})	TRAFFIC (cars/day)
1	2900	1.3	17675
3	790	0.9	907
4	3620	2.1	>20000
5	2580	0.7	241
6	3250	1.3	7752
7	410	0.4	260

Considerable differences in lead concentration existed within and between sites. For instance, dust lead concentrations varied from less than 500 ppm to over 3 000 ppm at the various sites.

In general, street dust lead concentrations were above the range for lead in dusts of natural origin, but were in the range which are typical for urban areas. There are no generally accepted maximum permissible levels or guidelines for lead in surface dust, but there exists a recommended level of 500 ppm for the Greater London area in the United Kingdom. In all but one case the average dust lead concentrations exceeded this level.

The mean dust lead concentrations (ppm) of the one millimetre fraction and total sample, as well as the mass loading (ug/2 sq.ft.) of the samples at sites on and away from major roads are given in Table 6.18.

TABLE 6.18

DUST LEAD CONCENTRATIONS AT SITES ON AND AWAY FROM MAJOR ROADS

		On Major Road	Away From Major Road
1mm Fraction (ppm)	Mean	1999	737
	Std.Dev.	226	358
Total Sample (ppm)	Mean	1671	450
	Std.Dev.	551	318
Mass Loading (ug/2sq.ft.)	Mean	16534	7054
	Std.Dev.	16001	4420

As can be seen, the mean lead concentrations of both the one millimetre fraction and the total sample were considerably higher at sites on major roads. The non-parametric Kruskal-Wallis test was performed which indicated that there were significant variations in the dust lead levels between sites. The differences in dust lead concentration with respect to the position of sites on and away from major roads were statistically significant for both the one millimetre fraction and the total sample ($p = 0.0339$). Lead levels expressed as mass loadings

were also higher on major roads than away from major roads, but this difference was not statistically significant ($p = 0.4795$).

Comparing the lead concentration of the one millimetre fraction with that of the total sample, it was evident that overall the smaller particle fractions had higher lead concentrations. This finding has been confirmed by others (Archer and Barratt, 1976; Fergusson and Ryan, 1984). A correlation analysis was performed, which indicated a statistically significant relationship between lead concentration in the one millimetre fraction and total sample ($r = 0.82277$; $p = 0.0001$) (Figure 6.6). No statistically significant relationship between mass loading (ug/2 sq.ft.) and concentration (ppm) was found.

A correlation existed between dust lead and air lead concentrations, which was statistically significant at the 5% significance level ($r = 0.8857$). A correlation was also found between dust lead concentrations and traffic volume ($r = 0.7714$); this however was not statistically significant.

From the dust lead monitoring study, it was evident that air lead levels had an influence on the lead concentration of street dusts in the area. For instance, at sites situated on major roads, dust lead concentrations were two to three-fold higher than at sites situated 200 to 300 metres away from major roads. A significant association between air lead levels and dust lead concentration was found.

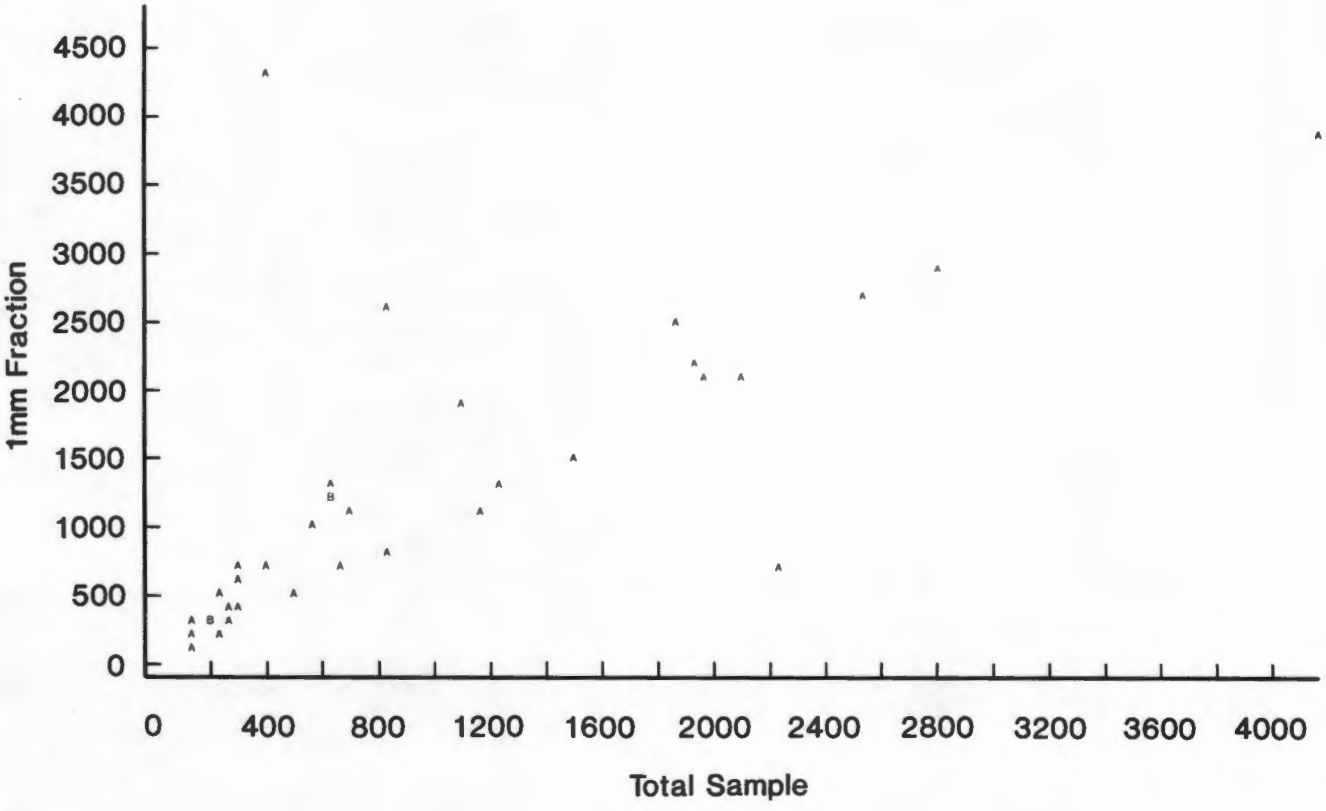


FIGURE 6.6

THE RELATIONSHIP BETWEEN DUST LEAD CONCENTRATION (ppm) IN 1 mm FRACTION AND TOTAL SAMPLE

Lead concentrations in the one millimetre fraction were found to be higher than in the larger fractions; no relationship was found between lead levels expressed in terms of mass loading (ug/2 sq.ft.) and concentration (ppm). This has implications for the choice of exposure index used in epidemiological studies.

6.3 DISCUSSION: LEAD IN THE ENVIRONMENT

6.3.1 AIR LEAD CONCENTRATIONS

From the environmental survey of air lead concentrations carried out, it was established that air lead levels varied from less than 0.5 ug/m^3 to above 1.5 ug/m^3, averaging 1.1 ug/m^3 throughout the year. These levels are considerably above the levels measured at other industrial and residential sites in Cape Town which average around 0.6 ug/dl in 1983 (Medical Officer of Health of the Divisional Council of the Cape, 1986). It is difficult to compare these readings with those obtained in other parts of the world, as sampling strategies differ from country to country. In the United States of America, for instance, where there are standards for the siting of air monitors, samplers can be placed anywhere between two and 15 metres from the ground (EPA, 1986b). Thus considerable variations may occur, both vertically and horizontally, from a source.

In general, concentrations may vary from 0.000076 ug/m^3 in remote areas to over 10 ug/m^3 near sources such as smelters. In confined areas such as tunnels and parking garages, lead concentrations can be 10 to 1 000 times greater than levels near roads or urban areas. Atmospheric lead concentrations are about 2.5 times greater in the central city than in outlying suburbs, and are even lower in rural areas (EPA, 1986b). In this respect it was interesting to note that considerable variation may occur on a microscale within an urban area, as was shown in the study area monitored here, which is only two square kilometres in size. New siting guidelines to monitor air lead levels on a microscale have been introduced recently in the United States of America.

In countries such as the United States of America and West Germany, the decreasing use of lead in petrol has been associated with a decrease in air lead concentrations (Southwood, 1983). In the United States, average lead concentrations have dropped significantly since 1977. Nearly all sites report annual averages below one ug/m^3 (EPA, 1986b). Beyond the immediate vicinity of urban areas, air lead levels decline to values of 0.1 to 0.5 ug/m^3 (EPA, 1986b).

In the United Kingdom, in general, the lead concentrations in rural areas are below 0.15 ug/m^3 and in most parts of the larger cities below one ug/m^3. Air lead concentrations in the United Kingdom are similar to those found in other industrial countries (Southwood, 1983).

The lowest air lead value reported is 0.000076 ug/m^3, measured at the South Pole (Maenhaut et al., 1979). According to the EPA (1986b) it seems likely that the concentration of natural lead in the atmosphere is between 0.00002 ug/m^3 and 0.00007 ug/m^3.

The present study of the distribution of lead in the environment shows that elevated levels of lead are present in air and dust samples in Woodstock. In particular, it was established that air and dust lead concentrations were significantly raised at sites situated in proximity to high density traffic. For instance, air lead levels at sites situated on major roads were nearly two-fold higher than at sites situated 200 to 300 metres away from major roads, with air lead levels being highest at sites with the highest density of traffic. At sites situated on major roads, air lead levels averaged 1.5 ug/m^3, at sites away from major roads air lead levels were around 0.8 ug/m^3.

Air lead levels had an influence on dust lead levels, which were similarly elevated at sites on major roads. Dust lead concentration varied from less than 500 ppm to more than 3 000 ppm.

Heavily travelled roads can be thought of as linear hotspots, especially in built up areas where narrow streets and tall buildings inhibit air circulation by producing a canyon-like

effect (Southwood, 1983). In cities, therefore, lead concentrations tend to be highest in high traffic areas.

It is generally recognised that petrol-derived lead aerosols are the major constituent of atmospheric lead, accounting for between 75 and 90% of atmospheric lead. Lead concentrations measured at a particular site, however, depend on a number of factors, including the nature of the traffic on the road, the nature of the traffic on the surrounding roads, contributions from possible industrial sources and other factors. Thus, although the data from this study suggest a relationship between traffic flow and atmospheric lead concentrations, the exact nature of the relationship is complex.

For example, there was no statistically significant difference in the lead concentrations at sites one and six, although site one carried more than twice the volume of traffic. The position of site six, situated on a steep incline, may have had an influence on the lead particulates released by vehicles. Additional information on the influence of traffic comes from an examination of daily air lead levels during the week (see Section 6.1.2.2).

The relatively low lead in air level measured at site seven (averaging 0.4 ug/m^3 during the year) most likely reflect the open nature of the area, making possible the rapid and widespread dispersion of aerosols. There is also virtually no commercial or industrial activity in this area, and a much less dense road

network. This is in contrast to the situation of sites two, three and five also in residential areas away from major roads, but in areas characterised by a degree of light industrial usage and a denser road network. The average annual lead in air level at these sites (considered together) was 0.9 ug/m^3.

Air lead levels were found to be on average two-fold higher in the winter months than in the summer. During this time period in particular, air lead levels at certain sites on major roads exceed the EPA standard of 1.5 ug/m^3 on occasions.

Air lead levels were also found to vary during the week, with the highest concentrations occuring on Fridays, when traffic volumes peak and the lowest occuring on Sundays when traffic is at a minimum. Minor industrial sources did not appear to play a major role in influencing air lead concentration in the area studied, the major influence coming from traffic, as indexed by both spatial and daily variations.

Seasonal variations in atmospheric lead concentrations have been observed at many locations (Nriagu, 1978c). In the United States of America, temporal variations in atmospheric lead occur, with maximum lead concentrations in winter, and minima in the summer (EPA, 1986b). Seasonality effects can be brought about by many different factors such as differences in driving habits or changes in climatic conditions which affect atmospheric dispersion and mixing processes (Nriagu, 1978c). Particularly

high concentrations of lead are usually associated with temperature inversions. This is the case with respect to air lead levels measured in Cape Town.

The spatial and temporal variations in atmospheric lead have implications for air monitoring strategies and epidemiological studies concerned with the relationship of exposure to atmospheric lead and the health impact on populations.

A figure of 0.8 ug/m^3 can be taken to be representative of the yearly average base-line level of atmospheric lead exposure of the population. In addition to this background level, children exposed to heavy traffic densities can be expected to be exposed to higher levels of lead in the environment, probably in the range 1.5 to 2.0 ug/m^3.

6.3.2 DUST LEAD CONCENTRATIONS

From the present study, it was established that street dust lead concentrations in the area varied from less than 500 ppm to over 3 000 ppm. In a review by Nriagu (in EPA, 1986b) street dust lead concentrations in the United States of America were found to range from 300 to 18 000 ppm in several cities. In a study of street dusts in five residential areas in Greater London, the mean street dust concentration was found to be 1 200 ppm - the highest value was 3 500 ppm and the lowest value 430 ppm (Duggan

and Williams, 1977). This is similar to the range of values reported in this study. In the city of Manchester, street dust lead levels averaged 970 ppm (Day et al., 1975). In Hong Kong street dust lead levels averaged 1 627 ppm (Lau and Wong, 1982)

It is very difficult, however, to compare readings from one place to another due to differences in sampling and analytical procedures used. Caution should therefore be used in comparing results of studies.

In general, in non-urban environments, street dust concentrations range from 80 to 130 ppm, whereas urban street dusts range from 1 000 to 20 000 ppm, according to the EPA (1986b). The EPA has used the value 90 ppm in street dust as that approximating baseline exposure, and a value of 1 500 ppm as typical of that of dust lead in an urban environment. According to Southwood (1983) concentrations of lead in urban street dusts are typically in the range 500 to 5 000 ppm. These are similar to the values obtained in this study.

Street dust lead concentrations in this study were also found to correlate with traffic density. In one study, in EPA (1986b) lead in street dust in Hong Kong ranged from 960 to 7 400 ppm, with no direct relationship to traffic density. In other studies in Hong Kong, however, values from 132 ppm at 20 vehicles/day to 3 876 ppm at 37 180 vehicles/day were reported. Fourteen sites showed a close correlation with traffic density (Lau and Wong, 1982).

In a study by Anagnostopolous (1983) levels of lead in street dust were found to correlate with traffic density. Similarly, in a study of heavy metal pollution in Christchurch, New Zealand, a significant correlation of lead in street dust with traffic density was found (Fergusson and Simmonds, 1983). Harrison (1979) also found that lead in street dust was related to traffic density. In a study in Manchester, however, there was little significant variation in dust lead concentrations with type of locality (main road, side street) or with position in the urban area (Day et al., 1975).

6.3.3 RELATIONSHIP BETWEEN ENVIRONMENTAL LEAD AND BLOOD LEAD CONCENTRATIONS AT SCHOOLS

As was noted in sections 4.1.4 and 5.1.6.5, there was a strong association between blood lead levels and the geographic position of schools with respect to traffic density. For instance, among both white and coloured pupils, children attending schools in the immediate proximity of heavy traffic had blood lead levels five to seven ug/dl higher than children attending schools away from traffic. The air lead levels measured at these schools were also higher than those at schools away from heavy density traffic. There remained a statistically significant association between blood lead levels and school, when socio-economic factors were taken into account. It is thus suggested that irrespective of differences with respect to socio-economic standing at schools,

an independent factor probably related to the position of schools with respect to traffic, contributes to variations in blood lead levels.

There is a wealth of published data on the relationship between environmental lead (particularly air lead) and blood lead, but much of it is difficult to interpret due to methodological weaknesses such as lack of representativeness of air lead data vis a vis human exposure situations. Understanding of exposure requires detailed knowledge of spatial and temporal variations in air lead levels. There is also a lack of consistency in the range of air lead measurements in different studies. There are, in addition, problems which relate to confounding factors which are often not taken into account.

Despite such problems, the EPA have estimated that for children, there is an increase in blood lead concentration of approximately two ug/dl by direct inhalation, for every one ug/m^3 increase in air lead concentration.

In a recent review, Brunekreef (1984) reviewed the relationship between air lead and blood lead in children. He looked at 19 studies carried out in 10 different countries. He found that for most studies of children there is an increase of three to five ug/dl for every one ug/m^3 increase in air lead concentration. Increases of one to two ug/dl occurred mainly at high values of blood lead. One reason for this discrepancy is probably due to

the curvilinear nature of the relationship between air lead and blood lead (Chamberlain, 1983). Chamberlain (1983) also reported that the relationship between air lead and blood lead in children is difficult to determine, due to the nature of confounding variables.

Brunekreef found that adjustment for confounders was absent or incomplete in nearly all studies. This is a most crucial aspect and renders many studies on the relationship between air lead and blood lead difficult to interpret. Brunekreef concluded that on the basis of published studies it was not possible to make a reliable quantitative estimate of the relationship between air lead and children's blood lead. Nevertheless, it may be concluded that the direct inhalation of aerosols is unlikely to make a major contribution to children's blood lead levels, in the range reported in the present study.

In a review of the data attempting to quantify the relationship between dust lead concentration and blood lead, the EPA (1986c) concluded that despite extreme difficulties, a median value of approximately two ug/dl per 1 000 ppm for children aged two to three years is appropriate. The level for older children is probably somewhat lower. Duggan, in a major review (1980, 1983b) has estimated that blood lead levels in children may rise by about five ug/dl for every 1 000 ppm of lead in dust.

It is thus reasonable to conclude that for children exposed to heavy traffic densities, either at school or in play, where air lead concentrations in the region 1.5 to 2 ug/m^3 may prevail in busy streets (in winter months) and contribute to dust lead concentrations which are in the range 500 to 3 500 ppm, direct inhalation of aerosols and, more importantly, ingestion of dust may account for blood lead levels being raised by a few ug/dl. Children of lower socio-economic status will show the greatest increases in blood lead levels at these exposures during winter months. In general for this working class, school age population of children aged six to seven years, exposed overall to air lead levels of 1.3 to 1.4 ug/m^3 (during winter months) and 410 to 3 620 ppm in dust lead concentration, blood lead concentrations can be expected not to be below 12 ug/dl among whites and 18 ug/dl among coloureds. Routine air and dust lead monitoring should be carried out in the area, particularly at those schools where blood lead levels are elevated.

CHAPTER 7

RESULTS AND DISCUSSION : CASE CONTROL STUDY

In the previous three chapters, the results pertaining to the cross-sectional analytical study as well as to the environmental study were discussed.

In this chapter, results relating to the case control study which was carried out in stage two of the study are described and discussed. The results of the home interview univariate analyses are given in Appendix E3. These are described below in Sections 7.1 to 7.10. A discussion of the results is given in Section 7.11.

7.1 HAND-MOUTH ACTIVITIES

7.1.1 PICA

There were significant differences between the cases and controls with respect to certain hand-mouth activities related to pica. More cases than controls had been observed to eat non-food items such as cement ($p = 0.0312$), plaster ($p = 0.0295$), soil ($p = 0.0410$), sticks ($p = 0.0016$), and matchsticks ($p = 0.0006$).

Pica for paint was not a significant factor among the cases, although it approached statistical significance (Table 7.1).

TABLE 7.1a

PAINT FLAKES (Q D1)

		Cases	Controls
Yes	No.	3	0
	%	11	0
No	No.	24	36
	%	89	100

Statistical Test : Fishers Exact Test
"p" value .0737

TABLE 7.1b

CEMENT (Q D1)

		Cases	Controls
Yes	No.	7	2
	%	26	6
No	No.	20	34
	%	74	94

Statistical Test : Fishers Exact Test
"p" value .0312

TABLE 7.1c

PLASTER (Q D1)

		Cases	Controls
Yes	No.	4	0
	%	15	0
No	No.	23	36
	%	85	100

Statistical Test : Fishers Exact Test
"p" value .0295

TABLE 7.1d

SOIL (Q D1)

		Cases	Controls
Yes	No.	10	5
	%	37	14
No	No.	17	31
	%	63	86

Statistical Test : Fishers Exact Test
"p" value .0410

TABLE 7.1e

STICKS (Q D1)

		Cases	Controls
Yes	No.	7	0
	%	26	0
No	No.	20	36
	%	74	100

Statistical Test : Fishers Exact Test
"p" value .0016

TABLE 7.1f

MATCHSTICKS (Q D1)

		Cases	Controls
Yes	No.	8	0
	%	30	0
No	No.	19	36
	%	70	100

Statistical Test : Fishers Exact Test
"p" value .0006

TABLE 7.1g

CIGARETTE ENDS (Q D1)

		Cases	Controls
Yes	No.	7	3
	%	26	8
No	No.	20	33
	%	74	92

Statistical Test : Fishers Exact Test
"p" value .0838

TABLE 7.1h

COAL (Q D1)

		Cases	Controls
Yes	No.	1	0
	%	4	0
No	No.	26	36
	%	96	100

Statistical Test : Fishers Exact Test
"p" value .4286

Parents first noticed their child eating the above-mentioned non-food items at the average age of two years. For the majority of children the habit had ceased by the age of three to four. Three of the cases (but no controls) still pursued the habit at age six to seven years. There was no statistically significant difference with respect to the frequency with which the habit was pursued among cases or controls.

7.1.2 OTHER MOUTHING ACTIVITIES

There was no significant difference with respect to the activity of sucking fingers or biting nails between cases and controls. With respect to certain other mouthing activities, however, there was a highly significant difference between cases and controls. For instance 54% of cases and only six percent of controls were regularly observed to put their hands or other objects in their mouths ($p = 0.0001$) (Table 7.2).

TABLE 7.2

MOUTHING ACTIVITY (Q D7)

		Cases	Controls
Frequently mouthes on hands and objects	No.	14	2
	%	54	6
Does not mouth on hands and objects	No.	11	30
	%	42	94
Don't know	No.	1	0
	%	4	0

Statistical Test : L.R. Chi-Square
"p" value .0001

There was no significant difference among cases and controls regarding the frequency of hand-washing activities, but significantly more cases than controls were reported to have noticeably dirty hands after play (p = 0.0392) (Table 7.3).

TABLE 7.3

DIRTY HANDS (Q D8)

		Cases	Controls
Dirty hands frequently	No	13	10
	%	68	36
Dirty hands infrequently	No	6	18
	%	32	64

Stats. Test : Fishers Exact
"p" value .0392

7.2. PLAY SITES

As is evident from Table 7.4 more cases than controls played more often outside than inside (p = 0.0360). There was also an indication that more cases than controls often played in busy streets, but this difference did not reach statistical significance.

TABLE 7.4
PLAY-SITES (Q D10)

		Cases	Controls
Inside	No.	6	18
	%	22	50
Outside	No.	21	18
	%	78	50

Statistical Test : Fishers Exact Test
"p" value .0360

7.3 SUPERVISION/STIMULATION

There were no significant differences between cases and controls with respect to the child's main caretaker (e.g. mother, sister), nor were there any significant differences regarding whether or not the child had attended a creche.

More controls than cases had their own toys (p = 0.0235), or books (p = 0.0023). Also, more controls than cases had been on a

long trip or journey to another town (p = 0.0358) and had visited a museum (p = 0.0447).

7.4 HOME ENVIRONMENT

There was a difference in the age of the homes of the cases (mean age 65 years) and controls (mean age 51 years), although in both instances the median age of the home was 50 years (p = 0.0080).

TABLE 7.5
AGE HOME (YEARS) (Q F3)

	Cases	Controls
No.	26	33
Mean	65	51
Std. Dev.	21.73	28.28
Median	50	50

Statistical Test : Kruskal-Wallis
"p" value .0080

With respect to the type of fuel used in the home, there were differences with respect to the use of electricity : 78% of the cases and 97% of the controls used electricity (p = 0.0360). There were no other statistically significant differences between the cases and controls with respect to other types of fuel used, although more cases than controls used paraffin. This difference approached statistical significance. There was a significant

difference between the two groups with respect to the number of smokers in the children's homes - more smokers lived in the homes of cases than controls (p = 0.0416).

7.4.1 CONDITION OF FLAKING SURFACES

As can be seen from Tables 7.6 and 7.7, there were significant differences between the cases and controls with respect to the condition of flaking surfaces both inside and outside the house. The homes of cases were considerably more dilapidated than the homes of controls, both inside (p = 0.0240) and outside (p = 0.0088).

TABLE 7.6

CONDITION OF INSIDE SURFACES

	Cases	Controls
Well maintained		
No	13	30
%	52	81
Deteriorating/ Dilapidated		
No	12	7
%	48	19

Stat.Test Fishers Exact
"p" Value .0240

TABLE 7.7

CONDITION OF OUTSIDE SURFACES

	Cases	Controls
Well maintained		
No	12	28
%	44	78
Deteriorating/ Dilapidated		
No	15	8
%	56	22

Stat. Test Fishers Exact
"p" Value .0088

7.4.2 LEAD CONCENTRATION (PPM) OF PAINT SAMPLES

(Appendix E4a-k)

The mean and median paint lead concentrations in the inside wall surfaces, weighted by number of samples per household, were slightly higher among the cases than the controls, but these differences were not statistically significant (Appendix E4a).

The lead concentrations in the indoor windowsill samples (Appendix E4b) were significantly higher than the indoor wall samples among both cases and controls but there were no significant differences between the cases and controls. The median lead levels in the windowsill samples for the two groups were two percent and 1.6% respectively.

Grouping all inside wall and windowsill samples together (Appendix E4c), it was evident that the cases had slightly higher mean and median levels than the controls, but these differences were not statistically significant.

There were no obvious differences in the lead concentrations in the outdoor samples between cases and controls, although the controls had slightly higher levels than the cases (Appendix E4d).

In general, the inside walls had slightly lower lead concentrations than the outside walls among both the cases and controls.

Grouping all inside and outside paint samples together, (weighted by the number of samples per household) it was evident that there were no significant differences in the average lead concentrations of the paint samples between the cases and controls (Appendix E4e). 44% of the cases and 26% of the controls had at least one sample with a lead concentration above or equal to one percent, but this difference was not statistically significant (Appendix E4k).

7.4.3 INTERIOR SURFACE DUST SAMPLES

As can be seen from Table 7.8, the lead levels in house dust (expressed in terms of mass-loading per unit area) were higher among the cases than controls (p = 0.0352).

TABLE 7.8

HOUSE DUST LEAD LEVELS (ug/2sq.ft)

	Cases	Controls
No	20	28
Mean	1747	82
Std.Dev.	4807	177
Median	64	26

Stat. Test	Mann-Whitney
"p" value	.0352

Also, more homes of cases than controls had mass loadings above 100 ug/2 sq. ft (equal to 100 ug/0.186 m^2), from either the bedroom or main living room (p = 0.0205) (Table 7.9).

TABLE 7.9

HOUSE DUST LEAD LEVELS

	Cases	Controls
> 100 ug/2 sq. ft.		
No.	12	6
%	50	19
< 100 ug/2 sq. ft.		
No.	12	26
%	50	81

Stat. Test	Fishers Exact
"p" value	.0205

There were no significant differences between the cases and controls with respect to lead levels in house dust expressed in terms of concentration. The median house dust lead concentration in the respective groups were 535 and 550 ppm. It was also observed that for both groups, median lead levels expressed in terms of concentration and mass-loading were higher in outside (veranda) samples than inside (Appendix E5).

7.4.4 WATER SAMPLES

No lead water pipes were found among the accessible pipes which were examined at the homes of the cases and controls. Among both cases and controls the lead concentration in all drinking water samples tested was within safety limits (<10ug/l).

7.4.5 AIR SAMPLES

From Table 7.10, it can be seen that there were no significant differences in the two-weekly air lead levels between the cases and controls over the sampling periods during which air lead measurements were obtained.

TABLE 7.10

MEAN AIR LEAD LEVELS (ug/m^3)

Cases	Controls
.57	.69

7.4.6 STREET DUST SAMPLES

There were no significant differences in the lead levels of the spot street-dust samples expressed in terms of concentration (ppm) or mass loading (ug/2 sq. ft.), between the cases and controls (Appendix E6). In both instances lead levels were elevated. Correlation analyses performed revealed that there was a statistically significant association between street-dust lead levels expressed in terms of concentration and mass-loading per unit area respectively ($r = 0.50319$, $p = 0.0009$).

7.4.7 HOME CLEANLINESS AND HYGIENE

From Table 7.11, it can be seen that the homes of the cases were in considerable need of attention as far as cleanliness in the home was concerned. For instance the homes of 54% of the cases and only 17% of the controls were in need of attention or unsatisfactory ($p = 0.0044$).

TABLE 7.11

CLEANLINESS SCORES

	Cases	Controls
Satisfactory		
No	11	29
%	46	83
In need of attention/ unsatisfactory		
No	13	6
%	54	17

Stat.test Fishers Exact
"p" value .0044

7.5 NUTRITIONAL STATUS

From a comparison of the distribution of the indicators in the study population with those of the international reference population, it was apparent that there were no statistically significant differences in overall nutritional status between the two groups. In general the nutritional status of both cases and controls was satisfactory.

There were also no statistically significant differences between the groups with respect to the number of cases and controls below 90% of their expected height for age or 80% of their expected weight for age (Appendix E7).

7.6 DIETARY HISTORY

There were no significant differences between cases and controls with respect to the frequency with which they consumed various food items, vis. fresh and tinned meat, fish, vegetables, fruit, mealie-meal, bread, cereals, cheese, eggs , milk, cooldrinks, cakes/puddings and sweets.

With regard to the type of pots used for cooking, more controls than cases used steel pots for cooking, whilst the majority of the controls and all the cases used aluminium pots ($p = 0.0076$).

7.7 SOCIO-DEMOGRAPHIC, CULTURAL AND ECONOMIC FACTORS

There were no differences with respect to blood lead levels and the gender of the cases and controls. There were significant differences between the cases and controls with respect to home language and religion. 41% of the cases and 71% of the controls spoke English at home ($p = 0.0309$). 45% of the cases and 10% of the controls were of the Christian faith, the rest were of the Islamic faith ($p = 0.0050$).

There was a statistically significant difference in the number of people living in the homes of the cases and the controls, with

the median number of people in the cases' homes being nine, and the median number of people in the controls' homes being six ($p = 0.0198$). The occupational density of cases' homes was also greater than controls ($p = 0.0069$) (Table 7.12).

TABLE 7.12

OCCUPATIONAL DENSITY

	Cases	Controls
No.	24	33
Mean	189	125
Std. Dev.	106	48
Median	159	100

Stat. Test	Mann-Whitney
"p" value	.0069

There were no significant differences with respect to the number of children or siblings living in the home. There were also no significant differences with respect to the number of rooms or sleeping rooms in the homes of the cases and controls. More cases than controls had their own rooms or beds - these differences approached statistical significance.

There was a significant difference in the monthly rent between cases and controls, with the median monthly rent in the two groups being R33-00 and R60-00 respectively (p = 0.0448).

There was a statistically significant difference in the mother's level of schooling between the two groups (p = 0.0313), but not with respect to father's level of schooling. The father's monthly income and the total family income were higher among the controls than the cases, but these differences did not reach statistical significance. There was no significant difference in the mother's income between the two groups. There was a significant difference in the total family income relative to the number of dependants, between the cases and controls (p = 0.0383) (Table 7.13).

TABLE 7.13

TOTAL FAMILY INCOME (RANDS/MONTH), RELATIVE TO NO. OF DEPENDANTS (Q G9)

	Cases	Controls
No.	16	25
Mean	152	252
Std. Dev.	99	192
Median	133	203
Stat. Test	Mann-Whitney	
"p" value	.0383	

There was also a statistically significant difference in the social class of the cases and controls as determined on the basis

of the job category classifications allotted to the bread winners of the families. A modified version of the British classification system (Registrar General, 1960) was used, whereby the first three categories were grouped together as one class, with the other two categories forming the lower two social class groups. 56% of the cases and 91% of the controls fell into the top category, whilst 44% of cases and nine percent of controls fell into categories two and three (p = 0.0044) (Table 7.14).

TABLE 7.14

SOCIAL CLASS

	Cases	Controls
Category 1	14	29
	56	91
Categories 2,3	11	3
	44	9
Stat. Test	Fishers Exact	
"p" value	.0044	

Three cases (11%) and one control had fathers who worked in the printing industry and one case and one control had fathers who were involved in panel-beating activities. The clothing industry and the construction industry were the single biggest employers of mothers and fathers respectively.

7.8. MEDICAL AND BEHAVIOURAL HISTORY

The majority of cases and controls (over 90%) were reported to be well at the time of the interview, although 11% of both groups were receiving some form of medical treatment.

There were no statistically significant differences with respect to the medical/behavioural factors tested for between groups, but cases were reported to be performing less well at school than controls ($p = 0.0269$), and nearly three times as many cases as controls were repeating grade one (41% vs 14%, $p = 0.0207$).

7.9 SUMMARY

In this part of the study, a number of factors related to the physical and social characteristics of the child's home environment emerged as being important, as well as some factors related to behavioural characteristics of children (Table 7.15).

TABLE 7.15

CHARACTERISTICS OF HIGH AND LOW RISK CHILDREN

	Cases (Percentage)	Controls	"p" value	Odds Ratio (95% C.I.)
Pica				
Matchsticks	30	0	.0006	Indet.
Sticks	26	0	.0016	Indet.
Cement	26	6	.0312	6.0 (1.1-31.8)
Plaster	15	0	.0295	Indet.
Soil	37	14	.0410	3.6 (1.1-12.3)
Mouthing	54	6	.0001	19.1 (3.7-97.0)
Dirty Hands	68	36	.0392	3.9 (1.1-13.5)
Outside Playsite	78	50	.0360	3.5 (1.1-10.7)
Own Books	46	86	.0023	7.4 (2.1-26.0)
Age Home (yrs)	65	51	.0080	
No. Smokers	2	1	.0416	
Flaking Ins. Surfaces	48	19	.0240	4.0 (1.3-12.4)
Flaking Out. Surfaces	56	22	.0088	4.4 (1.5-13.1)
House Dust (ug/2sq.ft)	64	26	.0352	
House Dust (>100ug/2sq.ft)	50	19	.0205	4.3 (1.3-14.4)
Cleanliness (unsat.)	54	17	.0044	5.7 (1.7-18.7)
Home Lang. English	41	71	.0309	3.5 (1.3-10.7)
Christian Religion	45	10	.0050	7.2 (1.7-30.9)
No. People in Home	9	6	.0198	
Occ. Density	159	100	.0069	
Monthly Rent (rands)	33	60	.0448	
Mother's Schooling (years completed)	6	7	.0313	
Total Family Income (rands/month)	133	203	.0383	
Social Class 1	56	91	.0044	

Sources of lead were found in the homes of both cases and controls, but were more accessible in the homes of cases than controls. The homes of the cases were more dilapidated than those of the controls (with more flaking lead paint), they had more lead rich dust and were in considerable need of attention as far as the overall cleanliness of the home was concerned. Lead

levels in water, air and street dust were not found to vary significantly between cases and controls, there were also no significant differences with respect to their overall nutritional status or reported dietary habits.

Significant differences with respect to socio-demographic, cultural and economic factors between cases and controls do emerge. Most of the cases were Afrikaans speaking and a significant proportion were of the Christian faith, whilst the controls were mainly English speaking and of the Islamic faith. The homes of the cases were more crowded than the controls. The mother's level of schooling was poorer among cases, and the total family income relative to the number of dependants was also lower among cases. Other potential indicators of socio-economic factors related to the type of pots used, the use of electricity, and the number of smokers in the home. It is thought that the greater use of aluminium pots by cases relates to matters of cost (these pots being considerably less expensive than, for example, steel pots). Their relevance is therefore thought to be in relation to socio-economic factors rather than exposure. Similarly with respect to the fact that there were more smokers in the homes of cases than controls, this is thought to reflect socio-economic factors rather than exposure via passive smoking. Even in smokers, it has been estimated that blood lead levels increase by six percent for every 10 cigarettes smoked per day (Hopper et al., 1982).

Particular attention was paid to the hand-mouth activities of children, and it was found from careful questioning that significant differences between the cases and controls existed with respect to a previous history of pica. A higher proportion of cases had a history of pica-related activity, although most children were not reported to have routinely ingested non-food items. Pica for paint was not a significant factor, but more cases than controls had been observed on occasion to eat items such as plaster, cement, soil, sticks and matchsticks. Most children did not pursue this habit beyond the age of four years.

Significant differences were also found between cases and controls with respect to the generalised mouthing activities of children, these being much more pronounced among cases than controls. There were also differences among the groups with respect to the proportion of cases observed to have noticeably dirty hands after play. Due to the possibility of confounding between factors, it was decided to carry out a multivariate analysis of the data.

7.10 MULTIVARIATE ANALYSES

Due to the relatively small numbers, logistic regression analyses were not considered appropriate. Thus from a list of the variables significant in the univariate analyses, a series of three variables was selected to be included together with the

variable 'group' (denoting classification as either a case or control) into a series of multi-way contingency tables. Statistically significant variables which were potentially indicative of the source or mechanism of exposure to lead were selected for entry into the multi-way tables. These included variables relating to the child's mouthing activities, pica (using the variable matchsticks), playsite, pots, home cleanliness index, outside flaking paint index, and interior surface dust-lead (mass-loading). Significant variables indicative of socio-demographic and economic standing which were selected for entry into the multi-way tables included those relating to the number of people living in the home, mother's schooling, occupational density and social class. Continuous variables were categorised as values less than or equal to the median and those above the median. The partial association between 'group' and one of the three factors (adjusted for the effects of the associations between group and the other two factors, as well as for the associations among all three factors) was obtained. The partial associations between 'group' and the other two factors were determined similarly. In this way it was possible to determine whether the 'exposure' variables were of significance when taking into account the socio-economic standing of the children.

The results of the multi-way contingency table analyses carried out are given in Table 7.16.

TABLE 7.16a

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(GROUP (A), MOUTHING BEHAVIOUR (B), MOTHER'S SCHOOLING (C), NUMBER OF PEOPLE (D))

EFFECT	CHISQUARE	"P" VALUE
AB	11.63	0.0006
AC	4.64	0.0312
AD	7.69	0.0056

TABLE 7.16b

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(GROUP (A), NUMBER OF PEOPLE (B), MOTHER'S SCHOOLING (C), PICA (D))

EFFECT	CHISQUARE	"P" VALUE
AB	5.00	0.0253
AC	4.51	0.0336
AD	5.82	0.0159

TABLE 7.16c

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(GROUP (A), NUMBER OF PEOPLE (B), MOTHER'S SCHOOLING (C), PLAY-SITE (D))

EFFECT	CHISQUARE	"P" VALUE
AB	2.30	0.1293
AC	3.33	0.0680
AD	1.39	0.2382

TABLE 7.16d

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(GROUP (A), NUMBER OF PEOPLE (B), MOTHER'S SCHOOLING (C), ALUM. POTS (D))

EFFECT	CHISQUARE	"P" VALUE
AB	4.32	0.0377
AC	4.20	0.0403
AD	2.04	0.1534

TABLE 7.16e

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(GROUP (A), NUMBER OF PEOPLE (B), MOTHER'S SCHOOLING (C), CLEANLINESS SCORE (D))

EFFECT	CHISQUARE	"P" VALUE
AB	1.96	0.1617
AC	1.20	0.2740
AD	3.41	0.0649

TABLE 7.16f

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(GROUP (A), NUMBER OF PEOPLE (B), MOTHER'S SCHOOLING (C), OUTSIDE SURFACES (D))

EFFECT	CHISQUARE	"P" VALUE
AB	3.38	0.0658
AC	3.72	0.0536
AD	2.51	0.1128

TABLE 7.16g

LOG-LINEAR MODEL

2-WAY INTERACTIONS

(GROUP (A), NUMBER OF PEOPLE (B), INTERIOR DUST LEAD (C), MOTHER'S SCHOOLING (D))

EFFECT	CHISQUARE	"P" VALUE
AB	3.19	0.0741
AC	2.07	0.1502
AD	4.67	0.0307

It was evident that there were no significant three-way interactions between factors, but there were significant two-way interactions between the variable 'group' and socio-economic indicators, as well as between 'group' and factors such as the child's mouthing behaviour and past history of pica-related activities. These findings are discussed below.

7.11 DISCUSSION

Whilst children did not exhibit symptoms of pica in the clinical sense of the term, there was nevertheless evidence of a past history of ingesting non-food items, although this was not pursued on a regular basis. Pica for paint, the main cause of exessively raised blood lead levels in children, was not found to be a significant factor in the range of blood lead levels studied here. The generalised mouthing activities of children were more

significant. Whilst the incidence of pica diminishes with increasing age in children, mouthing activities in older children may persist. This suggests a mechanism whereby these children may ingest lead, namely via the dust-hand-mouth pathway. Hands and play objects may become contaminated with lead-rich dust and children may ingest significant quantities of lead via the dust on their hands. This study suggests that those children with pronounced mouthing activities may ingest more lead than other children.

The importance of the dust-hand-mouth pathway was first emphasised by Sayre et al. in the mid 1970's. Sayre et al. (1974) compared the amount of lead on the hands of inner city children with suburban children aged nine months to six years and found that lead on children's hands was higher in the urban than suburban group.

In a study of exclusively inner city children, Charney et al. (1980) examined multiple sources and mechanisms of exposure in a group of children aged 18 months to six years, with blood lead levels between 40 and 70 ug/dl, and controls with blood lead levels below 29 ug/dl. House dust lead and lead on children's hands were found in significantly greater quantities among the experimental group. The high lead group also exhibited excessive mouthing activities and pica compared to the low lead group. A particular bias, however, may have had an influence on the results obtained. Mothers were aware of children's blood lead

levels, and had been questioned before the study about their children's habits. This could have biased the information obtained, particularly with respect to pica, which was a significant factor, especially for young black children.

In the present study, both parents and investigators were unaware of test results at the time that the home investigations were carried out. Also, in Charney et al.'s study, although the high and low lead groups were matched for socio-economic status, there was nevertheless a racial bias in the groups and the high lead group contained a larger proportion of subjects on medicaid.

Most workers who have found that lead in dust and the hand-mouth pathway is an important mechanism of exposure have studied children with very markedly elevated blood lead levels such as those living in certain urban areas or around lead smelters (Sayre et al., 1974; Lepow et al., 1974; Vostal et al., 1974; Lepow et al., 1975; Landrigan et al., 1975; Baker et al., 1977; Yankel et al., 1977; Schmitt et al., 1979; Charney et al., 1980; Roels et al., 1980; Gallacher et al., 1984). Other workers do not agree that lead in dust and soil is an important mechanism of exposure for young children (Ter Haar and Aronow, 1974; Barltrop et al., 1975).

In a trial on the effect of dust control measures on blood lead (Charney et al., 1983), it was found that subsequent to the intervention (which consisted of twice-monthly wet mopping at

children's homes and encouraging families to clean and wash children's hands frequently) blood lead levels among the cases dropped by 6.9 ug/dl as compared to 0.7 ug/dl among controls.

Charney et al.'s study has been criticised on the grounds that the cases and controls were not randomly assigned to their respective groups (Elwood and Gallacher, 1984). Also, there was a high drop-out rate among the subjects and it is not known how representative the final study group was.

The dust control programme was found to be most effective in children with very high lead levels (40 to 50 ug/dl) with little effect on reducing blood lead levels in the 30 to 35 ug/dl range. It therefore remains to be determined whether or not the control of household dust lead levels in the range reported in the present study will have a significant effect on reducing children's blood lead levels.

In the present study, when socio-economic/demographic factors were controlled for in the multivariate analysis, factors such as indoor dust were not statistically significant. It is therefore not clear whether indoor dust lead levels in the range reported here, contribute significantly to elevations in children's blood lead levels. It has been estimated that dust lead concentrations around 1 000 ppm contribute to raising children's blood lead levels by approximately two to five ug/dl (Duggan, 1980, 1983b; EPA, 1986c). In the present study, dust lead concentrations

averaged 500 ppm. These levels are slightly below the levels reported by Fergusson and Schroeder (1985) for old houses (pre 1950) which were found to average around 830 ppm. The measure of total lead available to the child (lead per unit area) is likely to be of more significance in raising blood lead levels. Whilst the levels of lead per unit area reported in this study were significantly lower than those reported by other workers, it should be noted that in the present study, carpeted areas representative of where children are likely to spend most of their time, were sampled. Other workers have sampled uncarpeted areas, which have higher lead concentrations than carpeted areas. (Solomon and Hartford, 1976), or have sampled areas where maximum concentrations of lead are likely to accumulate such as next to flaking windowsills.

There is disagreement about the relative contribution of various sources to indoor dust lead levels (Fergusson and Schroeder, 1985). The main sources of lead in house dust include paint, lead aerosols, soil and street dust. Dust lead levels in the homes of the children studied here are likely to derive from flaking painted surfaces, (which were found to have elevated lead concentrations, in many cases significantly above one percent) as well as from dust lead brought into the home from outside. As most of the homes had no gardens, soil is an unlikely contributor, but street dust may be of significance. Fergusson and Schroeder (1985) have suggested that in older houses with lead paint, roughly 50% of the lead in dust is likely to be

derived from petrol-derived lead aerosols and street dust, and the rest from paint. Other workers have suggested that in and around old houses with lead paint, paint is likely to be the major source of lead in dust (Ter Haar and Aronow, 1974; Culbard et al., 1983).

In the present study, as more cases than controls played more often outside than inside the home, it seems probable that outdoor sources may assume added significance, particularly as in the age group studied children play outside the home a great deal. The street dust lead levels in and around the home environments are elevated and are a potential source of lead, particularly for children whose habits expose them to this dust.

Coupled to the particular behavioural habits of the children, there was an overall lower level of supervision and stimulation among cases (measured by such factors as whether or not the child had (non-school) books at home or went on outings, for example, to museums. The larger number of people living in the homes of cases may also have a direct bearing on the quality of the parents' child-minding. Milar et al. (1980), Hunt et al. (1982), and Dietrich et al. (1985) have found that the quality of the care-giving environment may have an important influence on blood lead levels. Most of these studies however have assessed the significance of the quality of the care-giving environment for very young children aged two to three years and below.

Dietrich et al. (1985) found that a significant association between blood lead levels and the quality of the care-giving environment occurred, the strength of which increased with age in young children under the age of two years.

In the study by Milar et al. (1980) it was also found that deficits in the quality of the care-giving environment were significantly associated with increased blood lead levels ($\geq$ 31 ug/dl) in younger children, but not in older children (aged 31 to 78 months), although the results were in the same general direction as for younger children.

Hunt et al. (1982) studied 16 caretakers of hospitalised children with high blood lead levels and compared them to caretakers of children with low blood lead levels (mean = 30 ug/dl), on the basis of the quality of child care provided. Differences were found in the nature of the physical and emotional care provided. There were a number of biases in this study however. The study was not a double blind one and some caretakers had indicated the lead status of their children, i.e. that their children had been hospitalised. There was also a selection bias - for instance only those caretakers who were volunteers were selected for study.

Other factors, such as the differences found in the present study between cases and controls with respect to the mother's level of schooling and the relative level of affluence of the parents may

have a bearing on the house-keeping activities of parents. These factors relate also to the overall condition in which the home was maintained. In general, factors relating to the overall condition of the home (for example, the condition of the surfaces, the cleanliness of the home, the amount of lead rich dust in the home) are likely to be of significance in determining to what extent sources of lead present in the home environment become accessible. Thus, whilst significant levels of lead in potential sources such as paint or dust are a necessary prerequisite for exposure to occur and were shown to be present in the homes of both cases and controls, homes in a good state of repair, such as those of the controls, present fewer opportunities for potential sources to become accessible.

In general a lack of adequate parental care may result in a lack of supervision of children, which in turn may predispose children to be exposed to and ingest, sources of lead in the environment, found to be readily available in the homes of the high risk children studied.

It appears therefore that factors related to parental house-keeping practices have an important influence on the extent to which potential sources in the home environment become accessible to young children. In this urban community, where sources of lead were shown to be present in and around the homes of both cases and controls, the degree to which sources are <u>accessible</u>, becomes of increasing significance. Certain patterns of behaviour

exhibited among cases are of particular importance in determining to what extent ingestion of lead occurs. The degree of parental supervision may play a role in determining to what extent patterns of excessive mouthing are allowed to persist unmodified.

CHAPTER 8

CONCLUSIONS

8.1 RISK FACTORS FOR LEAD EXPOSURE

This study was concerned with risk factors for childhood lead exposure, in a school-age inner city population living in Cape Town, South Africa. In South Africa increased lead absorption occurs in certain urban areas, but there has been no systematic attempt to determine the risk factors for lead exposure in children. In order to implement appropriate preventive measures to control childhood lead exposure, it is necessary to determine the risk factors for environmental exposure to lead.

The failure to include relevant variables is one of the biggest problems in studies of childhood lead exposure. This study has elucidated several important variables that co-vary with lead in a particular South African inner-city community, with its unique character and individual features.

The coloured community living in the inner-city area of Woodstock was the main focus of the study. Due to the implementation of the Group Areas Act in South Africa, social stratification is not reflected in residential differentiation among the coloured population. The implication for the study area of Woodstock is

that the population is heterogenous with respect to socio-economic and other factors. This allowed for the consideration of many social as well as environmental risk factors in the study. In considering environmental exposure, there was a need to examine total exposure in the environment at large, as well as in and around the home environment. In considering social factors, there was a need to study economic, demographic, cultural and behavioural factors.

Two different study designs were used in investigating the risk factors for lead exposure in the school-age population (six to seven years) living in the inner-city. The study was thus carried out in two stages. In stage one a cross-sectional analytical study was carried out among first grade school children. The aims of this study were firstly, to determine the blood lead distribution of first grade inner city children living in the Woodstock area. Secondly, it was hoped to identify the risk factors associated with increased blood levels in children, using a questionnaire. Subjective (self-reported) measures of factors potentially associated with environmental exposure to lead were used. Anthropometric measurements and measurements of other haematological factors, were also performed on children, and their relationship with blood levels determined. An environmental study was carried out simultaneously, using objective measures of environmental lead, to determine average concentration levels of lead in the environment. The exposure measures took account of spatial and temporal variations in

environmental lead, a problem which has not been given adequate attention in many studies. Lastly, it was hoped to identify a smaller subset of children with high and low blood lead levels respectively, for an in-depth case control study.

The case control study was carried out in stage two of the study and used objective measures of environmental lead, as well as self reported measures relating mainly to social risk factors. The case control study studied simultaneously the relation at individual level between physical factors (air, water, dust, paint lead), their availability to the group (flaking surfaces, unclean, dusty homes) and the possible role of economic, demographic, cultural and, particularly, behavioural factors on increasing children's risk. This design improved on many ecological studies which have used measures of exposure at the group level and fail to account for the complex confounding and interaction of factors.

The overall findings were that in this inner city environment, with elevated levels of lead in the environment at large, social factors are of importance in determining the degree of exposure among the coloured population. Certain socio-demographic, cultural and economic factors were found to co-vary with lead. Although lead sources were available in the environment at large and in the home, social factors determine to what degree the

population is at risk. Thus the environment predisposes to risk, but is not sufficient to cause elevated blood lead levels to occur in children.

Baseline exposure in the environment is nevertheless of significance in influencing blood lead levels of children attending schools in direct proximity to traffic. Such children have elevated blood lead levels, which are raised independently of other potentially confounding factors.

For children with significantly elevated blood lead levels, the direct inhalation of lead aerosols and ingestion of lead in water were not found to be important risk factors. Hand-contamination and mouthing were found to be associated with increased risk. A history of pica was also important, but in most children pica had ceased, whilst mouthing persisted. In this community, where women occupy the central position in the family, parental supervision is likely to influence the child's behaviour, and particularly, hand-mouth activities. It seems likely that outside sources of environmental lead may be of particular significance, especially in this school-age population where cases were reported to play more outside than inside.

Some of the major findings of this study are summarised in more detail in the following sections, and a conceptual framework is proposed to illustrate how the various risk factors act to increase a child's risk of having an elevated blood lead level.

8.1.1 FACTORS AFFECTING THE DISTRIBUTION OF BLOOD LEAD LEVELS IN CHILDREN

It was established that the average blood lead level of the population was 15 ug/dl, with eight percent having blood lead levels $\geq$ 25 ug/dl, the United States of America action level (Section 4.1). A difference in blood lead levels with respect to ethnic group was found, coloured children having an average blood lead concentration of 18 ug/dl, and white children averaging 12 ug/dl, while 13% of coloured children, but no white children, had blood lead levels at or above the United States of America action level of 25 ug/dl (Section 4.1.1).

The differences in blood lead levels between the two groups is likely to be due in part to differences in socio-demographic and economic standing between the groups. Whilst both coloureds and whites in the area are predominantly working class, it was established that the annual mean income level among coloureds was substantially lower than among whites. Whites were also better educated and lived in larger homes with smaller families than coloureds (Section 5.1.3).

Using questionnaires as a survey instrument, risk factors related to certain environmental and socio-demographic, cultural and economic factors were determined (Sections 5.1.4 and 5.1.5).

Particular factors of importance which emerged in coloureds were socio-economic status and the nature of the child's family. Blood lead levels were inversely related to the level of affluence of the family and the educational level of the child's parents. In addition, children who came from overcrowded homes, single parent families, or families with many siblings had higher blood lead levels than others.

Cultural factors such as the child's religion and home language were also found to be related to blood lead levels. For instance, English speakers and children of the Islamic faith were found to have lower blood lead levels than Afrikaans speakers or children of the Christian faith. Both these factors are known to be related to socio-economic status in this community. There may also be a cultural component of importance, for instance the emphasis placed in Islam on personal bodily cleanliness. Also, the Muslim family is a close-knit entity which provides protection and support for the proper care of children.

Factors related to the home environment were also found to be predictors of blood lead levels. For instance, the reported state of repair of the home, the level of dust in the home and the proximity of factories to the home emerged as being significant. None of the factories in the area were major contributors to lead emissions in the ambient air, and are not thought to be important factors related to exposure.

Children from homes reported to be in a poor state of repair and children from dusty homes had higher blood lead levels than other children. These factors are in turn related to socio-economic factors and were not significant when socio-economic factors were controlled (Section 5.1.5.2). Water piping in the majority of homes was made of copper and only one or two homes reported the presence of lead water pipes.

There was also a relationship between the nature of the child's main play site and blood lead levels, although this did not reach statistical significance among coloured children. Among coloured children, playing mainly in the back yard or on the street was associated with higher blood lead levels, and playing inside or in the front yard was associated with lower blood lead levels. Playing mainly in the house was also protective for white children, but playing in the front yard was not. This relationship was statistically significant. This finding suggests that exposure to lead may be of more significance for this age group in the outer environment than in the inside home environment.

Certain dietary items were found to be associated with blood lead levels, but the specific food items of importance differed for the coloured and white groups. In general however, the overall nutritional status of the groups was satisfactory and children were not undernourished. From haemoglobin, haematocrit and red

cell level determinations, it was established that small differences occurred between groups, but in general they were not suffering from any abnormalities of the blood such as anaemia.

8.1.1.1 Effect of School

It was established that significant differences in blood lead concentrations occurred at the various schools which were attended by children (Section 4.1.4). No significant differences in nutritional status were found between schools grouped on the basis of their residential areas (Section 4.3). There was also a relationship between the residential zone where children lived and their blood lead levels (Section 4.1.4). For instance, children living in zone three had lower blood lead levels than children living in zones one and two. Nevertheless, the zone where children lived was not found to be significant when the school attended by the child was taken into account. The factor 'school' remained significant, irrespective of the socio-economic status of the children, when a contingency table analysis was carried out (Section 5.1.5.2).

There was a strong association between blood lead levels and the geographic position of schools with respect to traffic density - children attending schools situated in direct proximity to high density traffic had blood lead levels five to seven ug/dl higher than children attending other schools. Atmospheric and dust lead

concentrations were determined in the environmental study, which was carried out simultaneously with the blood lead and questionnaire survey (Chapter Six).

8.1.1.2 Lead in the Environment at Large

From the environmental surveys of air and dust lead levels carried out in Woodstock, it was established that air lead levels varied from less than 0.5 ug/m^3 to above 1.5 ug/m^3 (Section 6.1.1), and dust lead concentrations varied from less than 550 ppm to above 3 000 ppm (Section 6.2).

Air and dust lead levels were significantly elevated at sites situated directly on major roads (Section 6.1.1.). Air lead levels at sites situated on major roads were about two fold higher than at sites situated 200-300 metres away from major roads, with air lead levels being highest at sites with the highest density of traffic. At sites situated on major roads, air lead levels averaged around 1.5 ug/m^3: at sites away from major roads, air lead levels were around 0.8 ug/m^3. Traffic had an influence on dust lead levels, which were similarly elevated at sites on major roads (Section 6.2).

Air lead levels were also found to vary during the week, with the highest concentrations occurring on Fridays when traffic volumes peak, and the lowest occurring on Sundays, when traffic is at a

minimum (Section 6.1.2.2). Minor industrial sources did not appear to play a major role in influencing air lead levels in the area studied; the major influence coming from traffic as indicated by both spatial and daily variations.

Air lead levels were found to be on average two-fold higher in the winter months than in the summer (Section 6.1.2.1). During the winter period in particular, air lead levels at certain sites on major roads exceed the EPA standard of 1.5 ug/m^3 on occasions.

A figure of 0.8 ug/m^3 represents the yearly average baseline level of atmospheric lead exposure of the population. In addition, children exposed to heavy traffic densities (for instance, at school and during play) can be expected to be exposed to higher levels of lead in the environment, especially during winter months, when levels may reach 1.5 to two ug/m^3 on major roads. The range of air lead levels reported in this study supports the contention that direct inhalation of aerosols is unlikely to make a major contribution to the exposure of the general population. Exposure via indirect pathways (for example, dust) may be of more significance in raising blood lead levels a few ug/dl for sectors of the population unduly exposed. These may include high risk sectors of the population such as children who attend schools on major roads and children who play regularly on the streets. Dust lead concentrations are significantly elevated on major roads.

In general, this part of the study shows that for this school age population aged six to seven years, exposed overall to air lead levels of 1.3 - 1.4 ug/m^3 during winter months preceding the blood lead tests, and to between about 500 and 3 500 ppm in dust lead concentrations, it was shown that blood lead concentrations are unlikely to be below 12 ug/dl among whites and 18 ug/dl among coloureds.

8.1.2 HIGH-RISK CHILDREN AND LEAD IN THE HOME ENVIRONMENT

From the case control study of high risk children with elevated blood lead levels (Chapter Seven), it was established that factors related to the condition of children's homes, and certain behavioural characteristics of children, were associated with raised blood lead levels. The homes of children with blood lead levels greater than or equal to 24 ug/dl were found to be in a worse state of repair than the homes of children with blood lead levels below 15 ug/dl. The homes had more flaking surfaces, more lead-rich dust and an overall lower level of cleanliness (Section 7.4).

Paint with a concentration of one percent lead or more was found to be present in the homes of 44% of cases and 26% of controls, but this difference was not statistically significant (Section 7.4.2). Lead rich dust was also found to be present in the homes of both cases and controls, but a significantly greater

proportion of cases (50%) than controls (19%) had elevated dust lead levels expressed in terms of mass loading (total lead per unit area). The homes of both cases and controls had mean dust lead concentrations of around 500 ppm (Section 7.4.3).

Factors such as the amount of flaking surfaces and lead-rich dust were not statistically significant when confounding socio-economic/demographic factors such as mother's schooling and occupational density were taken into account (Section 7.10).

Air lead levels and street dust lead levels at the homes of children were not found to vary significantly between cases and controls, nor did the lead concentration in water samples (Sections 7.4.4 and 7.4.5). No lead pipes were found among the accessible water pipes examined. There was also no major difference in the nutritional status of these high and low blood lead level children, nor in their dietary habits as reported by responders (Sections 7.5 and 7.6).

Significant differences were found with respect to certain behavioural characteristics of cases and controls. Careful questioning revealed a past history of pica-related activity; these were associated with a broad range of items such as cement, soil and matchsticks. Most children did not routinely ingest non-food items and the habit had only been pursued on occasion, according to responders. Most had not pursued the habit beyond the age of four (Section 7.1.1).

A much higher proportion of cases than controls were found to engage in pronounced mouthing activities, having been regularly observed to put their hands and objects into their mouths (Section 7.1.2). This suggests a mechanism whereby children might ingest lead, namely via the dust-hand-mouth pathway. There was also evidence that cases received less supervision and stimulation than controls (Section 7.3). The larger family size among the cases may play an important role in affecting child-minding. Large family size could provide opportunity for children's behaviour to proceed unmodified, and for them to be more exposed to sources of accessible lead present in the home environment and the environment at large.

It is not clear whether or not potential sources such as lead rich dust inside or outside the home are of more significance. Cases were observed more frequently to play outside than inside the home (Section 7.2). Children playing in busy streets may ingest significant quantities of lead rich dust. It is likely that both inside and outside sources play a role, although outside sources may be of particular significance for older children.

8.1.3 A CONCEPTUAL FRAMEWORK FOR CHILDHOOD LEAD EXPOSURE

This study has shown that in the presence of environmental lead, coloured children of low socio-economic status are at particular risk. Also at increased risk are coloured and white children who attend schools in the proximity of heavy density traffic.

In the inner-city environment studied here, where lead is a pervasive source, the social circumstances of children determine to a large degree the extent to which they are at risk. Some of the risk factors for lead exposure were: being coloured, Afrikaans speaking, of the Christian faith, attending school in the proximity of high density traffic, living in old, dilapidated housing with flaking lead-based painted surfaces, lead rich dust, living in small, overcrowded homes, where family income is low, parental educational level is low, there is a lack of stimulation and parental supervision of children, children mouth on dirty hands and other objects and play more often outside than inside.

It is suggested that where lead is present in the environment (for instance in and around the home, at school), the potential for exposure exists. As indicated earlier, lead in the environment is necessary, but not always sufficient for exposure to occur. It is proposed that cultural and economic components influence parental house-keeping abilities and the degree to which lead in the environment is accessible to children. Thus for instance, being of the Islamic faith may influence the house-

keeping practices of families, as emphasis is placed on personal hygiene and cleanliness in the home. This could in turn reduce exposure to lead rich dust in the home. Economic factors too, influence the house-keeping practises of families, as well as the state of housing directly. Where resources are limited, homes may be kept in a deteriorating condition with flaking paint and lead rich dust.

Demographic factors such as overcrowded homes and large families can limit the ability of parents to supervise children's behaviour. This can lead to children's play-sites being unsupervised and the hand-mouth activities proceeding unmodified, which may directly lead to ingestion of lead by children. Where dust lead levels are elevated, but not excessively so, children's mouthing behaviour may be of particular importance.

There is a need for further studies to incorporate hand-lead measurements of children, and for studies to incorporate follow-up designs so that the temporal sequence of events can be determined with greater certainty. Also, larger studies are needed so that the proportion of variability in blood lead levels due to the various risk factors can be quantified.

8.2 IMPLICATIONS OF THE STUDY FOR THE PREVENTION OF CHILDHOOD LEAD EXPOSURE

The results of this study suggest that every effort should be made in South Africa to lower the baseline blood lead concentration of inner-city high risk populations. This would increase the safety margin for children unduly exposed to lead by way of their behaviour, family circumstances, home condition or other factors. Primary prevention strategies that are independent of individual social, economic, behavioural or cultural factors may be successful in reducing population lead levels, providing a small benefit to all individuals. They usually involve long term strategies and considerable economic costs, however.

In the United States of America and Europe, the source most amenable to control has been lead in petrol. In the period during which this study was carried out, South Africa had a maximum level of lead in petrol of 0.836 g/l. Acknowledgement on the part of the government of the contribution of this source to lead in the environment, and the recent steps taken to lower this level to 0.6 g/l are welcomed. Nevertheless, considerable further restrictions on lead in petrol will be required to bring South Africa into line with the rest of the Western world. The environmental lead levels in the urban area studied are elevated, particularly in the winter months, and substantial reductions in the lead content of petrol, the major contributor to air lead

levels, will have a positive effect on air quality in these areas. It is hypothesised that blood lead levels of pupils attending schools in direct proximity to heavy density traffic will drop, if controls on lead in petrol are introduced.

Planning of the siting of institutions such as schools, creches and hospitals, should take into account the volume of traffic at proposed sites and every effort should be made to site such institutions away from major roads with high traffic densities.

An attempt should also be made to limit other sources of lead in the environment. The lead concentration in modern decorating paints applied on surfaces likely to be chewed by children should be restricted. In particular, the lead content used on items such as pencils and toys should be stringently regulated to protect children with pronounced hand-mouth activities.

Prevention strategies aimed at the individual (both parent and child), modifying their behaviour (improving housekeeping practices, decreasing house dust, improving child supervision to reduce mouthing and playing in high risk environments such as busy streets) are also necessary. It is recognized however that these strategies are difficult to implement without improving the social and economic circumstances of children's families, which is so urgently needed in this country.

An intermediate approach involves the stripping of highly leaded painted surfaces found in homes with deteriorating surfaces which are accessible to young children. This would also reduce the amount of lead rich dust in and around the home. The costs of these approaches, if borne at national, local or regional level may effectively reduce children's opportunity for exposure.

Secondary prevention strategies are also necessary, which aim to identify high risk children, reduce their body lead burden and risk of subsequent exposure. There is an urgent need to screen in low socio-economic, inner city areas like Woodstock, in other parts of the country. Once high risk areas are identified, resources can be targetted to young children and families at highest risk. With rapid urbanisation in South Africa, it is likely that the inner city areas will deteriorate further, placing increased cohorts of children at risk of lead exposure.

In general, an educational effort should be directed at the public in general and the authorities in particular, to make people aware of the sources of lead in the environment and of risk factors for lead exposure.

REFERENCES

Aggarwal, A.L., Patel, T.S., Rayani, C.V., Chatterjee, S.K., 1979 : Biologic Effects of Airborne Lead on Occupationally Exposed Traffic Policemen and Permanent Shopkeepers Stationed at Ahmedabad City. The Indian Journal of Medical Research 70 : 650-656.

Alexander, F.W., 1974 : The Uptake of Lead by Children in Differing Environments. Environmental Health Perspectives May : 155-160.

American Academy of Pediatrics, 1969 : Prevention, Diagnosis and Treatment of Lead Poisoning in Childhood. Pediatrics 44 (2) : 291-298.

Anagnostopolous, A., 1983 : Air Lead and Dust Lead in Thessaloniki. In : Lead Versus Health : Sources and Effects of Low Level Lead Exposure. Eds. : M. Rutter and R. Russell Jones. John Wiley and Sons, New York.

Annest, J.L., Pirkle, J.L., Makuc, D., Neese, J.W., Bayse, D.D., Kovar, G., 1983 : Chronological Trend in Blood Lead Levels Between 1976 and 1980. The New England Journal of Medicine 308 (23) : 1373-1377.

Annual Report of the Medical Officer of Health of the Divisional Council of the Cape, 1986 : Cape Town.

Archer, A., Giltrow, J.P., Waldron, H.A., 1980 : Blood Lead Concentrations in Pre-school Children in Birmingham. Journal of the Royal Society of Medicine 73 (5) : 328-332.

Archer, A., Barratt, R.S., 1976 : Lead Levels in Birmingham Dust. The Science of the Total Environment 6 : 275-286.

Badham, A., 1987: Rediscovering Woodstock. Contree 21 : 21-25.

Baker, E.L., Folland, D.S., Taylor, T.A., Frank, M., Peterson, W., Lovejoy, G., Cox, D., Houseworth, J., Landrigan, P.J., 1977 : Lead Poisoning in Children of Lead Workers. The New England Journal of Medicine 296 : 260-261.

Barltrop, D., 1966 : The Prevalence of Pica. American Journal of Diseases of Childhood 112 : 116-123.

Barltrop, D., Thornton, I., Strehlow, C.D., Webb, J.S., 1975 : Absorption of Lead from Dust and Soil. Postgraduate Medical Journal 51 : 801-804.

Batson, E., 1944 : Notes on the Concept and Measurement of Overcrowding. Report SS27. University of Cape Town, Rondebosch, South Africa.

Bellinger, D.C., Needleman, H.L., 1983 : Lead and the Relationship Between Maternal and Child Intelligence. Journal of Pediatrics (St. Louis) 102 : 523-527.

Bellinger, D.C., Needleman, H.L., Leviton, A., Waternaux, C., Rabinowitz, M.B., Nichols, M.L., 1984 : Early Sensory - Motor Development and Prenatal Exposure to Lead. Neurobehavioural Toxicology and Teratology 6 : 387-402.

Bellinger, D.C., Leviton, A., Waternaux, C., Needleman, H., Rabinowitz, M., 1987 : Longitudinal Analyses of Prenatal and Postnatal Lead Exposure and Early Cognitive Development. The New England Journal of Medicine 316 (17) : 1037-1043.

Billick, I.H., Curran, A.S., Shier, D.R., 1979 : Analysis of Pediatric Blood Levels in New York City for 1970-1976. Environmental Health Perspectives 31 : 183-190.

Billick, I.H., Curran, A.S., Shier, D.R., 1980 : Relation of Pediatric Blood Lead Levels to Lead in Gasoline. Environmental Health Perspectives 34 : 213-217.

Blackfan, K.D., 1917: Lead Poisoning in Children With Especial Reference to Lead as a Cause of Convulsions. American Journal of Medical Science 153 : 877-887.

Blanksma, L.A., Sachs, H.K., Murray, E.F., O'Connell, M.J., 1969 : Incidence of High Blood Lead Levels in Chicago Children. Pediatrics 44 (5) : 661-667.

Bornschein, R.L., Rabinowitz., M.B., 1985 : (Foreward). The Second International Conference on Prospective Studies of Lead. Cincinnati, Ohio, April 9-11 1984. Environmental Research 38 : 1-2.

Bradley, J.E., Powell, A.E., Niermann, W., McGrady, K.R., Kaplan, E., 1956 : The Incidence of Abnormal Blood Levels of Lead in a Metropolitan Pediatric Clinic. The Journal of Pediatrics 49 (1) : 1-6.

British Medical Association, 1922 : An Historical Account of the Occurrence and Causation of Lead Poisoning Among Queensland Children. The Medical Journal of Australia 1 : 148-152.

Brunekreef, B., Veenstra, S.J., Biersteker, K., Boleij, J.S.M., 1981 : The Arnehm Lead Study : 1. Environmental Research 25 : 441-448.

Brunekreef, B., Noy, D., Biersteker, K., Boleij, J., 1983 : Blood Lead Levels of Dutch City Children and Their Relationship to Lead in the Environment. Journal of the Air Pollution Control Association 33 (9) : 872-876.

Brunekreef, B.D., 1984 : The Relationship Between Air Lead and Blood Lead in Children : A Critical Review. The Science of the Total Environment 38 : 79-123.

Burrows, N.F., Rendle-Short, J., Hanna, D., 1951 : Lead Poisoning in Children. Report of Five Cases. With Special Reference to Pica. British Medical Journal 1 : 329-334.

Byers, R.K., Lord, E.E., 1943 : Late Effects of Lead Poisoning on Mental Development. American Journal of Diseases of Children 66 (5) : 471-494.

Byers, R.K., 1959 : Review of the Literature and Report on 45 Cases. Pediatrics 23 : 585-603.

Calder, I.C., Roder, D.M., Esterman, A.J., Lewis, M.J., Harrison, M.C., Oldfield, R.K., 1986 : Blood Lead Levels in Children in the North-West of Adelaide. The Medical Journal of Australia 144 : 509-513.

Cape Town Chamber of Commerce, 1986 : Deproclaim Woodstock. No. 44 1986-11-05.

Caprio, R.J., Margulis, H.L., Joselow, M.M., 1974 : Lead Absorption in Children and its Relationship to Urban Traffic Density. Archives of Environmental Health 28 : 195-197.

Centres for Disease Control, 1985 : Preventing Lead Poisoning in Young Children. A Statement by the Centres for Disease Control. U.S. Department of Health and Human Services, USA.

Chamberlain, A.C., Clough, W.S., Heard, M.J., Newton, D., Stott, A.N.B., Wells, A.C., 1975 : Uptake of Inhaled Lead from Motor Exhaust. Postgraduate Medicine Journal 51 : 790-794.

Chamberlain, A.C., 1983 : Effect of Airborne Lead on Blood Lead. Atmospheric Environment 17 (4) : 677-692.

Chambers, J.M., Cleveland, W.S., Kleiner, B., Tukey, P.A., 1983 : Graphical Methods for Data Analysis. Wadsworth International Group, Belmont, California.

Charney, E., Sayre, J., Coulter, M., 1980 : Increased Lead Absorption in Inner City Children : Where Does the Lead Come From? Pediatrics 65 (2) : 226-231.

Charney, E., Kessler, B., Farfel, M., Jackson, D., 1983 : Childhood Lead Poisoning : A Controlled Trial of the Effect of Dust-Control Measures on Blood Lead Levels. The New England Journal of Medicine 309 : 1089-1093.

Chisholm, J.J., Harrison, H.E., 1956 : The Exposure of Children to Lead. Pediatrics 18 : 943-957.

Chisholm, J.J. Kaplan, E., 1968 : Lead Poisoning in Childhood - Comprehensive Management and Prevention. The Journal of Pediatrics 73 (6) : 942-950.

Chisholm, J.J., 1973 : Management of Increased Lead Absorption and Lead Poisoning in Children. The New England Journal of Medicine 289 (19) : 1016-1018.

Chisholm, J.J., 1985 : Ancient Sources of Lead and Lead Poisoning in the United States Today. The Western Journal of Medicine 143 (3) : 380-381.

Christian, J.R., Celewycz, B.S., Andelman, S.L., 1964 : A Three-Year Study of Lead Poisoning in Chicago. Part I : Epidemiology. American Journal of Public Health 54 (8) : 1241-1245.

Clark, N.S., 1950 : Lead Poisoning in Infancy. Archives of Disease in Childhood 25 : 297-301.

Cohen, C.J., Bowers, G.N., Lepow, M.L., 1973 : Epidemiology of Lead Poisoning - A Comparison Between Urban and Rural Children. The Journal of the American Medical Association 226 (12) : 1430-1433.

Cohen, G.J., Ahrens, W.E., 1959 : Chronic Lead Poisoning : A Review of Seven Years Experience at the Children's Hospital, District of Columbia. The Journal of Pediatrics 54 (3) : 271-284.

Commission of Inquiry Into Occupational Health (Erasmus Commission), 1976 : Government Gazette RP55/1976. Pretoria, South Africa.

Culbard, E., Thornton, I., Watt, J., Moorcroft, S., Brooks, K., Thompson, M., 1983 : Metal Contamination of Dusts and Soils in Urban and Rural Households in the United Kingdom. 2. In : Trace Substances in Environmental Health - XVII A Symposium. Ed. : D. Hemphill. University of Missouri, Columbia, USA.

Daines, R.H., Smith, D.W., Feliciano, A., Trout, J.R., 1972 : Air Levels of Lead Inside and Outside of Homes. Industrial Medicine and Surgery 41 : 26-28.

David, O.J., Wintrob, H.L., Arcoleo, C.G., 1982 : Blood Lead Stability. Archives of Environmental Health 37 (3) : 147-151.

Davies, J.C.A., 1985 : Lead Toxicity in South Africa (Editorial). South African Medical Journal 67 (21) : 833-834.

Davis, J.M., Svendsgaard, P.J., 1987 : Lead and Child Development. Nature 329 (6137) : 297-300.

Day, J.P., Hart, M., Robinson, M.S., 1975 : Lead in Urban Street Dust. Nature 253 (Jan 31) : 343-345.

De Silva, P.E., Donnan, M.B., 1980 : Blood Lead Levels in Victorian Children. The Medical Journal of Australia : 2(6) : 315-319.

Denny, F.M., 1985 : An Introduction to Islam. Macmillan, New York.

Department of Health and Social Security, 1980 : Lead and Health. Report of a DHSS Working Party on Lead in the Environment. HMSO, London.

Department of Health, 1979 : Lead Poisoning in Children. Department of Health, Pretoria, South Africa.

Deveaux, P., Kibel, M.A., Dempster, W.S., Pocock, F., Formenti, K., 1986 : Blood Lead Levels in Preschool Children in Cape Town. South African Medical Journal 69 : 421-424.

Dewar, D., Uytenbogaardt, R., 1978 : Housing : A Comparative Evaluation of Urbanism in Cape Town. Urban Problems Research Unit, University of Cape Town, Cape Town, South Africa.

Dietrich, K.N., Krafft, K.M., Pearson, D.T., Harris, L.C., Bornschein, R.L., Hammond, P.B., Succop, P.A., 1985 : Contribution of Social and Developmental Factors to Lead Exposure During the First Year of Life. Pediatrics 75 (6) : 1114-1119.

Dixon, W.S., 1981 : P4F Program of the BMDP Statistical Software. Berkeley, California.

Drossos, C.G., Mavroidis, K., Papadopoulou-Daifotis, Z., Michalodimitrakis, D.N., Salamalikis, L.X., Gounaris, A.K., Varonos, D.D., 1982 : Environmental Lead Pollution in Greece. American Industrial Hygiene Association Journal 43 (10) : 796-798.

Duggan, M.J., Williams, S., 1977: Lead in Dust in City Streets. The Science of the Total Environment 7 : 91-97.

Duggan, M.J., 1980 : Lead in Urban Dust : An Assessment. Water, Air and Soil Pollution 14 : 309-321.

Duggan, M.J., 1983a : The Uptake and Excretion of Lead by Young Children. Archives of Environmental Health 38(4) : 246-247.

Duggan, M.J., 1983b : Contribution of Lead in Dust to Children's Blood Lead. Environmental Health Perspectives 50 : 371-381.

Elwood, P.C., Gallacher, J., Toothill, C., 1982 : Lead in Petrol. British Medical Journal 284 : 1189-1190.

Elwood, P.C., 1983 : Changes in Blood Lead Concentrations in Women in Wales 1972-1982. British Medical Journal 286 : 1553-1555.

Elwood, P.C., 1984 : Abuses of Epidemiology in Environmental Research. In : Proceedings International Conference on Environmental Pollution: 249-256. July 1984, London.

Elwood, P.C., Gallacher, J., 1984 : Effect of Dust Control on Blood Lead. (Letter). The New England Journal of Medicine 310 (14) : 924-925.

Elwood, P.C., 1986 : The Sources of Lead in Blood : A Critical Review. The Science of the Total Environment 52 : 1-23.

Environmental Protection Agency, 1986a : Air Quality Criteria for Lead. Vol I of IV. EPA-600/8-83/028aF. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina, U.S.A.

Environmental Protection Agency, 1986b : Air Quality Criteria for Lead. Vol II of IV. EPA-600/8-83/028bF. Environmental Criteria and Assessment Office. Research Triangle Park, North Carolina, U.S.A.

Environmental Protection Agency, 1986c : Air Quality Criteria for Lead. Vol III of IV. EPA-600/8-83/028cF. Environmental Criteria and Assessment Office. Research Triangle Park, North Carolina, U.S.A.

Environmental Protection Agency, 1986d : Air Quality Criteria for Lead. Vol IV of IV. EPA-600/8-83/028dF. Environmental Criteria and Assessment Office. Research Triangle Park, North Carolina, U.S.A.

Fagan, G., 1986 : Western Woodstock. Urban Conservation Studies, Gabriel Fagan Architects, Cape Town.

Farah, C.E., 1970 : Islam : Beliefs and Observances. Woodbury, New York.

Fergusson, J.E., Simmonds, P.R., 1983 : Heavy Metal Pollution at an Intersection Involving a Busy Urban Road in Christchurch, New Zealand. 1. Levels of Cr, Mn, Fe, Ni, Cu, Zn, Cd and Pb in Street Dust. New Zealand Journal of Science 26 : 219-228.

Fergusson, J.E., Ryan, D.E., 1984 : The Elemental Composition of Street Dust From Large and Small Urban Areas Related to City Type, Source and Particle Size. The Science of the Total Environment 34 : 101-116.

Fergusson, J.E., Schroeder, R.J., 1985 : Lead in House Dust of Christchurch, New Zealand : Sampling, Levels and Sources. The Science of the Total Environment 46 : 61-72.

Fine, P.R., Thomas, C.W., Suhs, R.H., Cohnberg, R.E., Flashner, B.A., 1972 : Pediatric Blood Lead Levels. A Study in 14 Illinois Cities of Intermediate Population. The Journal of the American Medical Association 221 (13) : 1475-1479.

Flindt, M.L.H., King, E., Walsh, D.W., 1976 : Blood Lead and Erythrocyte Aminolaevulinic Acid Dehydratase Levels in Manchester Taxi Drivers. British Journal of Industrial Medicine 33 : 79-84.

Fosse, G., Wesenberg, G.B.R., 1981 : Lead, Cadmium, Zinc and Copper in Deciduous Teeth of Norwegian Children in the Pre-Industrial Age. International Journal of Environmental Studies 16 : 163-170.

Freeman, R., 1970 : Chronic Lead Poisoning in Children. A Review of 90 Children Diagnosed in Sydney 1948-1967 1 : Epidemiological Aspects. The Medical Journal of Australia 1 : 641-647.

Fulton, M., Thomson, G., Hunter, R., Raab, G., Laxen, D., Hepburn, W., 1987 : Influence of Blood Lead on the Ability and Attainment of Children in Edinburgh. The Lancet 1 (8544) : 1221-1226.

Gallacher, J.E.J., Elwood, P.C., Phillips, K.M., Davies, B.E., Jones, D.T., 1984 : Relation Between Pica and Blood Lead in Areas of Differing Lead Exposure. Archives of Disease in Childhood 59 : 40-44.

Garnys, V.P., Freeman, R., Smythe, L.E., 1979 : Lead Burden of Sydney School Children. Department of Analytical Chemistry, University of New South Wales, Australia.

Garside, J., 1987 : Open Areas in Cape Town - The Struggle for Their Identity. Regional Topic Paper 87/8. South African Institute of Race Relations, Cape Western Region, South Africa.

Gibson, J.L., 1904 : A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning Amongst Queensland Children. The Australian Medical Gazette 23 : 149-153.

Gibson, J.L., 1917 : The Diagnosis, Prophylaxis and Treatment of Plumbic Ocular Neuritis Amongst Queensland Children. The Medical Journal of Australia 2 : 201-204.

Gilfillan, S.C., 1965 : Lead Poisoning and the Fall of Rome. Journal of Occupational Medicine 7 : 53-60.

Gillet, J.A., 1955 : An Outbreak of Lead Poisoning in the Canklow District of Rotherham. The Lancet 1 : 1118-1121.

Goldsmith, J.R., Hexter, A.C., 1967 : Respiratory Exposure to Lead : Epidemiological and Experimental Dose-Response Relationships. Science 158 : 132-134.

Goldwater, L.J., Hoover, A.W., 1967 : An International Study of "Normal" Levels of Lead in Blood and Urine. Archives of Environmental Health 15 : 60-63.

Gordon, I., Whitehead, T.P., 1949 : Lead Poisoning in an Infant from Lead Nipple-Shields. The Lancet 2 : 647-652.

Grandjean, P., Nielsen, O.V., Shapiro, I.M., 1979 : Lead Retention in Ancient Nubian and Contemporary Populations. Journal of Environmental Pathology and Toxicology 2 : 781-787.

Greenberg, M., Jacobziner, H., McLaughlin, M.C., Fuerst, H.T., Pelliteri, O., 1958 : A Study of Pica in Relation to Lead Poisoning. Pediatrics 22 (4) : 756-760.

Greengard, J., 1966a : Lead Poisoning in Childhood. Clinical Pediatrics 5 (5) : 269-276.

Greengard, J., 1966b : Lead Poisoning in Children (Foreword). Clinical Pediatrics 5 (5) : 268.

Griggs, R.C., Sunshine, I., Newill, V.A., Newton, B.W., Buchanan, S., Rasch, C.A., 1964 : Environmental Factors in Childhood Lead Poisoning. The Journal of the American Medical Association 187 (10) : 703-707.

Grobler, S.R., Rossouw, R.J., Maresky, L.S., 1985 : Blood Lead Levels in a Remote, Unpolluted Rural Area in South Africa. South African Medical Journal 68 (5) : 323-324.

Gross, S.B., 1979 : Oral and Inhalation Lead Exposures in Human Subjects (Kehoe Balance Experiments). Department of Environmental Health, University of Cincinnati, U.S.A.

Gross, S.B., 1981 : Human Oral and Inhalation Exposures to Lead : Summary of Kehoe Balance Experiments. Journal of Toxicology and Environmental Health 8 : 333-377.

Hammond, P.B., 1977 : Exposure of Humans to Lead. Annual Review of Pharmacology and Toxicology 17 : 197.

Hammond, P.B., O'Flaherty, E.J., Gartside, P.S., 1981 : The Impact of Air Lead on Blood Lead in Man - A Critique of the Recent Literature. Food, Cosmetics and Toxicology 19 : 631-638.

Harris, I., 1976 : Lead Encephalopathy. South African Medical Journal 50(35) : 1371-1373.

Harrison, R.M., 1979 : Toxic Metals in Street and Household Dusts. The Science of the Total Environment 11 : 89-97.

Harrison, R.M., Laxen, D.P.H., 1981 : Lead Pollution : Causes and Control. New York, NY : Chapman and Hall.

Harvey, P., Hamlin, M., Kumar, R., 1983 : The Birmingham Blood Lead Study. Paper given at the Annual Conference of the British Psychological Society. York 10th April 1983.

Hecker, L.H., Allen, H.E., Dinman, B.D., Neel, J., 1974 : Heavy Metals Levels in Acculturated and Unacculturated Populations. Archives of Environmental Health 29 : 181-185.

Hinton, D., Coope, P.A., Malpress, W.A., Janus, E.D., 1986 : Trends in Blood Lead Levels in Christchurch (NZ) and Environs 1978-85. Journal of Epidemiology and Community Health 40 : 244-248.

Hirai, I., 1927 : Meningism of Sucklings in Japan from Lead Poisoning. Archives of Pediatrics 44 : 137-138.

Hofreuter, D.H., Catcott, E.J., Keenan, R.G., Xintaras, C., 1961 : The Public Health Significance of Atmospheric Lead. Archives of Environmental Health 3 : 568-574.

Holt, L.E., 1923 : Lead Poisoning. American Journal of Diseases of Children 25 : 229-233.

Hopper, J.L., Balderas, A., Mathews, J.D., 1982 : Analysis of Variation in Blood Lead Levels in Melbourne Families. The Medical Journal of Australia 2 (12) : 537-577.

Hunt, T.J., Hepner, R., Seaton, K.W., 1982 : Childhood Lead Poisoning and Inadequate Child Care. American Journal of Diseases of Children 136 : 538-542.

Hunter, D., 1978 : The Ancient Metals (Chapter 5). In : The Diseases of Occupations. Hodder and Stoughton, London.

Hunter, J., 1986 : The Distribution of Lead. In : The Lead Debate : The Environment, Toxicology and Child Health. Eds : R. Lansdown and W. Yule. Croom Helm, London and Sydney.

Jacobziner, H., Raybin, H.W., 1962 : The Epidemiology of Lead Poisoning in Children. Archives of Pediatrics 79 : 72-76.

Jacobziner, H., 1966 : Lead Poisoning in Childhood : Epidemiology, Manifestations and Prevention. Clinical Pediatrics 5 (5) : 277-286.

Johnson, D.E., Tillery, J.B., Prevost, R.J., 1975 : Levels of Platinum, Palladium, and Lead in Populations of Southern California. Environmental Health Perspectives 12 : 27-33.

Kato, K., 1932 : Lead Meningitis in Infants. American Journal of Diseases of Children 44 : 569-591.

Kawai, M., Okamoto, Y., Katagiri, Y., 1987 : Variation of Blood Lead Levels With Age in Childhood. International Archives of Occupational and Environmental Health 59 : 91-94.

Kehoe, R.A., 1961a : The Metabolism of Lead in Man in Health and Disease : the Normal Metabolism of Lead. Journal of the Royal Institute of Public Health and Hygiene 24 : 81-97.

Kehoe, R.A., 1961b : The Metabolism of Lead in Man in Health and Disease : the Metabolism of Lead Under Abnormal Conditions. Journal of the Royal Institute of Public Health and Hygiene 24 : 129-143.

Kehoe, R.A., 1961c : The Metabolism of Lead in Man in Health and Disease : Present Hygienic Problems Relating to the Absorption of Lead. Journal of the Royal Institute of Public Health and Hygiene 24 : 177-203.

Kilroe-Smith, T.A., 1987 : 'Lead' Up The Garden Path. South African Journal of Science 83 : 672-673.

Landrigan, P.J., Gehlbach, S.H., Rosenblum, B.F., Shoults, J.M., Candelaria R.M., Barthel, W.F., Liddle, J.A., Smrek, A.L., Staehling, N.W., Sanders, J.F, 1975 : Epidemic Lead Absorption Near an Ore Smelter : The Role of Particulate Lead. The New England Journal of Medicine 292 (3) : 123-129.

Lansdown, R., Yule, W., Urbanowicz, M.A., Millar, I.B., 1983 : Blood Lead, Intelligence, Attainment and Behaviour in School Children : Overview of a Pilot Study. In : Lead Versus Health : Sources and Effects of Low Level Lead Exposure. Eds: M. Rutter and R. Russell Jones. John Wiley and Sons, Chichester.

Lansdown, R., 1986 : Lead, Intelligence, Attainment and Behaviour. In : The Lead Debate : The Environment, Toxicology and Child Health. Eds. R. Lansdown and W. Yule. Croom Helm, London and Sydney.

Lau, W.M., Wong, H.M., 1982 : An Ecological Survey of Lead Contents in Roadside Dusts and Soils in Hong Kong. Environmental Research 28 : 39-54.

Lepow, M.L., Bruckman, L., Rubino, R.A., Markowitz, S., Gillette, M., Kapish, J., 1974 : Role of Airborne Lead in Increased Burden of Lead in Hartford Children. Environmental Health Perspectives 7 : 99-102.

Lepow, M.L., Bruckman, L., Gillette, M., Markowitz, S., Rubino, R., Kapish, J., 1975 : Investigations into Sources of Lead in the Environment of Urban Children. Environmental Research 10 : 415-426.

Lin Fu, J.S., 1975 : Undue Lead Absorption and Lead Poisoning in Children - An Overview. In : International Conference on Heavy Metals in the Environment. Symposium Proceedings Vol III, Toronto, Canada, October 27-31.

Maenhaut, W., Zoller, W.H., Duce, R.A., Hoffman, G.L., 1979 : Concentration and Size Distribution of Particulate Trace Elements in the South Polar Atmosphere. Journal of Geophysical Research 84 : 2421-2431.

Mahaffey, K.R., Annest, J.L., Roberts, J., Murphy, R.S., 1982 : National Estimates of Blood Lead Levels : United States 1976-1980. The New England Journal of Medicine 307 (10) : 573-579.

Manton, W.I., 1977 : Sources of Lead in Blood : Identification by Stable Isotopes. Archives of Environmental Health 32 : 149-159.

McBride, W.G., Black, B.P., English, B.J., 1982 : Blood Lead Levels and Behaviour of 400 Preschool Children. The Medical Journal of Australia 2 : 26-29.

McCord, C.P., 1953 : Lead and Lead Poisoning in Early America. Benjamin Franklin and Lead Poisoning. Industrial Medicine and Surgery 22 (9) : 393-399.

McCord, C.P., 1954 : Lead and Lead Poisoning in Early America - Clinical Lead Poisoning in the Colonies. Industrial Medicine and Surgery 23 : 120-125.

McDonald, J.M., Kaplan, E., 1942 : Incidence of Lead Poisoning in the City of Baltimore. The Journal of the American Medical Association 119 (11) : 870-872.

McKhann, C.F., 1926 : Lead Poisoning in Children. With Notes on Therapy. The American Journal of Diseases of Children 32 : 386-392.

McKhann, C.F., Vogt, E.C., 1933 : Lead Poisoning in Children. The Journal of the American Medical Association 101 (15) : 1131-1135.

McLaughlin, M.C., 1956 : Lead Poisoning in Children in New York City, 1950-1954. An Epidemiologic Study. New York State Journal of Medicine Dec 1 : 3711-3714.

Mellins, R.B., Jenkins, C.D., 1955 : Epidemiological and Psychological Study of Lead Poisoning in Children. The Journal of the American Medical Association 158 (1) : 15-20.

Milar, C.R., Schroeder, S.R., Mushak, P., Dolcourt, J.L., Grant, L.D., 1980 : Contributions of the Caregiving Environment to Increased Lead Burden of Children. American Journal of Mental Deficiency 84 (4) : 339-344.

Millichap, J.G., Llewellin, K.R., Roxburgh, R.C., 1952 : Lead Paint : A Hazard to Children. The Lancet 263 (6729) : 360-362.

Moncrieff, A.A., Koumides, O.P., Clayton, B.E., Patrick, A.D., Renwick, A.G.C., Roberts, G.E., 1964 : Lead Poisoning in Children. Archives of Disease in Childhood 39 (1) : 1-13.

Moore, M.R., Campbell, B.C., Goldberg, A., 1977 : Lead. (Chapter 3) In : The Environment and Man Vol 6. The Chemical Environment. Eds. J. Lenihan and W. Fletcher. Blackie, Glasgow.

Moore, M.R., 1986a : Lead in Humans. In : The Lead Debate : The Environment, Toxicology and Child Health. Eds : R Lansdown and W. Yule. Croom Helm, London and Sydney.

Moore, M.R., 1986b : Lead in Water. In : The Lead Debate : The Environment, Toxicology and Child Health. Eds: R. Lansdown and W. Yule. Croom Helm, London and Sydney.

Moore, M.R., 1986c : Lead in Paint. In : The Lead Debate : The Environment, Toxicology and Child Health. Eds: R. Lansdown and W. Yule. Croom Helm, London and Sydney.

Morris, R.L., 1980 : Spatial Patterns of Social Differentiation Among the Coloureds in Greater Cape Town. Unpublished M.A. Thesis. University of Cape Town, Cape Town, South Africa.

Murozumi, M., Chow, T.J., Patterson, C., 1969 : Chemical Concentrations of Pollutant Lead Aerosols, Terrestrial Dusts and Sea Salts in Greenland and Antarctic Snow Strata. Geochimica et Cosmochimica Acta 33 : 1247-1294.

Needleman, H.L., Gunnoe, C., Leviton, A., Reed, M., Peresie, H., Maher, C., Barrett, P., 1979 : Deficits in Psychologic and Classroom Performance of Children with Elevated Dentine Lead Levels. The New England Journal of Medicine 300 : 689-695.

Needleman, H.L., 1980 : Low Level Lead Exposure : The Clinical Implications of Current Research. Raven Press, New York.

Needleman, H.L., Landrigan, P.J., 1981 : The Health Effects of Low Level Exposure to Lead. Annual Review of Public Health 2 : 277-298.

Needleman, H.L., 1983 : The Neuropsychological Consequences of Low Level Exposure to Lead in Childhood. In : Lead Versus Health : Sources and Effects of Low Level Lead Exposure. Eds: M. Rutter and R. Russell Jones. John Wiley and Sons, Chichester, UK.

Newsweek, 1987 : The Spirit of Woodstock. Feb 16 : 16.

Nolan, B.M., 1982 : School Blood Lead Levels. The Medical Journal of Australia 1(2) : 57.

Nriagu, J.O., 1978a : Properties and the Biogeochemical Cycle of Lead. (Chapter 1). In : The Biogeochemistry of Lead in the Environment. Ed : J.O.Nriagu. North-Holland Biomedical Press. Elsevier.

Nriagu, J.O., 1978b : Lead in Soils, Sediments and Major Rock Types. (Chapter 2). In : The Biogeochemistry of Lead in the Environment. Ed : J.O. Nriagu. North-Holland Biomedical Press. Elsevier.

Nriagu, J.O., 1978c : Lead in the Atmosphere. (Chapter 6) In : The Biogeochemistry of Lead in the Environment. Ed : J.O. Nriagu. North-Holland Biomedical Press. Elsevier.

Nriagu, J.O., 1983 : Saturnine Gout Among Roman Aristocrats. The New England Journal of Medicine 308 (11) : 660-663.

O'Hara, D.M., 1982 : Social Factors in the Recurrence of Increased Lead Absorption in Children. In : Lead Absorption in Children. Eds. J.J. Chisholm and D.M. O'Hara. Urban and Schwarzenberg, Baltimore-Munich.

O'Toole, J., 1973 : Watts and Woodstock. Holt, Rinehart and Winston, New York.

Okubo, T., Tsuchiya, K., Nagasaki, M., Nakajima, T., Kamijyo, H., Mizoguchi, I., 1978 : A Further Study of Biological Effects of Lead on Urban and Suburban Tokyo School Children. International Archives of Occupational and Environmental Health 41 : 17-23.

Park, E.A., Jackson, D., Kajdi, L., 1931 : Shadows Produced by Lead in the X-Ray Pictures of the Growing Skeleton. American Journal of Diseases of Children 41 : 485-499.

Pickard, B.H., 1969 : Grand Parade : The Birth of Greater Cape Town. 1850-1913. Struik, Cape Town.

Piomelli, S., Corash, L., Corash, M.B., Seaman, C., Mushak, P., Glover, B., Padgett, R., 1980 : Blood Lead Concentrations in a Remote Himalayan Population. Science 210 : 1135-1137.

Poole, C., Smythe, L.E., Alpers, M., 1980 : Blood Lead Levels in Papua New Guinea Children Living in a Remote Area. The Science of the Total Environment 15 : 17-24.

Quinn, M.J., 1985 : Factors Affecting Blood Lead Concentrations in the U.K. : Results of the EEC Blood Lead Surveys, 1979-1981. International Journal of Epidemiology 14 (3) : 420-431.

Rabinowitz, M., Wetherill, G.W., Kopple, J.D., 1974 : Studies of Human Lead Metabolism by Use of Stable Isotope Tracers. Environmental Health Perspectives 7 : 145-153.

Rabinowitz, M.B., Kopple, J.D., Wetherill, G.W., 1980 : Effect of Food Intake and Fasting on Gastrointestinal Lead Absorption in Humans. American Journal of Clinical Nutrition 33 : 1784-1788.

Rapoport, M., and Kenney, A.S., 1939 : A Case of Lead Encephalopathy in a Breast-Fed Infant due to the Use of Lead Nipple Shields by the Mother. The Journal of the American Medical Association 112 (20) : 2040-2042.

Registrar General, 1960 : Classification of Occupations. HMSO. London.

Retief-Steyn, F., 1976 : Lood as Bedryfs en Openbare Gesondheidsgevaar in Suid Afrika. Unpublished M.D. Thesis. University of Pretoria, South Africa.

Ridd, R.E., 1981 : Position and Identity in a Divided Community. Colour and Religion in the District Six, Walmer Estate, Woodstock Area of Cape Town. Unpublished Ph.D. Thesis. Oxford University, United Kingdom.

Rodgers, T.S., Peck, J.R.S., Jupe, M.H., 1934 : Lead Poisoning in Children. The Lancet 2 : 129-133.

Roellin, H.B., Kilroe-Smith, T.A., Theodorou, P., 1988 : Quality Control for Analysing Lead in Blood : Evaluation and Comparison of Participating Laboratories. South African Journal of Science 84 : 233-234.

Roels, H.A., Buchet, J.P., Lauwerys, R., Hubermont, G., Bruaux, P., Claeys-Thoreau, F., Lafontaine, A., van Overschelde, J., 1976 : Impact of Air Pollution By Lead on the Heme Biosynthetic Pathway in School-Age Children. Archives of Environmental Health 31 : 310-316.

Roels, H.A., Buchet, J.P., Lauwerys, R.R., Bruaux, P., Claeys-Thoreau, F., LaFontaine, A., Verduyn, G., 1980 : Exposure to Lead by the Oral and the Pulmonary Routes of Children Living in the Vicinity of a Primary Lead Smelter. Environmental Research 22 : 81-94.

Rosner, D., Markowitz, G., 1985 : A 'Gift of God'? The Public Health Controversy Over Leaded Gasoline During the 1920's. American Journal of Public Health 75 (4) : 344-351.

Ruddock, J.C., 1924 : Lead Poisoning in Children. With Special Reference to Pica. The Journal of the American Medical Association 82 : 1682-84.

Russell, B.G., 1986 : South Africa's Position in the World's Supply of Minerals. Journal of the South African Institute of Mining and Metallurgy 86 (7) : 225-234.

Russell Jones, R.R., Stephens, R., 1983 : The Contribution of Lead in Petrol to Human Lead Intake. In : Lead Versus Health : Sources and Effects of Low Level Lead Exposure. Eds : M. Rutter and R. Russell Jones. John Wiley and Sons, New York.

Rutter, M., 1980 : Raised Lead Levels and Impaired Cognitive/Behavioural Functioning. A Review of the Evidence. Developmental Medicine and Child Neurology 22. Supplement 42.

Rutter, M., 1983 : Scientific Issues and the State of the Art in 1980. In : Lead Versus Health : Sources and Effects of Low Level Lead Exposure. Eds: M. Rutter and R. Russell Jones. John Wiley & Sons, Chichester, UK.

Sayre, J.W., Charney, E., Vostal, J., Pless, I.B., 1974 : House and Hand Dust as a Potential Source of Childhood Lead Exposure. American Journal of Diseases of Children 127 : 167-173.

Schlesselman, J.J., 1982 : Case Control Studies : Design, Conduct, Analysis. Oxford University Press, Oxford.

Schmitt, N., Philion, J.J., Larsen, A.A., Harnadek, M., Lynch, A.J., 1979 : Surface Soil as a Potential Source of Lead Exposure for Young Children. Canadian Medical Association Journal 121 : 1475-1478.

Scott, R.H., Stassen, G., 1983 : Implications of Lead in the South African Transport Scene. In : Lead in Petrol Seminar. Energy Research Unit, University of Cape Town, September 1983, Cape Town.

Shapiro, I.M., Mitchell, G., Davidson, I., Katz, S.H., 1975 : The Lead Content of Teeth : Evidence Establishing New Minimal Levels of Exposure in a Living Preindustrialized Human Population. Archives of Environmental Health 30 : 483-486.

Sherlock, J.C., Barltrop, D., Evans, W.H., Quinn, M.J., Smart, G.A., Strehlow, C., 1985 : Blood Lead Concentrations and Lead Intake in Children of Different Ethnic Origin. Human Toxicology 4 : 513-591.

Siegfried, W.R., Frost, P.G.H., Redelinghuys, E.P., van der Merwe, R.P., 1972 : Lead Concentrations in the Bones of City and Country Doves. South African Journal of Science 68 (9) : 229-230.

Sinn, W., 1980 : Relationship Between Lead Concentrations in the Air and Blood Lead Levels of People Living and Working in the Centre of a City. (Frankfurt Blood Lead Study) - I. International Archives Occupational and Environmental Health 47 : 93-118.

Sinn, W., 1981 : Relationship Between Lead Concentrations in the Air and Blood Lead Levels of People Living and Working in the Centre of a City. (Frankfurt Blood Lead Survey) - II. International Archives of Occupational and Environmental Health 48 : 1-23.

Skerfving, S., Schutz, A., Ranstam, J., 1986 : Decreasing Lead Exposure in Swedish Children, 1978-84. The Science of the Total Environment 58 : 225-229.

Smith, M., Delves, T., Lansdown, R., Clayton, B., Graham, P., 1983 : The Effects of Lead Exposure on Urban Children: The Institute of Child Health/ Southampton Study. Developmental Medicine and Child Neurology 25 Supplement 47.

Smith, M., 1986 : Lead in History. In : The Lead Debate : The Environment, Toxicology and Child Health. Eds. R. Lansdown and W.Yule. Croom Helm, London and Sydney.

Snodgrass, R.A., 1986 : Lead in South Africa. Journal of the South African Institute of Mining and Metallurgy 86 (4) : 97-111.

Solomon R.L., Hartford, J.W., 1976 : Lead and Cadmium in Dusts and Soils in a Small Urban Community. Environmental Science and Technology 10 (8) : 773-777.

Southwood, T.R.E., 1983 : Royal Commission on Environmental Pollution. Ninth Report. Lead in the Environment. HMSO, London.

Stark, A.D., Meigs, J.W., Fitch, R.A., DeLouise, E.R., 1978 : Family Operational Co-Factors in the Epidemiology of Childhood Lead Poisoning. Archives of Environmental Health 33 (5) : 222-225.

Stark, A.D., Quah, R.F., Meigs, J.W., DeLouise, E.R., 1982a : Relationship of Sociodemographic Factors to Blood Lead Concentrations in New Haven Children. Journal of Epidemiology and Community Health 36 : 133-139.

Stark, A.D., Quah, R.F., Meigs, J.W., Delouise, E.R., 1982b : The Relationship of Environmental Lead to Blood Lead Levels in Children. Environmental Research 27 : 372-383.

Stephenson, F.W.M., 1898 : Lead Poisoning in an Infant Four Weeks Old. The Lancet 2 : 1473.

Stewart, D.D., 1895 : Lead Convulsions : A Study of Sixteen Cases. American Journal of Medical Science 109 : 288-306.

Strong, R.A., 1920 : Meningitis, Caused by Lead Poisoning, in a Child of Nineteen Months. Archives of Pediatrics 37 : 532-537.

Suzuki, T., Kaneko, J., 1924 : Serous Meningitis in Infants from Lead Poisoning. The Journal of the American Medical Association 82 (22) : 1819-1820.

Tanis, A.L., 1955 : Lead Poisoning in Children. A.M.A. American Journal of Diseases of Children 89 : 325-331.

Taskinen, H., Nordman, H., Hernberg, S., Engstroem, K., 1981 : Blood Lead Levels in Finnish Preschool Children. The Science of the Total Environment 20 : 117-129.

Tepper, L.B., Levin, L.S., 1975 : A Survey of Air and Population Lead Levels in Selected American Communities. In : Lead. Eds: T.B. Griffin, J.H. Knelson. Academic Press, New York NY.

Ter Haar, G., Aronow, R., 1974 : New Information on Lead in Dirt and Dust as Related to the Childhood Lead Problem. Environmental Health Perspectives 7 : 83-90.

Tera, O., Schwartzman, D.W., Watkins, T.R., 1985 : Identification of Gasoline Lead in Children's Blood Using Isotopic Analysis. Archives of Environmental Health 40 (2) : 120-123.

Thomas, H.M., Blackfan, A.D., 1914 : Recurrent Meningitis Due to Lead, in a Child of Five Years. American Journal of Diseases of Children 8 : 377-380.

Thomas, H.V., Milmore, B.K., Heidbreder, G.A., Kogan, B.A., 1967 : Blood Lead of Persons Living Near Freeways. Archives of Environmental Health 15 : 695-702.

Time, 1987: South Africa : The "Graying" of a Nation. November 30 : 17.

Torrance, J.D., Mills, W., Green, A., Kilroe-Smith, T.A., Smith, A.N., 1985 : Erythrocyte Zinc Protoporphyrin Determination for Screening for Lead Exposure. South African Medical Journal 67 (21) : 848-850.

Travers, E., Rendle-Short, J., Harvey, C.C., 1956 : The Rotherham Lead-Poisoning Outbreak. The Lancet 2 : 113-116.

Turner, A.J., 1909 : Lead Poisoning in Childhood. British Medical Journal 1 : 895-897.

U.S. Public Health Service, 1971 : Medical Aspects of Childhood Lead Poisoning. Pediatrics 48 (3) : 464-468.

Urbanowicz, M.A., 1986 : The Uses of Lead Today. In : The Lead Debate : The Environment, Toxicology and Child Health. Eds. : R. Lansdown and W. Yule. Croom Helm, London and Sydney.

van Wyk, C.W., Grobler, S.R., 1983 : Lead Levels in Deciduous Teeth of Children From Selected Urban Areas in the Cape Peninsula. South African Medical Journal 63 (15) : 559

von Schirnding, Y.E., 1982 : A Study of the Impact of Lead in the Environment on School Children in the Cape Town Area. Unpublished M.Sc. Thesis. University of Cape Town, Rondebosch, South Africa.

von Schirnding, Y.E., Fuggle, R.F., 1984a : A Study of the Relationship Between Low Level Lead Exposure and Classroom Performance in South African Children. The International Journal of Biosocial Research 6 (2) : 97-106.

von Schirnding, Y.E., Fuggle, R.F., 1984b : Atmospheric, Soil and Dust Lead Levels in the Woodstock Area, Cape Town. In : Proceedings 6th International Conference on Air Pollution. 23-25 October, C.S.I.R. Pretoria, South Africa.

von Schirnding, Y.E., 1984 : Lead Toxicity and Poverty. Preliminary Case Study Investigations in Cape Town School Children. In : Proceedings Second Carnegie Inquiry into Poverty and Development in Southern Africa. 13-19 April, University of Cape Town, Rondebosch, South Africa.

von Schirnding, Y.E., Fuggle, R.F., 1986a : Zinc Protoporphyrin Levels and Lead Absorption in Children Attending Schools in the Cape Peninsula, South Africa. The Southern African Journal of Epidemiology and Infection 1 : 11-15.

von Schirnding, Y.E., Fuggle, R.F., 1986b : Exposure to Lead Amongst Urban School Children in Cape Town. Research Report 9/86/13. Environmental Evaluation Unit, University of Cape Town, Rondebosch, South Africa.

von Schirnding, Y.E., Fuggle, R.F., 1986c : Atmospheric, Soil and Dust Lead Levels in the Woodstock Area, Cape Town. Community Health in South Africa June-July : 8-10.

von Schirnding, Y.E., 1987a : Epidemiological Aspects of Exposure to Lead Amongst Urban School Children in Cape Town. Presented at the 6th Epidemiological Conference. 14-15 May, Cape Town, South Africa. (South African Journal of Epidemiology and Infection 1988 3, Supplement: 10-11).

von Schirnding, Y.E., 1987b : A Cross-Sectional Study of the Distribution and Determinants of Lead Levels in Cape Town School Children, South Africa. Presented at the 11th Scientific Meeting of the International Epidemiological Association. 8-13 August, Helsinki, Finland.

Vostal, J.J., Taves, E., Sayre, J.W., Charney, E., 1974 : Lead Analysis of House Dust : A Method for the Detection of Another Source of Lead Exposure in Inner City Children. Environmental Health Perspectives 7 : 91-97.

Waldron, H.A., 1973 : Lead Poisoning in the Ancient World. Medical History 17 : 391-399.

Waldron, H.A., Stoefen, D., 1974 : Subclinical Lead Poisoning. Academic Press, London and New York.

Waldron, H.A., 1975 : Lead Levels in Blood of Residents Near the M6-A38 (M) Interchange, Birmingham. Nature 253 : 3445-346.

Wells, R.B., Briggs, A.B., Vleggaar, C.M., 1983 : Measurement of Lead Levels in South Africa. In : Lead in Petrol Seminar. Energy Research Unit, University of Cape Town, September 1983, Cape Town.

White, N.W., Dempster, W.S., Pocock, F., Kibel, M.A., 1982 : Lead Absorption in Cape Children : A Preliminary Report. South African Medical Journal 62 (22) : 799-802.

Whittingdale, J., 1973 : The Development and Location of Industries in Greater Cape Town. Unpublished M.A. Thesis. University of Cape Town, Cape Town, South Africa.

Wilcox, H.B., Caffey, J.P., 1926 : Lead Poisoning in Nursing Infants. The Journal of the American Medical Association 86 (20) : 1514-1516. .

Williams, H., Schulze, W.H., Rothchild, H.B., Brown, A.S., Smith, F.R., 1933 : Lead Poisoning from the Burning of Battery Casings. The Journal of the American Medical Association 100 (19) : 1485-1489.

Wilson, D., Esterman, A., Lewis, M., Roder, D., Calder, I., 1986 : Children's Blood Lead Levels in the Lead Smelting Town of Port Pirie, South Australia. Archives of Environmental Health 41 (4) : 245-251.

Winneke, G., 1983 : Neurobehavioural and Neuropsychological Effects of Lead. In : Lead Versus Health : Sources and Effects of Low Level Lead Exposure. Eds: M. Rutter and R. Russell Jones. John Wiley and Sons, Chichester, UK.

World Health Organisation, 1977 : Environmental Health Criteria 3. Lead. WHO, Geneva

World Health Organisation, 1985 : Measuring Change in Nutritional Status. Guidelines for Assessing the Nutritional Impact of Supplementary Feeding Programmes for Vulnerable Groups. WHO, Geneva.

Wyllie, J., 1954 : A Family Outbreak of Lead Poisoning from the Burning of Storage Battery Casings. The Canadian Medical Association Journal 70 : 287-290.

Yaffe, Y., Flessel, C.P., Weselowski, J.J., Del Rosario, A., Guirguis, G.N., Matias, V., Gramlich, J.W., Kelly, W.R., Degarmo, T.E., Coleman, G.C., 1983 : Identification of Lead Sources in California Children Using the Stable Isotope Ratio Technique. Archives of Environmental Health 38 (4) : 237-245.

Yankel, A.J., von Lindern, I.H., Walter, S.D., 1977 : The Silver Valley Lead Study : The Relationship Between Childhood Blood Lead Levels and Environmental Exposure. Journal of the Air Pollution Control Association 27 (8) : 763-767.

Yule, W., Millar, I.B., Urbanowicz, M.A., 1981 : The Relationship Between Blood Lead Concentrations, Intelligence and Attainment in a School Population : A Pilot Study. Developmental Medicine and Child Neurology 23 : 567-576.

Yule, W., Lansdown, R., Hunter, J., Urbanowicz, M.A., Clayton B., Delves, T., 1983 : Blood Lead Concentrations in School Age Children: Intelligence, Attainment and Behaviour. Paper given at the Annual Conference of the British Psychological Society. York 10th April 1983.

Yule, W., 1986 : Methodological and Statistical Issues. In: The Lead Debate : The Environment, Toxicology and Child Health. Eds: R. Lansdown and W. Yule. Croom Helm, London and Sydney.

Ziegler, E.E., Edwards, B.B., Jensen, R.L., Mahaffey, K.R., Fomon, S.J., 1978 : Absorption and Retention of Lead by Infants. Pediatric Research 12 : 29-34.

APPENDIX A

CONSENT FORM

Dear Parent

You may have read in the newspapers recently about the problem of lead in our environment. Small amounts of lead are found in food, water, the air we breathe and in people. Children from schools all over Cape Town have been tested, and some have been found to have too much lead in their blood. This can make some children sick.

In the next few days, children at schools in this area will be tested at school to see how much lead they have in their blood. If you give permission, your child will have a small amount of blood taken from a vein in the arm, by a qualified sister. This blood will be examined for lead. Later on, a short questionnaire will be sent home with your child and you will be asked to answer some simple questions about your child. Someone may also visit you at your home to get more information that we may need. This will help us, and you, to know if there is a problem.

It would be much appreciated if you could sign the form below if you will allow your child to be tested. If you wish, you will be given the result of the test. If by any chance the level of lead in your child is thought to be high, you will be told about medical treatment that may be necessary.

Thank you for your co-operation.

Professor R. Fuggle

School of Environmental Studies, University of Cape Town

I give permission for my child (child's name:

.....) to have the blood lead test.

Signature : .....

I do not give permission for my child (child's name:

.....) to have the blood lead test.

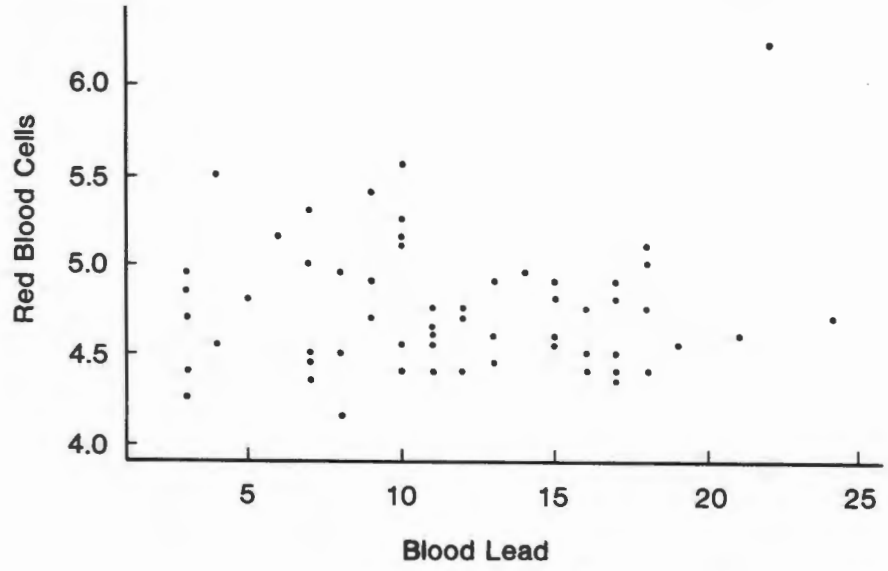
Signature : .....

Home address : .....

.....

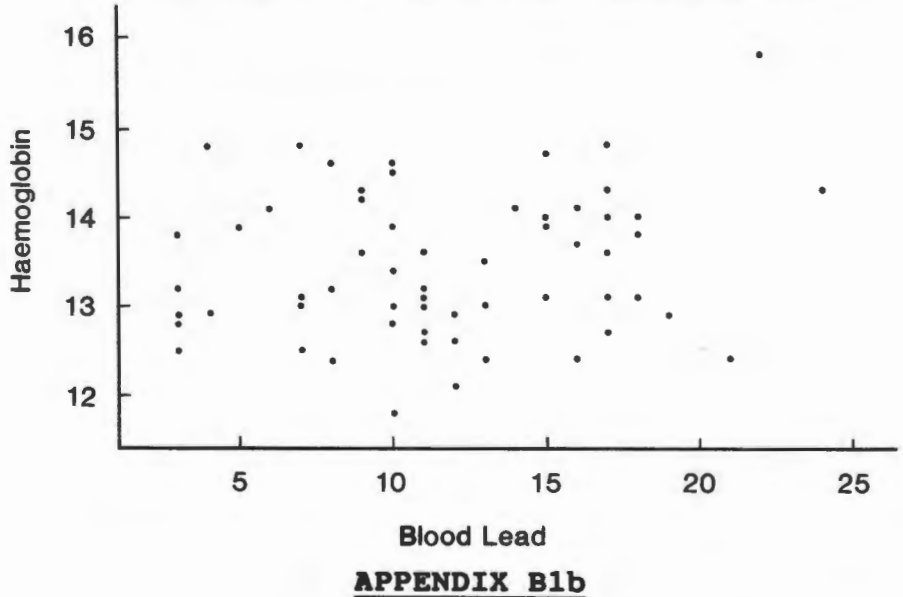
.....

.....



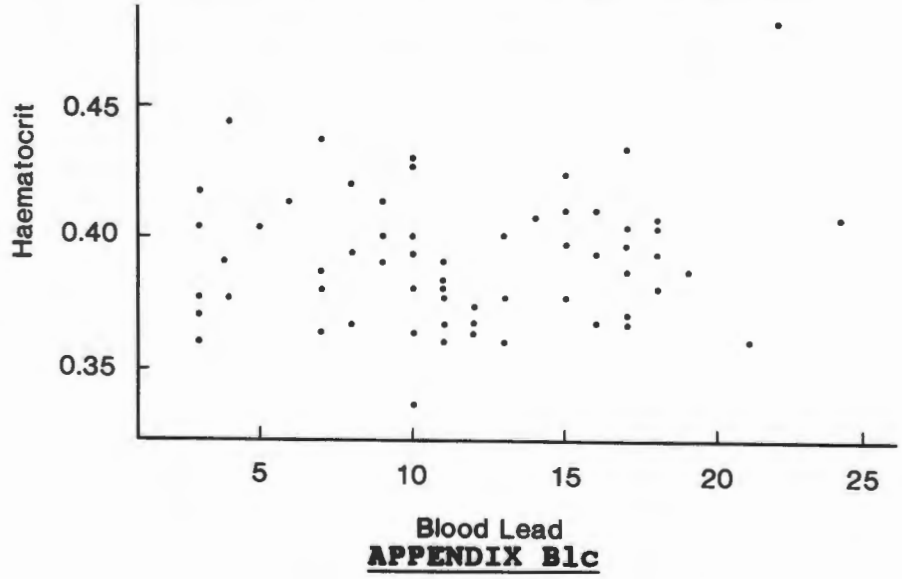
APPENDIX B1a

THE RELATIONSHIP BETWEEN RED BLOOD CELL LEVELS ($x10^{12}$/l) AND BLOOD LEAD LEVELS (ug/dl) (WHITE PUPILS)



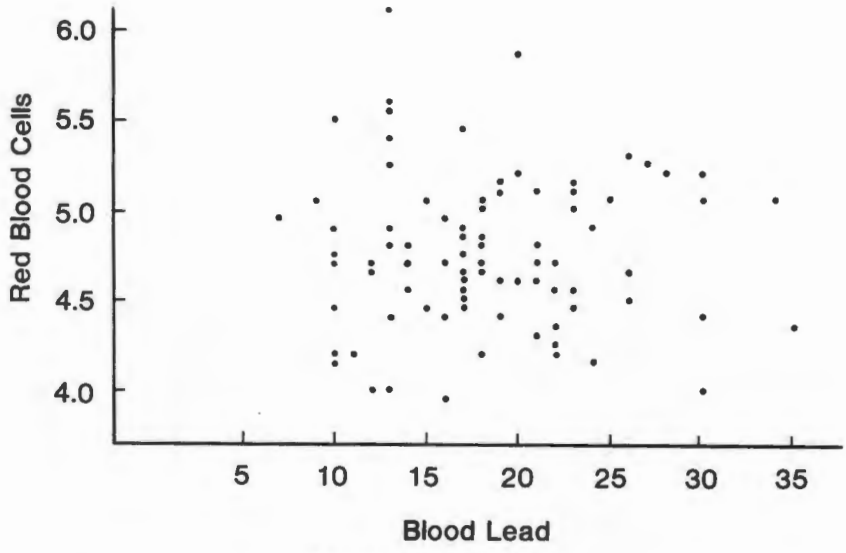
APPENDIX B1b

THE RELATIONSHIP BETWEEN HAEMOGLOBIN LEVELS (g/dl) AND BLOOD LEAD LEVELS (ug/dl) (WHITE PUPILS)



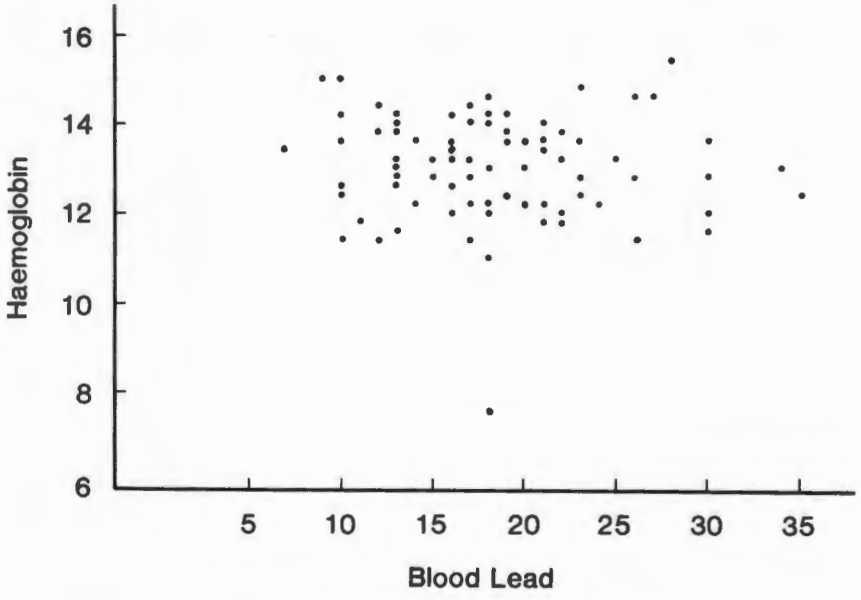
APPENDIX B1c

THE RELATIONSHIP BETWEEN HAEMATOCRIT LEVELS (l/l) AND BLOOD



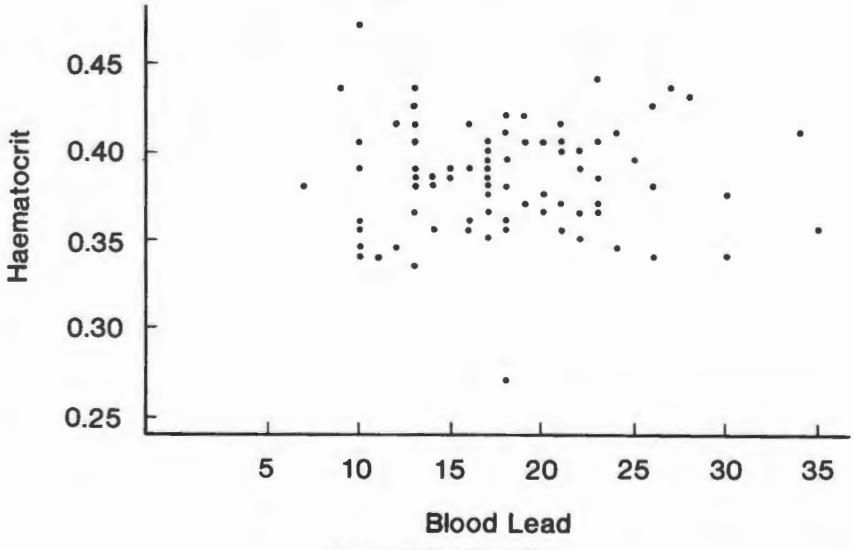
APPENDIX B2a

THE RELATIONSHIP BETWEEN RED BLOOD CELL LEVELS ($x10^{12}/l$) AND BLOOD LEAD LEVELS (ug/dl) (COLOURED PUPILS)



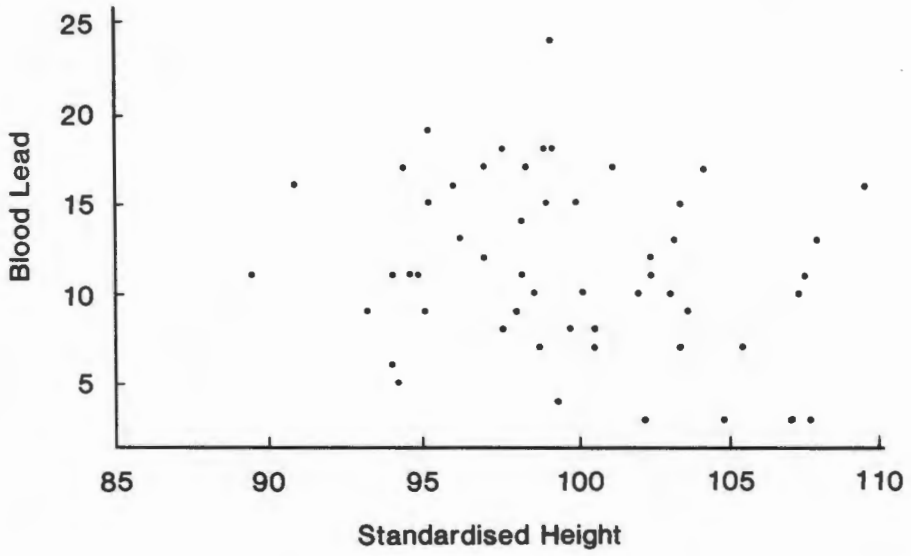
APPENDIX B2b

THE RELATIONSHIP BETWEEN HAEMOGLOBIN LEVELS (g/dl) AND BLOOD LEAD LEVELS (ug/dl) (COLOURED PUPILS)



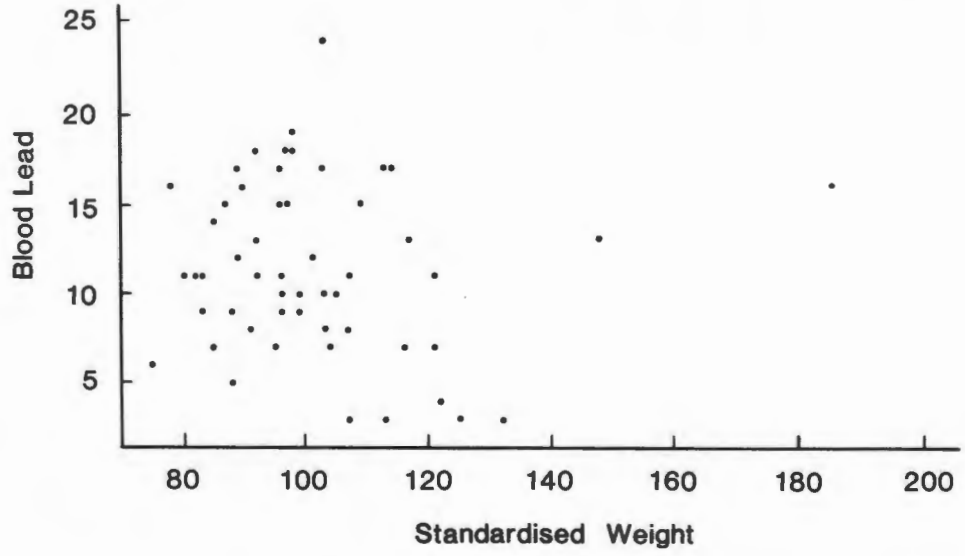
APPENDIX B2c

THE RELATIONSHIP BETWEEN HAEMATOCRIT LEVELS (l/l) AND BLOOD



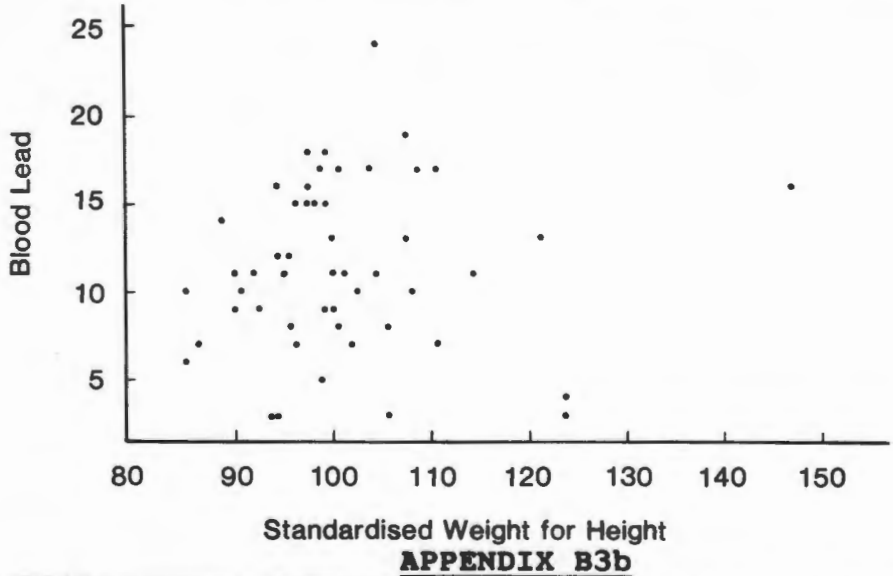
APPENDIX B3c

THE RELATIONSHIP BETWEEN STANDARDISED WEIGHT FOR HEIGHT AND BLOOD LEAD LEVELS (ug/dl) (WHITE PUPILS)



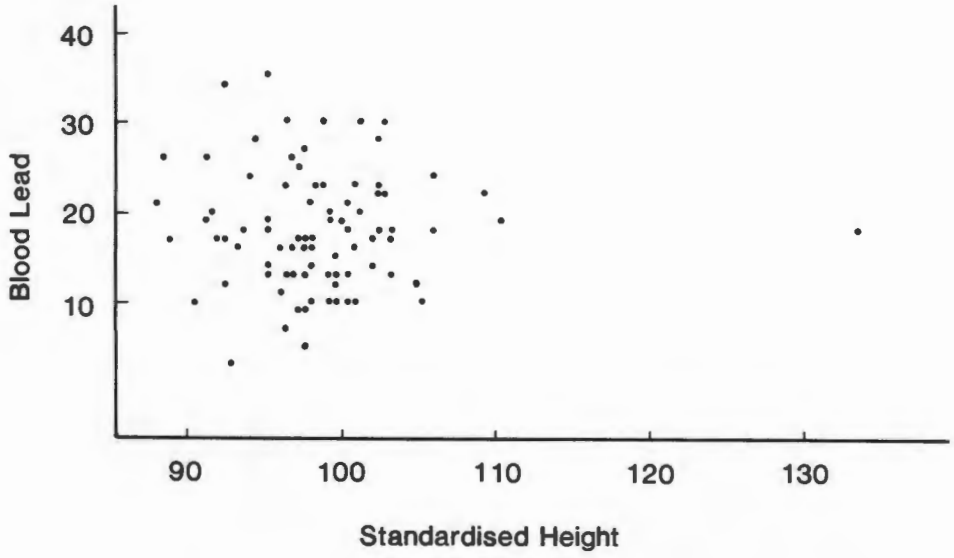
APPENDIX B3a

THE RELATIONSHIP BETWEEN STANDARDISED HEIGHT AND BLOOD LEAD LEVELS (ug/dl) (WHITE PUPILS)



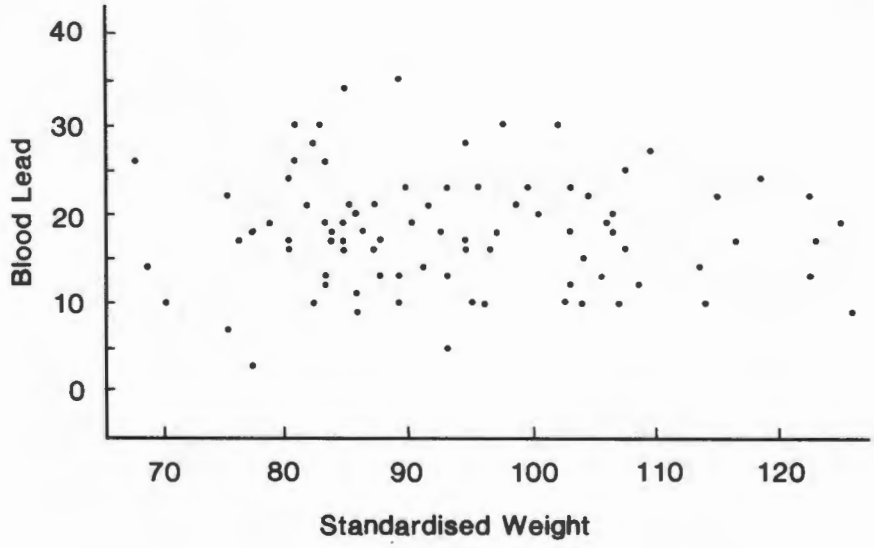
APPENDIX B3b

THE RELATIONSHIP BETWEEN STANDARDISED WEIGHT AND BLOOD LEAD



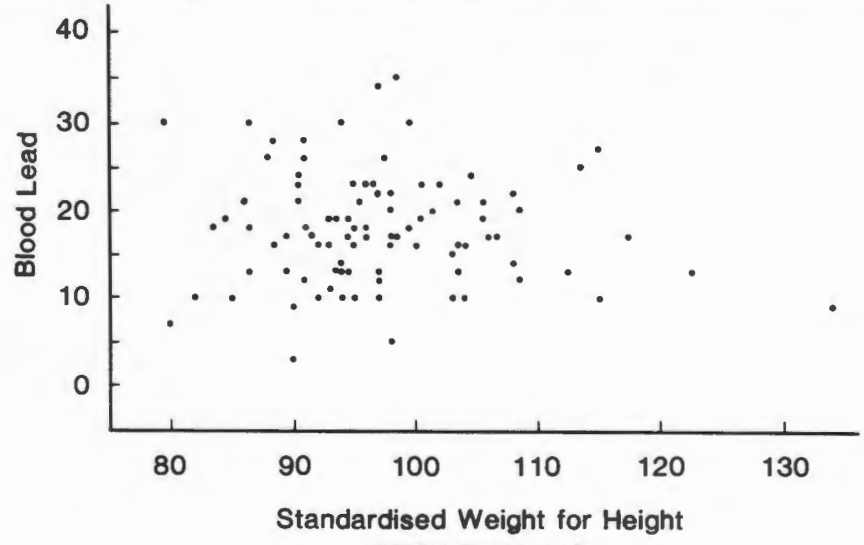
APPENDIX B4a

THE RELATIONSHIP BETWEEN STANDARDISED HEIGHT AND BLOOD LEAD LEVELS (ug/dl) (COLOURED PUPILS)



APPENDIX B4b

THE RELATIONSHIP BETWEEN STANDARDISED WEIGHT AND BLOOD LEAD LEVELS (ug/dl) (COLOURED PUPILS)



APPENDIX B4c

THE RELATIONSHIP BETWEEN STANDARDISED WEIGHT FOR HEIGHT AND

APPENDIX C

QUESTIONNAIRE

Dear Parent

As you know, your child was recently tested at school for lead. Unfortunately we have not yet received the results of the blood test for your child, but hope to have these by the end of the year. Once the results are available, it will be necessary for us to determine whether or not your child needs any preventative treatment.

In order for us to do this, we need certain information about your child. We need to know about your child's general health, everyday activities and behaviour, diet, home and family. We would appreciate your help with this, and ask you to fill in the answers to the questions on these pages. Most of the questions are very easy and you only have to answer "yes" or "no" - at the most it will take you about half an hour to complete.

WHAT TO DO :

Who should fill it in : The child's mother (or guardian) should answer the questions, but if this is not possible, then the person with the best knowledge about the child should fill it in.

How to answer the questions : There are two ways of answering a question : for example, you may be asked a question, and you will see a dotted line beneath the question. You simply write the answer on the dotted line.

Example

Question : When was your child born?

Answer : .....*1/7/1976*.....

(Continued)

For most of the questions, however, you will simply be asked to put a tick ( ) in the box next to the correct answer. Unless you are told that you may tick more than one box, tick ONLY ONE box, even if you have a little difficulty in deciding which one to tick.

Example

Question : How does your child usually get to school?

Answer : Car —

Bicycle —

Walks —

Bus or Train —

A tick is placed in the box next to "WALKS", if the child usually walks to school.

Example

Question : Is your child well at present?

Answer: Yes —

No —

Don't know —

A tick is placed in the box next to "DON'T KNOW" if you are not sure whether your child is well or not.

If you are asked a question which does not apply to your child, for instance "When did your child first start chewing painted things?", and your child never did this, just leave that question blank and go on to the next one.

(Continued)

Please answer the questions as best as you can, as this will determine how we can best help your child, if we find there is a problem. Some questions may sound a little funny to you, but they are important for me to know! Absolutely NO-ONE except the University researchers will see the questionnaire you have filled in. The results will be kept entirely confidential.

Please fill in the answers as soon as possible, and return the form with your child, in the sealed envelope, and we will collect it the following morning at school. If you cannot return it the next day, please return it as soon as you possibly can.

Thank you very much for your assistance. We hope this will help you and your child.

Yours faithfully

Professor R F Fuggle
Director
School of Environmental
Studies

(Continued)

SECTION A

1. What is your name? (the name of the person filling in the questionnaire).

   .....

2. What is your relation to the child (for example, child's mother, child's aunt, etc.)?

   .....

3. What is your address?

   .....

4. What is your phone number? .....

5. What is your child's first name?

   .....

6. What is your child's surname?

   .....

7. When was your child born? (Please give day, month and year).

   .....

8. What language does your child speak at home? (Please tick the right box : for example, if your child speaks English at home, place a tick in the box next to "English", like this :

   English __

   (If your child speaks two languages at home, you may tick two boxes).

   English __

   Afrikaans __

   Portuguese __

   Other __

(Continued)

9. What school does your child go to?

.....

10. How does your child normally get to school? (Please place a tick in one box only).

Car ___

Bicycle ___

Walks ___

Bus or Train ___

Don't know ___

11. Is this the first time your child is doing Grade One?

Yes ___

No ___

12. If your child failed before, is this the first time it is repeating Grade One? (Leave this question blank, if your child has never failed).

Yes ___

No ___

(Continued)

13. Is your child's work at school

Excellent ___

Good ___

Average ___

Poor ___

Don't know ___

14. Does your child walk alongside any busy road with lots of cars, on the way to school?

Yes ___

No ___

Don't know ___

(Continued)

15. After school, or on weekends, where does your child play most? (Tick only one box).

Inside the House ___

Garage ___

Carport ___

Backyard ___

Frontyard ___

Lane ___

Street ___

Other ___

Don't know ___

16. Does your child play often (for example, a few times during the week) on the sidewalk of busy streets with lots of cars?

Yes ___

No ___

Don't know ___

17. Does your child spend most afternoons during the week at someone else's home?

Yes ___

No ___

Don't know ___

(Continued)

18. Does your child spend most weekends at someone else's home?

Yes __

No __

Don't know __

19. How long has your child lived at his or her present home address?

Less than 6 months __

6 months - 1 year __

Between 1 - 2 years __

More than 2 years __

Don't know __

20. Did your child attend a creche or playschool before he/she went to school?

Yes __

No __

Don't know __

21. Was your child very active before she/he went to school?

Yes __

No __

Don't know __

(Continued)

22. Would you say he/she was more active than other children?

Yes __

No __

Don't Know __

23. Would you say he/she was over-active?

Yes __

No __

Don't Know __

24. Is your child particularly active now? (these days)

Yes __

No __

Don't Know __

25. Does your child have difficulty in concentrating for a period of time on a particular job?

Yes __

No __

Don't Know __

(Continued)

26. Is your child very easily distracted?

Yes __

No __

Don't Know __

27. Would you say he/she is more easily distracted than other children?

Yes __

No __

Don't Know __

28. Have you ever noticed your child eating or chewing on any of the following :

Paint Flakes Yes __

No __

Cement Yes __

No __

Plaster Yes __

No __

Soil, Earth Yes __

No __

(Continued)

Sticks	Yes	__
	No	__
Matchsticks	Yes	__
	No	__
Cigarette Ends	Yes	__
	No	__
Anything else that is not normal?	Yes	__
	No	__

28. Continued. If you answered yes, what other things does he or she eat or chew on?

.....

.....

.....

29. How old was your child when you first noticed him or her eating the things you ticked in the boxes, or described in the question above?

.....

(Continued)

30. Does he/she still eat these things?

Yes __

No __

Don't Know __

31. If your child doesn't eat these things anymore, how old was he/she when he/she stopped?

.....

32. How often did, or does, your child eat these things?

Everyday __

Every 2 or 3 days __

About once a Week __

Occasionally __

Don't know __

33. Is your child well at present?

Yes __

No __

Don't Know __

(Continued)

34. If your child is not well, what are the problems?

.....

.....

.....

.....

35. Is your child receiving medical treatment at the moment?

Yes ___

No ___

36. Has your child at any time suffered with the following:

Fits: Yes ___

No ___

Don't Know ___

Anaemia: Yes ___

No ___

Don't Know ___

Poor Speech: Yes ___

No ___

Don't Know ___

(Continued)

Kidney Problems: Yes __

No __

Don't Know __

Poor Hearing: Yes __

No __

Don't Know __

Poor Eyesight: Yes __

No __

Don't Know __

General Poor Health: Yes __

No __

Don't Know __

37. In the last two years has your child often suffered from :

Clumsiness: Yes __

No __

Don't Know __

(Continued)

Tiredness:	Yes	__
	No	__
	Don't Know	__
Loss of Appetite:	Yes	__
	No	__
	Don't Know	__
Poor Eating Habits:	Yes	__
	No	__
	Don't Know	__
Loss of Weight:	Yes	__
	No	__
	Don't Know	__
Vomiting:	Yes	__
	No	__
	Don't Know	__
Diarrhoea:	Yes	__
	No	__
	Don't Know	__

(Continued)

Constipation: Yes __

No __

Don't Know __

Pale complexion: Yes __

No __

Don't Know __

SECTION B : DIET

1. How often does your child eat tinned food?

Every day __

Every 2 - 3 days __

Once a Week __

Occasionally __

Never __

Don't Know __

2. About how many times a week does your child eat or drink :

Fresh Meat .....

Tinned Meat .....

Fresh Vegetables .....

(Continued)

Tinned Vegetables .....

Fresh Fish .....

Tinned Fish .....

Fresh Fruit .....

Tinned Fruit .....

Mealie Meal .....

Bread .....

Cereals .....

Cheese .....

Eggs .....

Milk .....

Cool drinks .....

Cakes/pudding .....

Sweets .....

3. What is your child's religion? .....

4. Describe any particular dietary habits your child may follow due to his/her religion or nationality

.....

(Continued)

5. Do you grow your own vegetables?

Yes __

No __

6. What type of pots do you normally use for cooking? (You may tick more than one box if necessary).

Steel __

Aluminium __

Iron __

Enamel __

Other __

Don't Know __

7. Do you use any pottery dishes for storing or cooking food in?

Yes __

No __

Don't Know __

(Continued)

SECTION C

HOUSING

1. Is your home

Owned ___

Rented ___

2. If you live in a flat, what floor do you live on?

Ground Floor ___

First Floor ___

Second Floor ___

Third Floor or higher ___

3. If you live in a house, is it

One Storey ___

Two Storeys ___

4. How many years ago was your home built? .....

5. How many rooms, not counting the kitchen and bathroom, does your home have? .....

6. How many rooms are used for sleeping in? .....

7. Do you regularly use any of the following in your home?

Electricity Yes ___

No ___

(Continued)

Paraffin	Yes	___
	No	___
Gas	Yes	___
	No	___
Wood	Yes	___
	No	___
Coal	Yes	___
	No	___
Candles	Yes	___
	No	___

Anything else .....

8. During the day, does a lot of sunlight come into your home?

Yes ___

No ___

Don't Know ___

(Continued)

9. Does a lot of fresh air come into your home?

Yes ___

No ___

Don't Know ___

10. Do you smoke?

Yes ___

No ___

11. How many people in the home smoke? .....

12. Is your home often

Very Dusty ___

Slightly Dusty ___

Not Dusty ___

13. Do you have an outside toilet?

Yes ___

No ___

14. What type of water piping (plumbing) does your home have?

Copper ___

Galvanised Iron ___

(Continued)

Lead ___

Other ___

Don't Know ___

15. Have your water pipes ever been replaced?

Yes ___

No ___

Don't Know ___

16. If yes, approximately how many years ago were they replaced?

.....

17. If they were replaced, what were they before?

Copper ___

Galvanised Iron ___

Lead ___

Other ___

Don't Know ___

(Continued)

18. Is there a large amount of paint peeling from the outside walls, outside doors or outside windowsills of your home?

Yes ___

No ___

Don't Know ___

19. If yes, please describe where paint is peeling from outside (for example, outside door, outside veranda, etc.).

.....

.....

.....

20. Is there a large amount of paint peeling from inside doors, walls or windowsills?

Yes ___

No ___

Don't Know ___

21. If yes, please describe where paint is peeling from inside (for example, kitchen door, bedroom windowsill, etc.).

.....

.....

.....

.....

(Continued)

22. Does your home need major repairs?

Yes __

No __

Don't Know __

23. List your major complaints with your home (for example, holes in roof, dirty water, peeling paint, overcrowding, etc.).

.....

.....

.....

.....

24. Are there any factories in the street where you live, or nearby? (5 minutes walking distance away).

Yes __

No __

Don't Know __

25. If yes, please list the factories in your street, or nearby to you (for example, coal power station, clothing factory, etc.).

.....

.....

.....

(Continued)

26. Do you notice any fumes or smoke coming out of any of the factories?

Yes __

No __

Don't Know __

27. If yes, which factory or factories?

.....

.....

28. Are there any petrol garages in your street or nearby? (two minutes walking distance away).

Yes __

No __

Don't Know __

29. Are there any workshops in your street or nearby (2 minutes walking distance away) (for example, panel beating/spray-painting, welding, etc.)?

Yes __

No __

Don't Know __

30. If yes, please list the workshops nearby to you.

....

.....

(Continued)

.....

.....

SECTION D

SOCIAL

1. How many people live in your home?

.....

2. How many children under the age of 12 live in your home?

.....

3. How many sisters and brothers does your child have?

.....

4. What are their ages? .....

5. How many people in your home work? .....

6. Does the child live with his/her (you may tick more than one box)

Mother __

Father __

Female Guardian __

Male Guardian __

(Continued)

7. What is the child's father's (or male guardian whom he/she lives with) job? (Please name the type of job - for example, cleaner, and the place, e.g. shipyard. If the child does not live with his/her father, or male guardian, leave this blank).

   Job .....

   Place .....

8. Which standard (or grade) did the father or male guardian leave school? .....

9. What is his monthly income? .....

10. What is the child's mother's (or female guardian whom he/she lives with) job? (Please name the type of job, for example, machinist and the place, for example, clothing factory).

    Job .....

    Place .....

11. Which standard (or grade) did the mother or female guardian leave school?

    .....

12. What is her monthly income?

    .....

13. What is your home's total monthly family income (the total income of everybody who works, in your home)?

    .....

14. How many people are supported on this income?

    .....

15. How many children under the age of 12 years are supported on this income?

    .....

(Continued)

16. If your home is rented, what is your monthly rent?

.....

17. Please describe the hobbies of people living in the house (for instance there may be people who fix cars, do welding, make pottery, etc.). Please list these types of activities.

.....

.....

.....

.....

.....

SECTION E

1. Did you have difficulty in answering this questionnaire?

Yes ___

No ___

2. Which questions did you find difficult to answer?

.....

.....

.....

(Continued)

3. Was it too long?

Yes ___

No ___

4. How long did it take you to complete the questionnaire?

.....

5. Comments: Please list any complaints or other comments about the questionnaire.

.....

.....

.....

.....

.....

.....

Thank you for answering these questions. Your assistance is much appreciated.

(Continued)

APPENDIX D1

CODE SHEET FOR QUESTIONNAIRE FREQUENCY ANALYSES

Unless otherwise indicated in the tables below, the following codes apply to all questions throughout :

. Missing
1. Yes
2. No
3. Don't know

HOME LANGUAGE (Q A8)

. Missing
1 English
2 Afrikaans
3 Portuguese
4 Other
5 English and Afrikaans
6 English and Portuguese

TRANSPORT TO SCHOOL (Q A10)

. Missing
1 Car
3 Walk
4 Bus or train

GRADE ONE REPEAT (Q A11)

. Missing
1 No
2 Yes

REPEAT-RATE (Q A12)

. Missing
1 Repeating grade one first time
2 Repeating grade one second time or more

SCHOOL WORK (Q A13)

. Missing
1 Excellent
2 Good
3 Average
4 Poor
5 Don't know

PLAY-SITE (Q A15)

. Missing
1 Inside house
2 Garage
3 Carport
4 Back yard
5 Front yard
6 Lane
7 Street
8 Other

RESIDENCE PRESENT ADDRESS (Q A19)

. Missing
1 Less than 6 months
2 6 months - 1 year
3 Between 1 - 2 years
4 More than 2 years
5 Don't know

FREQUENCY PICA (Q A32)

. Missing
1 Every day
2 Every 2 - 3 days
3 Once a week
4 Occasionally
5 Don't know

TINNED FOOD (Q B1)

. Missing
1 Every day
2 Every 2 - 3 days
3 Once a week
4 Occasionally
5 Never
6 Don't know

RELIGION (Q B3)

. Missing
2 "Malay" (Muslim)
3 Christian
5 Islam
6 Hindu

FLAT (Q C2)

. Missing
1 Ground floor
2 First floor
3 Second floor
4 Third floor or above

HOUSE (Q C3)

. Missing
1 One storey
2 Two storeys

HOUSE DUST (Q C12)

Missing
1 Home very dusty
2 Home slightly dusty
3 Home not dusty

WATER PIPES (Q C14)

. Missing
1 Copper
2 Galvanised iron
3 Lead
4 Other
5 Don't know

WATER PIPES PRIOR TO REPLACEMENT (Q C17)

. Missing
1 Copper
2 Galvanised iron
3 Lead
4 Other
5 Don't know

MOTHER (Q D6)

. Missing
1 Child lives with mother
2 Child lives without mother

FATHER (Q D6)

. Missing
1 Child lives with father
2 Child lives without father

APPENDIX D2

QUESTIONNAIRE SURVEY: FREQUENCY ANALYSIS (WHITE PUPILS)

(see A.D1 for codes)

HOME LANGUAGE (Q A8)

	Freq	Percent
1	27	69.231
3	1	2.564
5	5	12.821
6	6	15.385

TRANSPORT TO SCHOOL (Q A10)

	Freq	Percent
.	1	.
1	8	21.053
3	29	76.316
4	1	2.632

GRADE ONE REPEAT (Q A11)

	Freq	Percent
.	1	.
1	35	92.105
2	3	7.895

REPEAT-RATE (Q A12)

	Freq	Percent
.	34	.
1	3	60.000
2	2	40.000

SCHOOL WORK (Q A13)

	Freq	Percent
2	19	48.718
3	18	46.154
4	1	2.564
5	1	2.564

WALK SCHOOL BUSY STREETS (Q A14)

	Freq	Percent
.	1	.
1	19	50.000
2	19	50.000

PLAY-SITE (Q A15)

	Freq	Percent
.	1	.
1	11	28.947
2	1	2.632
3	1	2.632
4	11	28.947
5	10	26.316
7	2	5.263
8	2	5.263

PLAY BUSY STREETS (Q A16)

	Freq	Percent
1	4	10.256
2	35	89.744

WEEKDAYS OTHER HOMES (Q A18)

	Freq	Percent
1	8	20.513
2	31	79.487

WEEKENDS OTHER HOMES (Q A18)

	Freq	Percent
1	3	7.692
2	36	92.308

RESIDENCE PRESENT ADDRESS (Q A19)

	Freq	Percent
.	2	.
1	3	8.108
2	8	21.622
3	6	16.216
4	20	54.054

CRECHE ATTENDANCE (Q A20)

	Freq	Percent
1	23	58.974
2	16	41.026

VERY ACTIVE BEFORE SCHOOL (Q A21)

	Freq	Percent
1	34	87.179
2	3	7.692
3	2	5.128

MORE ACTIVE THAN OTHERS (Q A22)

	Freq	Percent
.	1	.
1	10	26.316
2	22	57.895
3	6	15.789

OVERACTIVE (Q A23)

	Freq	Percent
.	1	.
1	7	18.421
2	29	76.316
3	2	5.263

ACTIVE AT PRESENT (Q A24)

	Freq	Percent
.	4	.
1	30	85.714
2	3	8.571
3	2	5.714

DIFFICULTY CONCENTRATING (Q A25)

	Freq	Percent
.	2	.
1	11	29.730
2	21	56.757
3	5	13.514

EASILY DISTRACTED (Q A26)

	Freq	Percent
.	2	.
1	21	56.757
2	10	27.027
3	6	16.216

MORE EASILY DISTRACTED THAN OTHERS (Q A27)

	Freq	Percent
.	2	.
1	2	5.405
2	26	70.270
3	9	24.324

PAINT (Q A28)

	Freq	Percent
.	15	.
2	24	100.000

CEMENT (Q A28)

	Freq	Percent
.	15	.
2	24	100.000

PLASTER (Q A28)

	Freq	Percent
.	15	.
1	1	4.167
2	23	95.833

SOIL (Q A28)

	Freq	Percent
.	13	.
1	6	23.077
2	20	76.923

STICKS (Q A28)

	Freq	Percent
.	16	.
1	3	13.043
2	20	86.957

MATCHSTICKS (Q A28)

	Freq	Percent
.	16	.
1	4	17.391
2	19	82.609

CIGARETTE-ENDS (Q A28)

	Freq	Percent
.	15	.
1	1	4.167
2	23	95.833

OTHER ABNORMAL EATING HABITS (Q A28)

	Freq	Percent
.	9	.
1	3	10.000
2	27	90.000

AGE ONSET PICA (YEARS) (Q A29)

	Freq	Percent
.	27	.
1	6	50.000
2	1	8.333
3	1	8.333
4	1	8.333
6	3	25.000

PICA STILL PRESENT (Q A30)

	Freq	Percent
.	25	.
1	5	35.714
2	9	64.286

AGE STOP PICA (YEARS) (Q A31)

	Freq	Percent
.	31	.
2	2	25.000
3	1	12.500
4	2	25.000
5	1	12.500
6	2	25.000

FREQUENCY PICA (Q A32)

	Freq	Percent
.	29	.
3	1	10.000
4	8	80.000
5	1	10.000

CHILD WELL PRESENT (Q A33)

	Freq	Percent
.	1	.
1	36	94.737
2	1	2.632
5	1	2.632

MEDICAL TREATMENT (Q A35)

	Freq	Percent
.	5	.
1	2	5.882
2	32	94.118

FITS (Q A36)

	Freq	Percent
.	9	.
1	2	6.667
2	28	93.333

ANAEMIA (Q A36)

	Freq	Percent
.	8	.
1	3	9.677
2	28	90.323

POOR SPEECH (Q A36)

	Freq	Percent
.	9	.
1	4	13.333
2	26	86.667

KIDNEY PROBLEMS (Q A36)

	Freq	Percent
.	8	.
1	1	3.226
2	30	96.774

POOR HEARING (Q A36)

	Freq	Percent
.	8	.
1	5	16.129
2	26	83.871

POOR EYESIGHT (Q A36)

	Freq	Percent
.	9	.
1	4	13.333
2	26	86.667

GENERAL POOR HEALTH (Q A36)

	Freq	Percent
.	7	.
1	5	15.625
2	26	81.250
3	1	3.125

CLUMSINESS (Q A37)

	Freq	Percent
.	7	.
1	4	12.500
2	28	87.500

TIREDNESS (Q A37)

	Freq	Percent
.	6	.
1	7	21.212
2	25	75.758
3	1	3.030

LOSS OF APPETITE (Q A37)

	Freq	Percent
.	6	.
1	11	33.333
2	22	66.667

POOR EATING HABITS (Q A37)

	Freq	Percent
.	1	.
1	19	50.000
2	18	47.368
3	1	2.632

WEIGHT LOSS (Q A37)

	Freq	Percent
.	5	.
1	8	23.529
2	24	70.588
3	2	5.882

VOMITING (Q A37)

	Freq	Percent
.	5	.
1	6	17.647
2	28	82.353

DIARRHOEA (Q A37)

	Freq	Percent
.	6	.
1	3	9.091
2	30	90.909

CONSTIPATION (Q A37)

	Freq	Percent
.	9	.
1	1	3.333
2	28	93.333
3	1	3.333

PALE COMPLEXION (Q A37)

	Freq	Percent
.	5	.
1	4	11.765
2	28	82.353
3	2	5.882

TINNED FOOD (Q B1)

	Freq	Percent
2	4	10.256
3	6	15.385
4	22	56.410
5	7	17.949

FRESH MEAT (DAYS/WEEK) (Q B2)

	Freq	Percent
.	2	.
2	1	2.703
3	5	13.514
4	2	5.405
5	7	18.919
6	4	10.811
7	18	48.649

TINNED MEAT (DAYS/WEEK) (Q B2)

	Freq	Percent
.	24	.
0	12	80.000
1	3	20.000

FRESH VEGETABLES (DAYS/WEEK) (Q B2)

	Freq	Percent
.	6	.
0	1	3.030
1	2	6.061
2	2	6.061
4	5	15.152
5	3	9.091
6	1	3.030
7	19	57.576

TINNED VEGETABLES (DAYS/WEEK) (Q B2)

	Freq	Percent
.	16	.
0	8	34.783
1	6	26.087
2	7	30.435
3	1	4.348
4	1	4.348

FRESH FISH (DAYS/WEEK) (Q B2)

	Freq	Percent
.	10	.
0	1	3.448
1	19	65.517
2	7	24.138
3	2	6.897

TINNED FISH (DAYS/WEEK) (Q B2)

	Freq	Percent
.	27	.
0	9	75.000
1	2	16.667
2	1	8.333

FRESH FRUIT (DAYS/WEEK) (Q B2)

	Freq	Percent
.	8	.
1	1	3.226
2	1	3.226
3	3	9.677
4	7	22.581
5	2	6.452
7	17	54.839

TINNED FRUIT (DAYS/WEEK) (Q B2)

	Freq	Percent
.	20	.
0	9	47.368
1	9	47.368
2	1	5.263

MEALIE-MEAL (DAYS/WEEK) (Q B2)

	Freq	Percent
.	18	.
0	14	66.667
1	3	14.286
2	1	4.762
3	1	4.762
6	1	4.762
7	1	4.762

BREAD (DAYS/WEEK) (Q B2)

	Freq	Percent
.	3	.
4	1	2.778
5	2	5.556
6	3	8.333
7	30	83.333

CEREALS (DAYS/WEEK) (Q B2)

	Freq	Percent
.	5	.
0	2	5.882
1	1	2.941
2	1	2.941
3	1	2.941
4	2	5.882
5	1	2.941
6	3	8.824
7	23	67.647

CHEESE (DAYS/WEEK) (Q B2)

	Freq	Percent
.	9	.
1	5	16.667
2	9	30.000
3	7	23.333
4	2	6.667
5	3	10.000
7	4	13.333

EGGS (DAYS/WEEK) (Q B2)

	Freq	Percent
.	10	.
1	6	20.690
2	14	48.276
3	3	10.345
4	1	3.448
5	3	10.345
7	2	6.897

MILK (DAYS/WEEK) (Q B2)

	Freq	Percent
.	7	.
0	2	6.250
3	1	3.125
4	1	3.125
5	1	3.125
6	1	3.125
7	26	81.250

COOLDRINK (DAYS/WEEK) (Q B2)

	Freq	Percent
.	12	.
1	2	7.407
2	6	22.222
3	5	18.519
4	2	7.407
5	1	3.704
7	11	40.741

CAKES/PUDDING (DAYS/WEEK) (Q B2)

	Freq	Percent
.	15	.
1	16	66.667
2	6	25.000
4	1	4.167
7	1	4.167

SWEETS (DAYS/WEEK) (Q B2)

	Freq	Percent
.	12	.
1	4	14.815
2	3	11.111
3	1	3.704
4	2	7.407
5	3	11.111
6	1	3.704
7	13	48.148

RELIGION (Q B3)

	Freq	Percent
3	39	100.000

HOME-GROWN VEGETABLES (Q B5)

	Freq	Percent
1	3	7.692
2	36	92.308

STEEL POTS (Q B6)

	Freq	Percent
.	2	.
1	14	37.838
2	23	62.162

ALUMINIUM POTS (Q B6)

	Freq	Percent
.	2	.
1	29	78.378
2	8	21.622

IRON POTS (Q B6)

	Freq	Percent
.	2	.
2	37	100.000

ENAMEL POTS (Q B6)

	Freq	Percent
.	2	.
1	8	21.622
2	29	78.378

OTHER POTS (Q B6)

	Freq	Percent
.	2	.
1	1	2.703
2	36	97.297

POTTERY DISHES (Q B7)

	Freq	Percent
.	1	.
1	4	10.526
2	34	89.474

HOME OWNED (Q C1)

	Freq	Percent
.	2	.
1	10	27.027
2	27	72.973

FLAT (Q C2)

	Freq	Percent
.	30	.
1	1	11.111
2	3	33.333
3	2	22.222
4	3	33.333

HOUSE (Q C3)

	Freq	Percent
.	10	.
1	27	93.103
2	2	6.897

AGE OF HOME (YEARS) (Q C4)

No.	9
Mean	53.9
Std. Dev.	18.3
Median	50

NUMBER OF ROOMS (Q C5)

No.	39
Mean	3.9
Std. Dev.	1.4
Median	4

NUMBER OF SLEEPING ROOMS (Q C6)

No.	38
Mean	2.6
Std. Dev.	0.8
Median	3

ELECTRICITY (Q C7)

	Freq	Percent
1	39	100.000

PARAFFIN (Q C7)

	Freq	Percent
.	16	.
2	23	100.000

GAS (Q C7)

	Freq	Percent
.	13	.
1	6	23.077
2	20	76.923

WOOD (Q C7)

	Freq	Percent
.	15	.
1	2	8.333
2	22	91.667

COAL (Q C7)

	Freq	Percent
.	17	.
2	22	100.000

CANDLES (Q C7)

	Freq	Percent
.	15	.
1	4	16.667
2	20	83.333

SUNLIGHT (Q C8)

	Freq	Percent
1	37	94.872
2	2	5.128

FRESH AIR (Q C9)

	Freq	Percent
1	37	94.872
2	2	5.128

SMOKE (Q C10)

	Freq	Percent
.	1	.
1	26	68.421
2	12	31.579

NUMBER OF SMOKERS (Q C11)

No	37
Mean	1.8
Std. Dev.	1.0
Median	2

HOUSE DUST (Q C12)

	Freq	Percent
1	5	12.821
2	24	61.538
3	10	25.641

OUTSIDE TOILET (Q C13)

	Freq	Percent
1	9	23.077
2	30	76.923

WATER PIPES (Q C14)

	Freq	Percent
.	5	.
1	18	52.941
2	3	8.824
4	2	5.882
5	10	29.412
6	1	2.941

WATER PIPES REPLACED (Q C15)

	Freq	Percent
.	2	.
1	14	37.838
2	9	24.324
3	14	37.838

YEARS AGO WATER PIPES REPLACED (Q C16)

	Freq	Percent
.	30	.
1	4	44.444
2	3	33.333
3	1	11.111
4	1	11.111

WATER PIPES PRIOR TO REPLACEMENT (Q C17)

	Freq	Percent
.	26	.
1	5	38.462
2	3	23.077
4	1	7.692
5	4	30.769

OUTSIDE FLAKING PAINT (Q C18)

	Freq	Percent
.	3	.
1	10	27.778
2	25	69.444
3	1	2.778

INSIDE FLAKING PAINT (Q C20)

	Freq	Percent
.	1	.
1	7	18.421
2	30	78.947
3	1	2.632

MAJOR REPAIRS NEEDED (Q C22)

	Freq	Percent
.	2	.
1	14	37.838

FACTORIES (Q C24)

	Freq	Percent
1	16	41.026
2	22	56.410
3	1	2.564

FUMES (Q C26)

	Freq	Percent
.	9	.
1	3	10.000
2	23	76.667
3	4	13.333

PETROL GARAGES (Q C28)

	Freq	Percent
.	2	.
1	12	32.432
2	25	67.568

WORKSHOPS (Q C29)

	Freq	Percent
.	4	.
1	9	25.714
2	25	71.429
3	1	2.857

NUMBER OF PEOPLE (Q D1)

	Freq	Percent
No		38
Mean		5.2
Std. Dev.		1.5
Median		5

NUMBER OF CHILDREN UNDER TWELVE (Q D2)

	Freq	Percent
No		37
Mean		2.4
Std. Dev.		1.1
Median		2

NUMBER OF SIBLINGS (Q D3)

	Freq	Percent
No.		38
Mean		1.9
Std. Dev.		1.2
Median		2

NUMBER OF WORKING PEOPLE (Q D5)

	Freq	Percent
No		39
Mean		1.5
Std. Dev.		1.0
Median		1

MOTHER (Q D6)

	Freq	Percent
1	38	97.436
2	1	2.564

FATHER (Q D6)

	Freq	Percent
1	31	79.487
2	8	20.513

FEMALE GUARDIAN (Q D6)

	Freq	Percent
1	2	5.128
2	37	94.872

MALE GUARDIAN (Q D6)

	Freq	Percent
1	3	7.692
2	36	92.308

FATHER'S SCHOOLING (YEARS COMPLETED) (Q D8)

	Freq	Percent
.	9	.
1	1	3.333
4	2	6.667
6	4	13.333
7	8	26.667
8	8	26.667
9	5	16.667
10	2	6.667

FATHER'S INCOME (RANDS/MONTH) (Q D9)

No.	28
Mean	748
Std. Dev.	314
Median	653

MOTHER'S SCHOOLING (YEARS COMPLETED) (Q D11)

	Freq	Percent
.	6	.
0	1	3.030
3	1	3.030
5	4	12.121
6	7	21.212
7	10	30.303
8	9	27.273
10	1	3.030

MOTHER'S INCOME (RANDS/MONTH) (Q D12)

No.	10
Mean	376
Std. Dev.	159
Median	360

TOTAL FAMILY INCOME (RANDS/MONTH) (Q D13)

No.	27
Mean	831
Std. Dev.	418
Median	800

NUMBER OF DEPENDANTS (Q D14)

No.	36
Mean	4.6
Std. Dev.	1.4
Median	4

NUMBER OF DEPENDANTS UNDER TWELVE YEARS (Q D15)

No.	36
Mean	2.4
Std. Dev.	1.0
Median	2

RENT (RANDS/MONTH) (Q D16)

No.	24
Mean	121
Std. Dev.	59
Median	105.5

WOODSTOCK QUESTIONNAIRE SURVEY : FREQUENCY ANALYSIS (COLOURED PUPILS)

HOME LANGUAGE (Q A8)

	Freq	Percent
.	2	.
1	40	47.619
2	20	23.810
5	23	27.381
6	1	1.190

TRANSPORT TO SCHOOL (Q A10)

	Freq	Percent
.	6	.
1	24	30.000
3	52	65.000
4	4	5.000

GRADE ONE REPEAT (Q A11)

	Freq	Percent
.	1	.
1	75	88.235
2	10	11.765

REPEAT-RATE (Q A12)

	Freq	Percent
.	76	.
1	9	90.000
2	1	10.000

SCHOOL WORK (Q A13)

	Freq	Percent
.	5	.
1	11	13.580
2	36	44.444
3	30	37.037
4	2	2.469
5	2	2.469

WALK SCHOOL BUSY STREETS (Q A14)

	Freq	Percent
.	7	.
1	48	60.759
2	31	39.241

PLAY-SITE (Q A15)

	Freq	Percent
.	6	.
1	37	46.250
3	1	1.250
4	16	20.000
5	15	18.750
6	1	1.250
7	9	11.250
8	1	1.250

PLAY BUSY STREETS (Q A16)

	Freq	Percent
.	2	.
1	19	22.619
2	62	73.810
3	3	3.571

WEEKDAYS OTHER HOMES (Q A17)

	Freq	Percent
.	2	.
1	20	23.810
2	62	73.810
3	2	2.381

WEEKENDS OTHER HOMES (Q A18)

	Freq	Percent
.	3	.
1	9	10.843
2	73	87.952
3	1	1.205

RESIDENCE PRESENT ADDRESS (Q A19)

	Freq	Percent
.	6	.
1	2	2.500
2	2	2.500
3	4	5.000
4	71	88.750
5	1	1.250

CRECHE ATTENDANCE (Q A20)

	Freq	Percent
.	2	.
1	41	48.810
2	43	51.190

VERY ACTIVE BEFORE SCHOOL (Q A21)

	Freq	Percent
.	3	.
1	70	84.337
2	13	15.663

MORE ACTIVE THAN OTHERS (Q A22)

	Freq	Percent
.	4	.
1	24	29.268
2	29	59.756
3	9	10.976

OVERACTIVE (Q A23)

	Freq	Percent
.	4	.
1	15	18.293
2	63	76.829
3	4	4.878

ACTIVE AT PRESENT (Q A24)

	Freq	Percent
.	5	.
1	53	65.432
2	24	29.630
3	4	4.938

DIFFICULTY CONCENTRATING (Q A25)

	Freq	Percent
.	5	.
1	26	32.099
2	49	60.494
3	6	7.407

EASILY DISTRACTED (Q A26)

	Freq	Percent
.	11	.
1	30	40.000
2	35	46.667
3	10	13.333

MORE EASILY DISTRACTED THAN OTHERS (Q A27)

	Freq	Percent
.	11	.
1	4	5.333
2	48	64.000
3	23	30.667

PAINT (Q A28)

	Freq	Percent
.	28	.
1	1	1.724
2	57	98.276

CEMENT (Q A28)

	Freq	Percent
.	31	.
1	2	3.636
2	53	96.364

PLASTER (Q A28)

	Freq	Percent
.	31	.
1	1	1.818
2	54	98.182

SOIL (Q A28)

	Freq	Percent
.	32	.
1	1	1.852
2	53	98.148

STICKS (Q A28)

	Freq	Percent
.	32	.
1	3	5.556
2	51	94.444

MATCHSTICKS (Q A28)

	Freq	Percent
.	33	.
1	1	1.887
2	52	98.113

CIGARETTE-ENDS (Q A28)

	Freq	Percent
.	31	.
1	2	3.636
2	53	96.364

OTHER ABNORMAL EATING HABITS (Q A28)

	Freq	Percent
.	23	.
1	1	1.587
2	62	98.413

AGE ONSET PICA (YEARS) (Q A29)

	Freq	Percent
.	78	.
1	4	50.000
2	2	25.000
4	1	12.500
6	1	12.500

PICA STILL PRESENT (Q A30)

	Freq	Percent
.	76	.
1	1	10.000
2	9	90.000

AGE STOP PICA (YEARS) (Q A31)

	Freq	Percent
.	79	.
2	3	42.857
4	2	28.571
6	1	14.286
7	1	14.286

FREQUENCY PICA (Q A32)

	Freq	Percent
.	76	.
1	1	10.000
4	6	60.000
5	3	30.000

CHILD WELL PRESENT (Q A33)

	Freq	Percent
.	2	.
1	72	85.714
2	5	5.952
3	7	8.333

MEDICAL TREATMENT (Q A35)

	Freq	Percent
.	10	.
1	8	10.526
2	67	88.158
3	1	1.316

FITS (Q A36)

	Freq	Percent
.	21	.
1	3	4.615
2	62	95.385

ANAEMIA (Q A36)

	Freq	Percent
.	22	.
1	3	4.688
2	59	92.188
3	2	3.125

POOR SPEECH (Q A36)

	Freq	Percent
.	19	.
1	12	17.910
2	55	82.090

KIDNEY PROBLEMS (Q A36)

	Freq	Percent
.	23	.
2	63	100.000

POOR HEARING (Q A36)

	Freq	Percent
.	20	.
1	9	13.636
2	56	84.848
3	1	1.515

POOR EYESIGHT (Q A36)

	Freq	Percent
.	23	.
1	3	4.762
2	58	92.063
3	2	3.175

GENERAL POOR HEALTH (Q A36)

	Freq	Percent
.	18	.
1	8	11.765
2	59	86.765
3	1	1.471

CLUMSINESS (Q A37)

	Freq	Percent
.	21	.
1	8	12.308
2	56	86.154
3	1	1.538

TIREDNESS (Q A37)

	Freq	Percent
.	16	.
1	14	20.000
2	56	80.000

LOSS OF APPETITE (Q A37)

	Freq	Percent
.	17	.
1	24	34.783
2	45	65.217

POOR EATING HABITS (Q A37)

	Freq	Percent
.	13	.
1	31	42.466
2	41	56.164
3	1	1.370

WEIGHT LOSS (Q A37)

	Freq	Percent
.	20	.
1	15	22.727
2	48	72.727
3	3	4.545

VOMITING (Q A37)

	Freq	Percent
.	26	.
1	7	11.667
2	53	88.333

DIARRHOEA (Q A37)

	Freq	Percent
.	23	.
1	9	14.286
2	53	84.127
3	1	1.587

CONSTIPATION (Q A37)

	Freq	Percent
.	23	.
1	10	15.873
2	53	84.127

PALE COMPLEXION (Q A37)

	Freq	Percent
.	20	.
1	9	13.636
2	55	83.333
3	2	3.030

TINNED FOOD
(Q B1)

	Freq	Percent
.	11	.
1	6	8.000
2	3	4.000
3	15	20.000
4	42	56.000
5	8	10.667
6	1	1.333

FRESH MEAT (DAYS/WEEK)
(Q B2)

	Freq	Percent
.	21	.
1	1	1.538
2	3	4.615
3	7	10.769
4	8	12.308
5	7	10.769
6	7	10.769
7	32	49.231

TINNED MEAT (DAYS/WEEK)
(Q B2)

	Freq	Percent
.	51	.
0	28	80.000
1	5	14.286
2	1	2.857
7	1	2.857

FRESH VEGETABLES (DAYS/WEEK) (Q B2)

	Freq	Percent
.	22	.
0	1	1.563
1	4	6.250
2	10	15.625
3	17	26.563
4	1	1.563
5	4	6.250
6	3	4.688
7	24	37.500

TINNED VEGETABLES (DAYS/WEEK) (Q B2)

	Freq	Percent
.	50	.
0	10	27.778
1	15	41.667
2	6	16.667
3	4	11.111
5	1	2.778

FRESH FISH (DAYS/WEEK) (Q B2)

	Freq	Percent
.	24	.
0	1	1.613
1	46	74.194
2	11	17.742
3	2	3.226
4	2	3.226

TINNED FISH (DAYS/WEEK) (Q B2)

	Freq	Percent
.	61	.
0	18	72.000
1	7	28.000

FRESH FRUIT (DAYS/WEEK) (Q B2)

	Freq	Percent
.	24	.
1	1	1.613
2	3	4.839
3	5	8.065
4	4	6.452
5	2	3.226
6	1	1.613
7	46	74.194

TINNED FRUIT (DAYS/WEEK) (Q B2)

	Freq	Percent
.	55	.
0	9	29.032
1	20	64.516
2	2	6.452

MEALIE-MEAL (DAYS/WEEK) (Q B2)

	Freq	Percent
.	55	.
0	20	64.516
1	2	6.452
2	5	16:129
3	1	3.226
7	3	9.677

BREAD (DAYS/WEEK) (Q B2)

	Freq	Percent
.	22	.
1	1	1.563
5	4	6.250
6	2	3.125
7	57	89.063

CEREALS (DAYS/WEEK) (Q B2)

	Freq	Percent
.	35	.
0	3	5.882
1	4	7.843
2	5	9.804
3	2	3.922
5	3	5.882
7	34	66.667

CHEESE (DAYS/WEEK) (Q B2)

	Freq	Percent
.	32	.
0	2	3.704
1	12	22.222
2	14	25.926
3	12	22.222
4	1	1.852
5	4	7.407
7	9	16.667

EGGS (DAYS/WEEK) (Q B2)

	Freq	Percent
.	33	.
0	2	3.774
1	14	26.415
2	22	41.509
3	10	18.868
4	2	3.774
7	3	5.660

MILK (DAYS/WEEK) (Q B2)

	Freq	Percent
.	41	.
1	1	2.222
3	1	2.222
5	1	2.222
6	1	2.222
7	41	91.111

COOLDRINK (DAYS/WEEK) (Q B2)

	Freq	Percent
.	49	.
1	3	8.108
2	8	21.622
3	5	13.514
4	1	2.703
5	3	8.108
7	17	45.946

CAKES/PUDDING (DAYS/WEEK) (Q B2)

	Freq	Percent
.	48	.
0	1	2.632
1	14	36.842
2	10	26.316
3	7	18.421
4	1	2.632
7	5	13.158

SWEETS (DAYS/WEEK) (Q B2)

	Freq	Percent
.	28	.
1	4	6.897
2	4	6.897
3	6	10.345
6	1	1.724
7	43	74.138

RELIGION (Q B3)

	Freq	Percent
.	16	.
2	28	40.000
3	26	37.143
5	16	22.857

HOME-GROWN VEGETABLES (Q B5)

	Freq	Percent
.	3	.
1	3	3.614
2	80	96.386

STEEL POTS (Q B6)

	Freq	Percent
	7	.
1	43	54.430
2	36	45.570

ALUMINIUM POTS (Q B6)

	Freq	Percent
.	8	.
1	46	58.974
2	32	41.026

IRON POTS (Q B6)

	Freq	Percent
.	8	.
1	2	2.564
2	76	97.436

ENAMEL POTS (Q B6)

	Freq	Percent
.	8	.
1	6	7.692
2	72	92.308

OTHER POTS (Q B6)

	Freq	Percent
.	8	.
1	5	6.410
2	73	93.590

POTTERY DISHES (Q B7)

	Freq	Percent
.	7	.
1	7	8.861
2	72	91.139

HOME OWNED (Q C1)

	Freq	Percent
.	2	.
1	22	26.190
2	62	73.810

FLAT (Q C2)

	Freq	Percent
.	81	.
1	3	60.000
2	2	40.000

HOUSE (Q C3)

	Freq	Percent
.	36	.
1	38	76.000
2	12	24.000

AGE OF HOME (YEARS) (Q C4)

No	35
Mean	51.7
Std. Dev	24.7
Median	50

NUMBER OF ROOMS (Q C5)

No	77
Mean	3.4
Std. Dev	1.5
Median	3

NUMBER OF SLEEPING ROOMS (Q C6)

No	80
Mean	2.5
Std. Dev	1.1
Median	2

ELECTRICITY (Q C7)

	Freq	Percent
.	4	.
1	76	92.683
2	6	7.317

PARAFFIN (Q C7)

	Freq	Percent
.	30	.
1	12	21.429
2	44	78.571

GAS (Q C7)

	Freq	Percent
.	20	.
1	31	46.970
2	34	51.515

WOOD (Q C7)

	Freq	Percent
.	32	.
1	1	1.852
2	53	98.148

COAL (Q C7)

	Freq	Percent
.	31	.
1	6	10.909
2	49	89.091

CANDLES (Q C7)

	Freq	Percent
.	32	.
2	54	100.000

SUNLIGHT (Q C8)

	Freq	Percent
.	5	.
1	65	80.247
2	16	19.753

FRESH AIR (Q C9)

	Freq	Percent
.	32	.
1	53	98.148
2	1	1.852

SMOKE (Q C10)

	Freq	Percent
.	3	.
1	36	43.373
2	47	56.627

NUMBER OF SMOKERS (Q C11)

No.	77
Mean	2.1
Std. Dev.	1.8
Median	2

HOUSE DUST (QA C12)

	Freq	Percent
.	3	.
1	24	28.916
2	29	34.940
3	30	36.145

OUTSIDE TOILET (Q C13)

	Freq	Percent
.	3	.
1	47	56.627
2	36	43.373

WATER PIPES (Q C14)

	Freq	Percent
.	13	.
1	47	64.384
2	11	15.068
3	2	2.740
4	3	4.110
5	10	13.699

WATER PIPES REPLACED (Q C15)

	Freq	Percent
.	6	.
1	20	25.000
2	37	46.250
3	23	28.750

YEARS AGO WATER PIPES REPLACED (Q C16)

	Freq	Percent
.	70	.
1	6	37.500
2	1	6.250
3	5	31.250
4	2	12.500
6	1	6.250
8	1	6.250

WATER PIPES PRIOR TO REPLACEMENT (Q C17)

	Freq	Percent
.	63	.
1	10	43.478
2	5	21.739
4	1	4.348
5	7	30.435

OUTSIDE FLAKING PAINT (Q C18)

	Freq	Percent
.	5	.
1	26	32.099
2	50	61.728
3	5	6.173

INSIDE FLAKING PAINT (Q C20)

	Freq	Percent
.	12	.
1	17	22.973
2	55	74.324
3	2	2.703

MAJOR REPAIRS NEEDED (Q C22)

	Freq	Percent
.	10	.
1	24	31.579
2	43	56.579
3	9	11.842

FACTORIES (Q C24)

	Freq	Percent
.	3	.
1	50	60.241
2	32	38.554
3	1	1.205

FUMES (Q C26)

	Freq	Percent
.	17	.
1	4	5.797
2	56	81.159
3	9	13.043

PETROL GARAGES (Q C28)

	Freq	Percent
.	6	.
1	31	38.750
2	49	61.250

WORKSHOPS (Q C29)

	Freq	Percent
.	13	.
1	31	42.466
2	40	54.795
3	2	2.740

NUMBER OF PEOPLE (Q D1)

No	78
Mean	7.8
Std. Dev.	5.0
Median	6

NUMBER OF CHILDREN UNDER TWELVE YEARS (Q D2)

No	79
Mean	3.2
Std. Dev.	2.3
Median	3

NUMBER OF SIBLINGS (Q D3)

No.	80
Mean	2.3
Std. Dev.	1.7
Median	2

NUMBER OF PEOPLE WORKING (Q D5)

No	75
Mean	2.3
Std. Dev.	1.5
Median	2

MOTHER (Q D6)

	Freq	Percent
.	6	.
1	79	98.750
2	12	1.250

FATHER (Q D6)

	Freq	Percent
.	6	.
1	56	70.000
2	24	30.000

FEMALE GUARDIAN (Q D6)

	Freq	Percent
.	6	.
1	3	3.750
2	77	96.250

MALE GUARDIAN (Q D6)

	Freq	Percent
.	6	.
1	2	2.500
2	78	97.500

FATHER'S SCHOOLING (YEARS COMPLETED)(Q D8)

	Freq	Percent
.	17	.
1	1	1.449
2	1	1.449
3	6	8.696
4	6	8.696
5	6	8.696
6	16	23.188
7	10	14.493
8	16	23.188
9	2	2.899
10	5	7.246

FATHER'S INCOME (RANDS/MONTH) (Q D9)

No	50
Mean	517
Std. Dev.	350
Median	400

MOTHER'S SCHOOLING (YEARS COMPLETED) (Q D11)

	Freq	Percent
.	10	.
3	4	5.263
4	5	6.579
5	10	13.158
6	21	27.632
7	16	21.053
8	13	17.105
9	1	1.316
10	5	6.579

MOTHER'S INCOME (RANDS/MONTH) (Q D12)

No.	30
Mean	290
Std. Dev.	217
Median	200

TOTAL FAMILY INCOME (RANDS/MONTH) (Q D13)

No.	41
Mean	675
Std. Dev.	497
Median	520

NUMBER OF DEPENDANTS (Q D14)

No.	54
Mean	5.4
Std. Dev.	1.9
Median	5

NUMBER OF DEPENDANTS UNDER TWELVE YEARS (Q D15)

No.	60
Mean	2.6
Std. Dev.	1.0
Median	2

RENT (RANDS/MONTH) (Q D16)

No.	49
Mean	54
Std. Dev.	35
Median	50

APPENDIX D4

QUESTIONNAIRE SURVEY : CROSS TABULATIONS AND CORRELATION ANALYSES (WHITE PUPILS)

TRANSPORT TO SCHOOL (Q A10)

	Car, Bus, Train	Walk
No.	9	28
Mean (ug/dl)	8	11
Std. Dev.	6	5
Median	8	10
Stat. Test	Mann-Whitney	
"p" Value	.1101	

SCHOOL WORK (Q A13)

	Good	Average
No.	18	18
Mean (ug/dl)	11	10
Std. Dev.	5	5
Median	10	9
Stat. Test	Mann-Whitney	
"p" Value	.4463	

WALK SCHOOL BUSY STREETS (Q A14)

	Yes	No
No	18	19
Mean (ug/dl)	10	11
Std. Dev.	4	6
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.5423	

PLAY-SITE (Q A15)

	Inside House	Back Yard	Front Yard	Garage/ Carport
No	11	11	9	6
Mean (ug/dl)	8	9	13	13
Std. Dev.	4	5	4	4
Median	9	7	12	14
Stat. Test.	Kruskal-Wallis			
"p" Value	.0350			

WEEKDAYS OTHER HOMES (Q A17)

	Yes	No
No	7	31
Mean (ug/dl)	10	11
Std. Dev.	4	5
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.7626	

RESIDENCE PRESENT ADDRESS (Q A19)

	Less than 2 years	More than 2 years
No	17	19
Mean (ug/dl)	10	11
Std. Dev.	5	5
Median	9	11
Stat. Test.	Mann-Whitney	
"p" Value	.4176	

CRECHE ATTENDANCE (Q A20)

	Yes	No
No	23	15
Mean (ug/dl)	11	11
Std. Dev.	5	6
Median	10	9
Stat. Test.	Mann-Whitney	
"p" Value	.7193	

MORE ACTIVE THAN OTHERS (Q A22)

	Yes	No
No	9	22
Mean (ug/dl)	15	9
Std. Dev.	5	4
Median	13	9
Stat. Test.	Mann-Whitney	
"p" Value	.0059	

DIFFICULTY CONCENTRATING (Q A25)

	Yes	No
No	11	20
Mean (ug/dl)	8	11
Std. Dev.	4	5
Median	7	10
Stat. Test.	Mann-Whitney	
"p" Value	.1017	

EASILY DISTRACTED (Q A26)

	Yes	No
No	20	10
Mean (ug/dl)	10	13
Std. Dev.	5	5
Median	9	11
Stat. Test.	Mann-Whitney	
"p" Value	.0576	

LOSS OF APPETITE (Q A37)

	Yes	No
No	11	21
Mean (ug/dl)	8	12
Std. Dev.	4	6
Median	8	10
Stat. Test.	Mann-Whitney	
"p" Value	.0701	

POOR EATING HABITS (Q A37)

	Yes	No
No	18	18
Mean (ug/dl)	11	10
Std. Dev.	6	5
Median	10	9
Stat. Test.	Mann-Whitney	
"p" Value	.5154	

WEIGHT LOSS (Q A37)

	Yes	No
No	8	23
Mean (ug/dl)	11	11
Std. Dev.	5	6
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.9639	

FRESH MEAT (DAYS/WEEK) (Q B2)

	Yes	No
No	19	17
Mean (ug/dl)	10	11
Std. Dev.	6	4
Median	10	11
Stat. Test.	Mann-Whitney	
"p" Value	.4176	

FRESH VEGETABLES (DAYS/WEEK) (Q B2)

	0-6	7
No	13	19
Mean (ug/dl)	11	11
Std. Dev.	5	6
Median	10	11
Stat. Test.	Mann-Whitney	
"p" Value	.7729	

TINNED VEGETABLES (DAYS/WEEK) (Q B2)

	0	1-7
No	7	15
Mean (ug/dl)	12	11
Std. Dev.	5	6
Median	12	9
Stat. Test.	Mann-Whitney	
"p" Value	.3051	

FRESH FISH (DAYS/WEEK) (Q B2)

	0-1	2-7
No	20	8
Mean (ug/dl)	12	7
Std. Dev.	5	6
Median	11	4
Stat. Test.	Mann-Whitney	
"p" Value	.0389	

FRESH FRUIT (DAYS/WEEK) (Q B2)

	0-6	7
No	14	16
Mean (ug/dl)	12	9
Std. Dev.	6	5
Median	9	8
Stat. Test.	Mann-Whitney	
"p" Value	.1082	

TINNED FRUIT (DAYS/WEEK) (Q B2)

	0	1-7
No	8	10
Mean (ug/dl)	11	11
Std. Dev.	5	6
Median	11	9
Stat. Test.	Mann-Whitney	
"p" Value	.5930	

CEREALS (DAYS/WEEK) (Q B2)

	0-6	7
No	10	23
Mean (ug/dl)	11	10
Std. Dev.	5	5
Median	10	9
Stat. Test.	Mann-Whitney	
"p" Value	.5554	

CHEESE (DAYS/WEEK) (Q B2)

	0-2	3
No	14	15
Mean (ug/dl)	10	11
Std. Dev.	5	6
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.5543	

EGGS (DAYS/WEEK) (Q B2)

	0-2	3-7
No	19	9
Mean (ug/dl)	12	9
Std. Dev.	5	7
Median	10	7
Stat. Test.	Mann-Whitney	
"p" Value	.1598	

COOLDRINK (DAYS/WEEK) (Q B2)

	0-6	7
No	1	11
Mean (ug/dl)	12	7
Std. Dev.	5	4
Median	11	7
Stat. Test.	Mann-Whitney	
"p" Value	.0143	

CAKE/PUDDING (DAYS/WEEK) (Q B2)

	0-1	2-7
No	15	8
Mean (ug/dl)	11	12
Std. Dev.	6	5
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.6041	

SWEETS (DAYS/WEEK) (Q B2)

	0-6	7
No	13	13
Mean (ug/dl)	9	11
Std. Dev.	6	5
Median	9	11
Stat. Test.	Mann-Whitney	
"p" Value	.3276	

STEEL POTS (Q B6)

	Yes	No
No	14	23
Mean (ug/dl)	10	11
Std. Dev.	5	6
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.5299	

ALUMINIUM POTS (Q B6)

	Yes	No
No	29	8
Mean (ug/dl)	10	12
Std. Dev.	6	4
Median	9	11
Stat. Test.	Mann-Whitney	
"p" Value	.1438	

ENAMEL POTS (Q B6)

	Yes	No
No	8	29
Mean (ug/dl)	9	11
Std. Dev.	4	6
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.5293	

HOME OWNED (Q C1)

	Yes	No
No	10	26
Mean (ug/dl)	12	10
Std. Dev.	6	5
Median	11	9
Stat. Test.	Mann-Whitney	
"p" Value	.5238	

SMOKE (Q C10)

	Yes	No
No	26	11
Mean (ug/dl)	12	8
Std. Dev.	5	6
Median	10	5
Stat. Test.	Mann-Whitney	
"p" Value	.0803	

HOUSE DUST (Q C12)

	Very/Slightly Dusty	Not Dusty
No	29	9
Mean (ug/dl)	11	10
Std. Dev.	5	5
Median	10	11
Stat. Test.	Mann-Whitney	
"p" Value	.7830	

OUTSIDE TOILET (Q C13)

	Yes	No
No	9	29
Mean (ug/dl)	11	11
Std. Dev.	6	5
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.8229	

WATER PIPES REPLACED (Q C15)

	Yes	No
No	13	9
Mean (ug/dl)	12	9
Std. Dev.	7	3
Median	15	8
Stat. Test.	Mann-Whitney	
"p" Value	.1916	

OUTSIDE FLAKING PAINT (Q C18)

	Yes	No
No	10	24
Mean (ug/dl)	10	11
Std. Dev.	6	5
Median	8	10
Stat. Test.	Mann-Whitney	
"p" Value	.4042	

MAJOR REPAIRS NEEDED (Q C22)

	Yes	No
No	14	22
Mean (ug/dl)	10	11
Std. Dev.	5	6
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.06370	

FACTORIES (Q C24)

	Yes	No
No	16	21
Mean (ug/dl)	11	11
Std. Dev.	6	5
Median	9	10
Stat. Test.	Mann-Whitney	
"p" Value	.8900	

PETROL-GARAGES (Q C28)

	Yes	No
No	12	24
Mean (ug/dl)	10	11
Std. Dev.	6	5
Median	10	10
Stat. Test.	Mann-Whitney	
"p" Value	.6138	

WORKSHOPS (Q C29)

	Yes	No
No	9	24
Mean (ug/dl)	12	10
Std. Dev.	6	5
Median	12	9
Stat. Test.	Mann-Whitney	
"p" Value	.5034	

PARENTAL CARE (Q D6)

	One Parent	Two Parents
No	31	7
Mean (ug/dl)	11	11
Std. Dev.	6	3
Median	10	11
Stat. Test.	Mann-Whitney	
"p" Value	.5584	

APPENDIX D5

BLOOD LEAD CONCENTRATIONS (ug/dl)
(RESPONDENTS VS NON-RESPONDENTS)
(SIGNIFICANT VARIABLES)(COLOURED PUPILS)

EASILY DISTRACTED (Q A26)

	Respondents	Non-Respondents
No.	65	21
Mean	17	20
Stat Test	Mann-Whitney	
"p" Value	.0137	

MORE DISTRACTED THAN OTHERS (Q A27)

	Respondents	Non-Respondents
No.	52	34
Mean	16	20
Stat Test	Mann-Whitney	
"p" Value	.0033	

DIARRHOEA (Q A37)

	Respondents	Non-Respondents
No.	62	24
Mean	17	19
Stat Test	Mann-Whitney	
"p" Value	.0500	

FRESH MEAT (Q B2)

	Respondents	Non-Respondents
No.	65	21
Mean	17	20
Stat Test	Mann-Whitney	
"p" Value	.0153	

CHEESE (Q B2)

	Respondents	Non-Respondents
No.	54	32
Mean	16	20
Stat Test	Mann-Whitney	
"p" Value	.0159	

EGGS (Q B2)

	Respondents	Non-Respondents
No.	53	33
Mean	16	20
Stat Test	Mann-Whitney	
"p" Value	.0131	

MILK (Q B2)

	Respondents	Non-Respondents
No.	45	41
Mean	15	20
Stat Test	Mann-Whitney	
"p" Value	.0040	

COOLDRINK (Q B2)

	Respondents	Non-Respondents
No.	37	49
Mean	16	19
Stat Test	Mann-Whitney	
"p" Value	.0073	

CAKE/PUDDING (Q B2)

	Respondents	Non-Respondents
No.	38	48
Mean	16	19
Stat Test	Mann-Whitney	
"p" Value	.0123	

COAL (Q C7)

	Respondents	Non-Respondents
No.	55	31
Mean	18	16
Stat Test	Mann-Whitney	
"p" Value	.0467	

FRESH AIR (Q C9)

	Respondents	Non-Respondents
No.	54	32
Mean	16	20
Stat Test	Mann-Whitney	
"p" Value	.0026	

RENT (Q D16)

	Respondents	Non-Respondents
No.	49	37
Mean	19	16
Stat Test	Mann-Whitney	
"p" Value	.0130	

APPENDIX D6

VARIABLES EXCLUDED FROM STATISTICAL ANALYSES (COLOURED PUPILS)

Variable	Question Number
Repeat-Rate	A12
Weekends Other Homes	A18
Residence Present Address	A19
More Easily Distracted Than Others	A27
Paint Cement Plaster Soil Sticks Matchsticks Cigarette Ends Other Abnormal Eating Habits	A28
Age Onset Pica	A29
Pica Still Present	A30
Age Stop Pica	A31
Frequency Pica	A32
Child Well Present	A33
Medical Treatment	A35
Fits Anaemia Kidney Problems Poor Hearing Poor Eyesight General Poor Health	A36
Clumsiness Vomiting Diarrhoea Constipation Pale Complexion	A37
Tinned Meat Tinned Fish Tinned Fruit Bread Milk	B2
Home Grown Vegetables	B5
Iron Pots Enamel Pots Other Pots	B6

Pottery Dishes	B7
Flat	C2
Electricity Wood Coal Candles	C7
Fresh Air	C8
Years Ago Water Pipes Replaced	C16
Water Pipes Prior to Replacement	C17
Fumes/Smoke	C26
Mother Female Guardian Male Guardian	D6

APPENDIX D7

QUESTIONNAIRE SURVEY: CROSS TABULATIONS AND CORRELATION ANALYSES (COLOURED PUPILS)

HOME LANGUAGE (Q A8)

	English	Afrikaans	Eng & Afr
No.	40	20	23
Mean (ug/dl)	16	22	16
Std Dev.	6	6	5
Median	16	21	16
Stat. Test	Kruskal Wallis		
"p" Value	.0011		

TRANSPORT TO SCHOOL (Q A10)

	Car, Bus, Train	Walk
No.	28	52
Mean (ug/dl)	17	18
Std. Dev.	5	7
Median	16	18
Stat. Test	t-test	
"p" value	.2312	

GRADE ONE REPEAT (Q A11)

	Yes	No
No.	10	75
Mean (ug/dl)	20	17
Std. Dev.	6	6
Median	18	17
Stat. Test	Mann-Whitney	
"p" value	.1609	

SCHOOL WORK (Q A13)

	Excellent	Good	Average-Poor
No.	11	36	32
Mean	17	16	18
Std. Dev.	4	6	7
Median	17	16	18
Stat. Test	Kruskal Wallis		
"p" Value	.4518		

WALK SCHOOL BUSY STREETS (Q A14)

	Yes	No
No.	48	31
Mean (ug/dl)	18	17
Std. Dev.	7	5
Median	18	17
Stat. Test	Mann-Whitney	
"p" value	.4382	

PLAY-SITE (Q A15)

	Inside House	Backyard	Frontyard	Street
No.	37	16	15	9
Mean (ug/dl)	16	20	17	21
Std. Dev.	7	6	6	5
Median	15	18	17	21
Stat. Test	Kruskal Wallis			
Level of Significance "p" Value	.0704			

PLAY BUSY STREETS (Q A16)

	Yes	No
No.	19	62
Mean (ug/dl)	19	17
Std. Dev.	7	6
Median	17	17
Stat. Test	Mann-Whitney	
"p" value	.2496	

WEEKDAYS OTHER HOMES(Q A17)

	Yes	No
No.	20	62
Mean (ug/dl)	19	17
Std. Dev.	9	5
Median	17	17
Stat. Test	Median	
"p" value	.7076	

CRECHE ATTENDANCE(Q A20)

	Yes	No
No.	41	43
Mean (ug/dl)	16	19
Std. Dev.	6	6
Median	16	18
Stat. Test	Mann-Whitney	
"p" value	.0358	

VERY ACTIVE BEFORE SCHOOL(Q A21)

	Yes	No
No.	70	13
Mean (ug/dl)	18	16
Std. Dev.	6	4
Median	17	15
Stat. Test	Mann-Whitney	
"p" value	.2254	

MORE ACTIVE THAN OTHERS(Q A22)

	Yes	No
No.	24	49
Mean (ug/dl)	19	16
Std. Dev.	7	6
Median	17	17
Stat. Test	Mann-Whitney	
"p" value	.2097	

OVERACTIVE (Q A23)

	Yes	No
No.	15	63
Mean (ug/dl)	20	16
Std. Dev.	7	6
Median	18	17
Stat. Test	Mann-Whitney	
"p" value	.0884	

ACTIVE AT PRESENT (Q A24)

	Yes	No
No.	53	24
Mean (ug/dl)	18	17
Std. Dev.	7	6
Median	17	16
Stat. Test	Mann-Whitney	
"p" value	.4872	

DIFFICULTY CONCENTRATING (Q A25)

	Yes	No
No.	26	49
Mean (ug/dl)	17	17
Std. Dev.	6	6
Median	17	17
Stat. Test	t-test	
"p" value	.7003	

EASILY DISTRACTED (Q A26)

	Yes	No
No.	30	35
Mean (ug/dl)	16	17
Std. Dev.	7	6
Median	15	17
Stat. Test	t-test	
"p" value	.5486	

PICA (Q A28)

	Some Evidence	No Evidence
No.	8	78
Mean (ug/dl)	21	17
Std. Dev.	6	6
Median	21	17
Stat. Test	Mann-Whitney	
"p" value	.1025	

POOR SPEECH (Q A36)

	Yes	No
No.	12	55
Mean (ug/dl)	19	17
Std. Dev.	8	6
Median	19	17
Stat. Test	Mann-Whitney	
"p" value	.1683	

HEALTH EFFECTS (Q A36)

	1 or More Health Effects	No Health Effects
No.	29	57
Mean (ug/dl)	20	17
Std. Dev.	7	5
Median	19	17
Stat. Test	Mann-Whitney	
"p" value	.0263	

TIREDNESS (Q A37)

	Yes	No
No.	14	56
Mean (ug/dl)	19	17
Std. Dev.	8	6
Median	19	17
Stat. Test	Median	
"p" value	.2824	

LOSS OF APPETITE (Q A37)

	Yes	No
No.	24	45
Mean (ug/dl)	19	16
Std. Dev.	7	6
Median	18	16
Stat. Test	Mann-Whitney	
"p" value	.0736	

POOR EATING HABITS (Q A37)

	Yes	No
No.	31	41
Mean (ug/dl)	17	17
Std. Dev.	7	6
Median	17	17
Stat. Test	t-test	
"p" value	.8591	

WEIGHT LOSS (Q A37)

	Yes	No
No.	15	48
Mean (ug/dl)	22	16
Std. Dev.	6	6
Median	22	16
Stat. Test	Mann-Whitney	
"p" value	.0018	

HEALTH SYMPTOMS (Q A37)

	1 or More Symptoms	No Symptoms
No.	39	47
Mean (ug/dl)	18	17
Std. Dev.	6	7
Median	17	17
Stat. Test	Mann-Whitney	
"p" value	.8278	

TINNED FOOD(Q B1)

	1 Or More Days/Week	Occasionally/ Never
No.	24	50
Mean (ug/dl)	20	16
Std. Dev.	7	6
Median	18	17
Stat. Test	Mann-Whitney	
"p" value	.0290	

FRESH MEAT (Q B2)

	1-6 Days/Week	7 Days/Week
No.	33	32
Mean (ug/dl)	16	18
Std. Dev.	6	5
Median	14	17
Stat. Test	Mann-Whitney	
"p" value	.0393	

FRESH VEGETABLES (Q B2)

	0-3 Days/Week	4-7 Days/Week
No.	32	32
Mean (ug/dl)	18	16
Std. Dev.	6	5
Median	17	16
Stat. Test	Mann-Whitney	
"p" value	.5780	

TINNED VEGETABLES (Q B2)

	0-1 Day/Week	2-7 Days/Week
No.	25	11
Mean (ug/dl)	15	22
Std. Dev.	5	5
Median	16	22
Stat. Test	Mann-Whitney	
"p" value	.0036	

FRESH FISH (Q B2)

	0-1 Day/Week	2-7 Days/Week
No.	47	15
Mean (ug/dl)	17	16
Std. Dev.	6	4
Median	16	18
Stat. Test	Mann-Whitney	
"p" value	.8560	

TINNED FISH (DAYS/WEEK) (Q B2)

	0	1-7
No.	18	7
Mean	18	14
Std. Dev.	6	7
Median	18	13
Stat. Test	Mann-Whitney	
"p" value	.1629	

FRESH FRUIT (Q B2)

	1-6 Days/Week	7 Days/Week
No.	16	46
Mean (ug/dl)	17	17
Std. Dev.	6	6
Median	16	17
Stat. Test	Mann-Whitney	
"p" value	.8011	

MEALIE-MEAL (Q B2)

	Never	1-7 Days/Week
No.	20	11
Mean (ug/dl)	15	20
Std. Dev.	5	5
Median	15	17
Stat. Test	Mann-Whitney	
"p" value	.0459	

CEREALS (Q B2)

	0-5 Days/Week	7 Days/Week
No.	17	34
Mean (ug/dl)	18	16
Std. Dev.	6	6
Median	18	16
Stat. Test	Mann-Whitney	
"p" value	.1666	

CHEESE (Q B2)

	0-2 Days/Week	3-7 Days/Week
No.	28	26
Mean (ug/dl)	18	15
Std. Dev.	6	5
Median	17	15
Stat. Test	Mann-Whitney	
"p" value	.0807	

EGGS (DAYS/WEEK) (Q B2)

	1	2	3-7
No.	16	22	15
Mean (ug/dl)	18	16	15
Std. Dev.	5	4	4
Median	16	15	17
Stat. Test	Kruskal-Wallis		
"p" value	.7303		

COOLDRINK (Q B2)

	1-5 Days/Week	7 Days/Week
No.	20	17
Mean (ug/dl)	16	16
Std. Dev.	7	4
Median	13	16
Stat. Test	Median	
"p" value	.5007	

CAKE/PUDDING (Q B2)

	0-1 Day/Week	2-7 Days/Week
No.	15	23
Mean (ug/dl)	15	16
Std. Dev.	6	6
Median	16	16
Stat. Test	t-test	
"p" value	.7561	

SWEETS (Q B2)

	1-6 Days/Week	7 Days/Week
No.	15	43
Mean (ug/dl)	15	18
Std. Dev.	7	6
Median	15	17
Stat. Test	Mann-Whitney	
"p" value	.1131	

RELIGION (Q B3)

	Islam	"Malay" (Muslim)	Christian
No.	16	28	26
Mean (ug.dl)	13	16	20
Std. Dev.	6	5	6
Median	12	16	18
Stat. Test	Kruskal-Wallis		
"p" value	.0018		

STEEL POTS (Q B6)

	Yes	No
No.	43	36
Mean (ug/dl)	17	19
Std. Dev.	6	6
Median	17	18
Stat. Test	Mann-Whitney	
"p" value	.2154	

ALUMINIUM POTS (Q B6)

	Yes	No
No.	46	32
Mean (ug/dl)	19	16
Std. Dev.	7	5
Median	18	16
Stat. Test	Mann-Whitney	
"p" value	.0197	

HOME OWNED (Q C1)

	Yes	No
No.	22	62
Mean (ug/dl)	14	19
Std. Dev.	6	6
Median	13	18
Stat. Test	Mann-Whitney	
"p" value	.0090	

HOUSE (Q C3)

	1 Storey	2 Storeys
No.	38	12
Mean (ug/dl)	17	19
Std. Dev.	6	8
Median	17	17
Stat. Test	Median	
"p" value	.7522	

AGE HOME (Q C4)

No.	35
Corr. Coeff (r)	.0884
Stat. Test	Spearman Correlation
"p" value	.6137

NUMBER OF ROOMS (Q C5)

No.	77
Corr. Coeff (r)	-.20180
Stat. Test	Spearman Correlation
"p" value	.0784

NUMBER OF SLEEPING ROOMS (Q C6)

No.	80
Corr. Coeff. (r)	-.23984
Stat. Test	Spearman Correlation
"p" value	.0321

PARAFFIN (Q C7)

	Yes	No
No.	12	44
Mean (ug/dl)	21	17
Std. Dev.	6	7
Median	21	17
Stat. Test	Mann-Whitney	
"p" value	.1306	

GAS (Q C7)

	Yes	No
No.	31	34
Mean (ug/dl)	18	18
Std. Dev.	6	7
Median	18	17
Stat. Test	t-test	
"p" value	.7699	

SUNLIGHT (Q C8)

	Yes	No
No.	65	16
Mean (ug/dl)	17	21
Std. Dev.	6	7
Median	17	19
Stat. Test	Mann-Whitney	
"p" value	.0408	

SMOKE (Q C10)

	Yes	No
No.	36	47
Mean (ug/dl)	19	17
Std. Dev.	7	6
Median	18.5	17
Stat. Test	Mann-Whitney	
"p" value	.1923	

NUMBER OF SMOKERS (Q C11)

No.	69
Corr. Coeff. (r)	.1699
Stat. Test	Spearman
"p" value	.1629

HOUSE DUST (Q C12)

	Very Dusty	Slightly Dusty	Not Dusty
No.	24	29	30
Mean (ug/dl)	21	16	16
Std. Dev.	7	5	5
Median	19	17	15
Stat. Test	Kruskal-Wallis		
"p" value	.0280		

OUTSIDE TOILET (Q C13)

	Yes	No
No.	47	36
Mean (ug/dl)	19	16
Std. Dev.	7	5
Median	18	17
Stat. Test	Mann-Whitney	
"p" value	.1120	

WATER PIPES (Q C14)

	Copper	Other
No.	47	16
Mean	18	18
Std. Dev.	6	5
Median	17	18
Stat. Test	t-test	
"p" value	.7564	

WATER PIPES REPLACED (Q C15)

	Yes	No
No.	20	37
Mean (ug/dl)	16	18
Std. Dev.	7	7
Median	16	17
Stat. Test	t-test	
"p" value	.3595	

OUTSIDE FLAKING PAINT (Q C18)

	Yes	No
No.	26	50
Mean (ug/dl)	20	16
Std. Dev.	8	5
Median	18	17
Stat. Test	Median	
"p" value	.1495	

INSIDE FLAKING PAINT (Q C20)

	Yes	No
No.	17	55
Mean (ug/dl)	19	17
Std. Dev.	8	5
Median	17	17
Stat. Test	Median	
"p" value	.8053	

MAJOR REPAIRS NEEDED (Q C22)

	Yes	No
No.	24	43
Mean (ug/dl)	20	16
Std. Dev.	7	5
Median	18	16
Stat. Test	Median	
"p" value	.0305	

FACTORIES (Q C24)

	Yes	No
No.	50	32
Mean (ug/dl)	19	15
Std. Dev.	7	5
Median	18	15
Stat. Test	Median	
"p" value	.0011	

PETROL GARAGES (Q C28)

	Yes	No
No.	31	49
Mean (ug/dl)	20	16
Std. Dev.	7	5
Median	19	17
Stat. Test	Median	
"p" value	.0761	

WORKSHOPS (Q C29)

	Yes	No
No.	31	40
Mean (ug/dl)	19	17
Std. Dev.	7	6
Median	18	17
Stat. Test	Mann-Whitney	
"p" value	.5189	

NUMBER OF PEOPLE (Q D1)

No.	78
Corr. Coeff. (r)	.23953
Stat. Test	Spearman Correlation
"p" value	.0347

NUMBER OF CHILDREN UNDER TWELVE YEARS (Q D2)

No.	79
Corr. Coeff. (r)	.14897
Stat. Test	Spearman Correlation
"p" value	.1901

NUMBER OF SIBLINGS (Q D3)

No.	80
Corr. Coeff. (r)	.24012
Stat. Test	Spearman Correlation
"p" value	.0319

NUMBER OF PEOPLE WORKING (Q D5)

No.	75
Corr. Coeff.(r)	-.03243
Stat. Test	Spearman Correlation
"p" value	.7824

PARENTAL CARE (Q D6)

	Two Parents	One Parent
No.	56	24
Mean (ug/dl)	16	21
Std. Dev.	6	7
Median	16	21
Stat. Test	Median	
"p" value	.0255	

FATHER'S SCHOOLING (Q D8)

	Up To 6 Years	More Than 6 Years
No.	36	33
Mean (ug/dl)	20	15
Std. Dev.	6	5
Median	19	14
Stat. Test	Mann-Whitney	
"p" value	.0011	

FATHER'S INCOME (Q D9)

No.	50
Corr. Coeff. (r)	-.56448
Stat. Test	Spearman Correlation
"p" value	.0001

MOTHER'S SCHOOLING (Q D11)

	Up To 6 Years	More Than 6 Years
No.	40	36
Mean (ug/dl)	21	15
Std. Dev.	7	5
Median	20	16
Stat. Test	Median	
"p" value	.0062	

MOTHER'S INCOME (Q D12)

No.	30
Corr. Coeff. (r)	-.32533
Stat. Test	Spearman Correlation
"p" value	.0794

TOTAL FAMILY INCOME (Q D13)

No.	41
Corr. Coeff. (r)	-.48046
Stat. Test	Spearman Correlation
"p" value	.0015

NUMBER OF DEPENDANTS (Q D14)

No.	54
Corr. Coeff. (r)	.22425
Stat. Test	Spearman Correlation
"p" value	.1031

NUMBER OF DEPENDANTS UNDER TWELVE (Q D15)

No.	60
Corr. Coeff. (r)	.11858
Stat. Test	Spearman Correlation
"p" value	.3669

RENT (Q D16)

No.	49
Corr. Coeff. (r)	-.05886
Stat. Test	Spearman Correlation
"p" value	.6879

OCCUPATIONAL DENSITY

No.	75
Corr. Coeff. (r)	.45786
Stat. Test	Spearman Correlation
"p" value	.0001

TOTAL FAMILY INCOME RELATIVE TO NUMBER OF DEPENDANTS

No.	39
Corr. Coeff. (r)	-.58321
Stat. Test	Spearman Correlation
"p" value	.0001

APPENDIX D8

SIGNIFICANT ASSOCIATIONS BETWEEN SIGNIFICANT VARIABLES (COLOURED PUPILS)

Variable	Statistical Test	Significance Level
Occupational Density Home Language (Q A8)	Kruskal-Wallis	p = 0.0040
Total Family Income (Q D13) Home Ownership (Q C1)	Mann Whitney	p = 0.0135
Income Relative to Number of Dependants Home Ownership (Q C1)	Mann Whitney	p = 0.0191
Total Family Income (Q D13) Factories (Q C24)	Mann Whitney	p = 0.0001
Occupational Density Factories (Q C24)	Mann Whitney	p = 0.0305
Income Relative to Number of Dependants Factories (Q C24)	Mann Whitney	p = 0.0018
Total Family Income (Q D13) Mother's Schooling (Q D11)	Mann Whitney	p = 0.0005
Occupational Density Mother's Schooling (Q D11)	Mann Whitney	p = 0.0283
Income Relative to Number of Dependants Mother's Schooling (Q D11)	Mann Whitney	p = 0.0008
Total Family Income (Q D13) Father's Schooling (Q D8)	Mann Whitney	p = 0.0100
Occupational Density Father's Schooling (Q D8)	Mann Whitney	p = 0.0056
Income Relative to Number of Dependants Father's Schooling (Q D8)	Mann Whitney	p = 0.0018
Total Family Income (Q D13) School (Q A9)	Mann Whitney	p = 0.0021
Occupational Density School (Q A9)	Mann Whitney	p = 0.0232

Home Language (Q A8) Mother's Schooling (Q D11)	Chi-square	p = 0.0001
Incomea Relative to Number of Dependants School (Q A9)	Mann Whitney	p = 0.0030
Home Language (Q A8) School (Q A9)	Chi-square	p = 0.0014
Tinned Vegetables (Q B2) Factories (Q C24)	Chi-square	p = 0.0222
Tinned Vegetables (Q B2) School (Q A9)	Chi-square	p = 0.0006
Home Ownership (Q C1) Mother's Schooling (Q D11)	Chi-square	p = 0.0012
Home Ownership (Q C1) Religion (Q B3)	Chi-square	p = 0.0004
Factories (Q C24) Father's Schooling (Q D8)	Chi-square	p = 0.0213
Factories (Q C24) School (Q A9)	Chi-square	p = 0.0007
Mother's Schooling (Q D11) School (Q A9)	Chi-square	p = 0.0005
Mother's Schooling (Q D11) Religion (Q B3)	Chi-square	p = 0.0043
Father's Schooling (Q D8) Mother's Schooling (Q D11)	Chi-square	p = 0.0001
Father's Schooling (Q D8) School (Q A9)	Chi-square	p = 0.0152
School (Q A9) Religion (Q B3)	Chi-square	p = 0.0056
Total Family Income (Q D13) Income Relative to Number of Dependants.	Spearman	p = 0.0001

APPENDIX E1

HOME WALKABOUTS AND CLEANLINESS RATING SCALE

ENVIRONMENTAL LEAD : PROJECT B

(HOME ENVIRONMENT ASSESSMENTS)

Family Cleanliness Scale*

1. Are there smells? (e.g. stale cigarette smoke, rotting food)

   .....

2. Is the floor covering in any room stained with dirt or soiled? (covered in bits, crumbs, etc.)

   .....

3. Is the general decorative level poor - obviously in need of attention? (e.g. badly stained wallpaper, broken windows)

   .....

4. Are the furniture or furnishings badly stained with dirt or soiled?

   .....

---

* Adapted from Marjorie Smith, Dept of Child Psychiatry, Hospital for Sick Children, London.

5. Do the furnishings in the house look as if they have not been dusted for a considerable period of time?

.....

6. Do the kitchen sink, work surfaces, or cupboard doors look as if they have not been washed for a considerable period?

.....

7. Do the cooking implements, cutlery or crockery remain unwashed until they are needed again?

.....

8. Does the lavatory, bath or basin show ingrained dirt?

.....

9. Are the informants' or childrens' clothing clearly unwashed or hair matted and unbrushed?

.....

10. Is the garden or yard uncared for, and/or strewn with rubbish?

.....

ENVIRONMENTAL LEAD : PROJECT B

(HOME ENVIRONMENT ASSESSMENTS)

Walkabouts

1. Is the house well-ventilated?

.....

2. Spacious?

.....

3. Are there furnishings?

.....

4. Is there carpeting in most rooms?

.....

5. Are there doormats?

.....

6. Are there any potentially dangerous structural or health defects? (e.g. missing floorboards, crumbling walls, etc.)

.....

7. Is there paint flaking from inside doors, windowsills, walls? (note room, surface, extent of flaking)

.....

8. Is there paint flaking from outside walls, windowsills, doors? (note room, surface, extent of flaking)

.....

9. What type of water piping is present? (note construction material of inlet and outlet pipes)

.....

10. Is there a back garden? (approx. area)

.....

11. Is there a front garden? (approx. area)

.....

12. Is there a yard?

.....

13. Any particular items lying in yard?

.....

14. Any structural or health hazards?

.....

15. Is the immediate neighbourhood aesthetically pleasing - trees, grass?

.....

16. Are there any factories, garages, workshops, major roads within 2 - 5 minutes walking distance? (note details).

.....

17. General comments.

.....

.....

APPENDIX E2

HOME INTERVIEWS

ENVIRONMENTAL LEAD: PROJECT B (Case Control Study)

A.

1. Name of Interviewee .....
2. Relation to Child .....
3. Home Language .....
4. Religion .....

B. Child's Medical History

1. Is X well at present? .....
2. Is X receiving medical treatment at present?.....
3. Has X ever stayed overnight in hospital? .....
4. Over the past 12 months has X had outpatient treatment? .....

C. Diet

1. Has X got a good appetite? .....
2. How many times / week does X eat tinned food? .....
3. How many times / week does X eat:

   fresh meat ..... tinned meat .....
   fresh veg ..... tinned veg .....
   fresh fish ..... tinned fish .....
   fresh fruit ....tinned fruit .....
   mealie meal .....
   bread .....
   cereals .....
   cheese .....
   eggs .....
   milk .....

cool drinks .....

cakes/pudding .....

sweets .....

4. What type of pots do you normally use for cooking?

D. Hand-Mouth Activities, General Activities (Supervision, Stimulation, Behaviour)

1. Have you ever noticed X eating

paint flakes .....

cement .....

plaster .....

soil/earth .....

sticks .....

matchsticks .....

cigarette ends .....

coal .....

other .....

2. How old was X when you first noticed this? .....

3. Does X still eat these things? .....

4. How old was X when he/she stopped? .....

5. How often did, or does X eat these things? .....

6. Do you notice X often sucking fingers or biting nails? .....

7. Do you notice X often putting hands, other objects in his mouth? .....

8. Does X often have noticeably dirty hands after playing? .....

Does X ever wash his hands on his own? .....

9. When does X wash his hands? .....

10. Where does X play most after school? .....

11. Does X often play at someone else's home? .....

12. Does X often play on the sidewalk of busy streets? ....

13. Did X attend a creche or playschool? .....

14. Who takes care of X most of the time? .....
15. What does X usually do after school? .....
16. Does X have his own toys? ..... books? .....
17. Does X play with toys often? .....
18. Does X play mainly alone or with other children? .....
19. What time does X go to bed? .....
20. Does X have his own room? ....., own bed?.....
21. Do family members ever take X on outings? .....
22. Has X ever been on a long trip or journey to another town? .....
23. Has X ever been to a museum? .....
24. How is X doing at school? .....
25. Did X pass grade one the first time round? .....
26. Is X an active or quiet child? .....
27. Do you think X is more active than normal? .....
28. Do you often need to use physical punishment? .....

E. Family Characteristics

1. How many people live in your home? .....
2. How many children under the age of 13 live here? .....
   Their ages? .....
3. How many sisters and brothers does X have? .....
   Ages? .....
4. Does X live with both parents? .....
5. What is your marital status? .....
6. Do any of the adults have particular hobbies? .....
   .....

F. Housing Characteristics

1. How long has X lived here? .....
2. Is your home owned or rented? .....
3. Do you know how old this house is? .....

4. How many rooms are there, excluding the bathroom and kitchen? .....
5. How many rooms are used for sleeping in? .....
6. What fuel do you use for cooking, heating etc? .....
7. How many people in the home smoke? .....
8. Has there been any painting, decorating or renovation in your house in the past year? .....
   Past two years? .....
9. Do you have a car? .....
10. Where is it kept? .....
11. Are motor repairs done at home? .....

G. Socio-Economic Status

1. What is your monthly rent? .....
2. What is the mother's level of schooling? .....
3. Does the mother work outside the home? .....
   Job? .....
4. What is the mother's monthly income? .....
5. What is the father's level of schooling? .....
6. What is the father's job? .....
7. What is the father's monthly income? .....
8. What is the total family income? (monthly) .....
9. How many people are supported on this? .....
10. How many children under the age of 13 are supported? .....

APPENDIX E3

HOME INTERVIEW ANALYSES

HOME LANGUAGE (Q A3)

	Cases	Controls
English		
No.	9	22
%	41	71
Afrikaans		
No.	13	9
%	59	29

Statistical Test : L.R. Chi Square
"p" value .0309

RELIGION (Q A4)

	Cases	Controls
Muslim		
No.	12	26
%	54	90
Christian		
No.	10	3
%	45	10

Statistical Test : L.R. Chi Square
"p" value .0050

CHILD WELL PRESENT (Q B1)

		Cases	Controls
Yes	No.	25	32
	%	93	91
No	No.	2	3
	%	7	9

Statistical Test : Fishers Exact Test
"p" value 1.000

MEDICAL TREATMENT (Q B2)

		Cases	Controls
Yes	No.	3	4
	%	11	11
No	No.	24	31
	%	89	89

Statistical Test : Fishers Exact Test
"p" value 1.00

HOSPITAL (Q B3)

		Cases	Controls
Yes	No.	8	7
	%	31	20
No	No.	18	28
	%	69	80

Statistical Test : Fishers Exact Test
"p" value .3789

FRESH MEAT (Q C3)

	Cases	Controls
No.	27	36
Mean	5	5
Std. Dev.	1.8	1.8
Median	5	6

Statistical Test : Kruskal-Wallis
"p" value .6887

FRESH VEGETABLES (Q C3)

	Cases	Controls
No.	27	36
Mean	4	4
Std. Dev.	2.8	2.3
Median	4	3

Statistical Test : Kruskal-Wallis
"p" value .6505

FRESH FISH (Q C3)

	Cases	Controls
No.	27	36
Mean	1	1
Std. Dev.	1.35	.63
Median	1	1

Statistical Test : Kruskal-Wallis
"p" value .7157

FRESH FRUIT (Q C3)

	Cases	Controls
No.	27	36
Mean	5	5
Std. Dev.	2.4	2.27
Median	7	7

Statistical Test : Kruskal-Wallis
"p" value .4967

MEALIE MEAL (Q C3)

	Cases	Controls
No.	27	36
Mean	.94	.94
Std. Dev.	2.05	2.03
Median	0	0

Statistical Test : Kruskal-Wallis
"p" value .9291

BREAD (Q C3)

	Cases	Controls
No.	27	36
Mean	7	6
Std. Dev.	.53	1.6
Median	7	7

Statistical Test : Kruskal-Wallis
"p" value .3661

CEREAL (Q C3)

	Cases	Controls
No.	27	36
Mean	4	5
Std. Dev.	3.07	2.95
Median	4	7

Statistical Test : Kruskal-Wallis
"p" value .4338

CHEESE (Q C3)

	Cases	Controls
No.	27	36
Mean	3	3
Std. Dev.	1.84	2.14
Median	2	3

Statistical Test : Kruskal-Wallis
"p" value .5147

EGGS (Q C3)

	Cases	Controls
No.	27	36
Mean	3	3
Std. Dev.	2.04	1.92
Median	2	2

Statistical Test : Kruskal-Wallis
"p" value .4057

MILK (Q C3)

	Cases	Controls
No.	27	36
Mean	5	6
Std. Dev.	3.17	2.05
Median	7	7

Statistical Test : Kruskal-Wallis
"p" value .0933

COOLDRINK (Q C3)

	Cases	Controls
No.	27	36
Mean	5	4
Std. Dev.	2.43	2.85
Median	7	3

Statistical Test : Kruskal-Wallis
"p" value .0974

CAKE/PUDDING (Q C3)

	Cases	Controls
No.	27	36
Mean	3	3
Std. Dev.	3.51	2.26
Median	2	2

Statistical Test : Kruskal-Wallis
"p" value .3011

SWEETS (Q C3)

	Cases	Controls
No.	27	36
Mean	6	5
Std. Dev.	2.46	2.43
Median	7	7

Statistical Test : Kruskal-Wallis
"p" value .7470

TINNED MEAT (Q C3)

	Cases	Controls
No.	27	36
Mean	.11	.02
Std. Dev.	.32	.16
Median	0	0

Statistical Test : Kruskal-Wallis
"p" value .1830

TINNED VEGETABLES (Q C3)

	Cases	Controls
No.	26	36
Mean	1	1
Std. Dev.	.92	1.02
Median	1	1

Statistical Test : Kruskal-Wallis
"p" value .9581

TINNED FISH (Q C3)

	Cases	Controls
No.	27	36
Mean	0	0
Std. Dev.	.63	.65
Median	0	0

Statistical Test : Kruskal-Wallis
"p" value .2706

TINNED FRUIT (Q C3)

	Cases	Controls
No.	27	36
Mean	0	0
Std. Dev.	.79	.78
Median	0	0

Statistical Test : Kruskal-Wallis
"p" value .5484

ALUMINIUM POTS (Q C4)

		Cases	Controls
Yes	No.	26	27
	%	100	75
No	No.	0	9
	%	0	25

Statistical Test : Fishers Exact Test
"p" value .0076

STEEL POTS (Q C4)

		Cases	Controls
Yes	No.	2	9
	%	8	25
No	No.	24	27
	%	92	75

Statistical Test : Fishers Exact Test
"p" value .1006

PAINT FLAKES (Q D1)

		Cases	Controls
Yes	No.	3	0
	%	11	0
No	No.	24	36
	%	89	100

Statistical Test : Fishers Exact Test
"p" value .0737

CEMENT (Q D1)

		Cases	Controls
Yes	No.	7	2
	%	26	6
No	No.	20	34
	%	74	94

Statistical Test : Fishers Exact Test
"p" value .0312

PLASTER (Q D1)

		Cases	Controls
Yes	No.	4	0
	%	15	0
No	No.	23	36
	%	85	100

Statistical Test : Fishers Exact Test
"p" value .0295

SOIL (Q D1)

		Cases	Controls
Yes	No.	10	5
	%	37	14
No	No.	17	31
	%	63	86

Statistical Test : Fishers Exact Test
"p" value .0410

STICKS (Q D1)

		Cases	Controls
Yes	No.	7	0
	%	26	0
No	No.	20	36
	%	74	100

Statistical Test : Fishers Exact Test
"p" value .0016

MATCHSTICKS (Q D1)

		Cases	Controls
Yes	No.	8	0
	%	30	0
No	No.	19	36
	%	70	100

Statistical Test : Fishers Exact Test
"p" value .0006

CIGARETTE ENDS (Q D1)

		Cases	Controls
Yes	No.	7	3
	%	26	8
No	No.	20	33
	%	74	92

Statistical Test : Fishers Exact Test
"p" value .0838

COAL (Q D1)

		Cases	Controls
Yes	No.	1	0
	%	4	0
No	No.	26	36
	%	96	100

Statistical Test : Fishers Exact Test
"p" value .4286

AGE ONSET PICA (YEARS) (Q D2)

	Cases	Controls
No.	19	12
Mean	2	2
Std. Dev.	1.13	.75
Median	2	2

Statistical Test : Kruskal-Wallis
"p" value .8626

PICA STILL PRESENT (Q D3)

		Cases	Controls
Yes	No.	3	0
	%	16	0
No	No.	16	12
	%	84	100

Statistical Test : Fishers Exact Test
"p" value .02645

AGE STOP PICA (YEARS) (Q D4)

	Cases	Controls
No.	14	8
Mean	4	3
Std. Dev.	1.97	1.13
Median	4	3

Statistical Test : Kruskal-Wallis
"p" value .4827

FREQUENCY PICA (Q D5)

		Cases	Controls
Everyday	No.	8	4
	%	42	40
Every 2-3 days	No.	3	1
	%	16	10
Occasionally	No.	6	5
	%	32	50
Don't know	No.	2	0
	%	10	0

Statistical Test : L.R. Chi-Square
"p" value .7706

FINGER SUCKING/NAIL BITING (Q D6)

		Cases	Controls
Frequently sucks fingers	No.	6	4
	%	23	11
Frequently bites nails	No.	2	4
	%	8	11
Frequently sucks fingers & bites nails	No.	3	0
	%	11	0
Does not suck fingers or bite nails	No.	15	27
	%	58	77

Statistical Test : L.R. Chi-Square
"p" value .1317

MOUTHING ACTIVITY (Q D7)

		Cases	Controls
Frequently mouthes on hands and objects	No.	14	2
	%	54	6
Does not mouth on hands and objects	No.	11	30
	%	42	94
Don't know	No.	1	0
	%	4	0

Statistical Test : L.R. Chi-Square
"p" value .0001

DIRTY HANDS (Q D8)

		Cases	Controls
Dirty hands frequently	No	13	10
	%	68	36
Dirty hands infrequently	No	6	18
	%	32	64

Statistical Test : Fishers Exact
"p" value .0392

HAND WASHING (Q D8)

		Cases	Controls
Washes hands on own	No.	8	10
	%	31	28
Does not wash hands on own	No.	3	8
	%	11	22
Not sure	No.	14	18
	%	54	50

Statistical Test : L.R Chi-Square
"p" value .6047

WASHTIME (Q D9)

		Cases	Controls
Washes hands meal-times only	No.	8	14
	%	38	42
Washes hands when dirty only	No.	2	2
	%	9	6
Washes hands on various occasions	No.	11	17
	%	52	51

Statistical Test : L.R. Chi-Square
"p" value .8656

PLAY-SITES (Q D10)

		Cases	Controls
Inside	No.	6	18
	%	22	50
Outside	No.	21	18
	%	78	50

Statistical Test : Fishers Exact Test
"p" value .0360

OTHER HOMES (Q D11)

		Cases	Controls
Yes	No.	15	15
	%	56	42
No	No.	10	21
	%	37	58
Don't know	No.	2	0
	%	7	0

Statistical Test : L.R. Chi-Square
"p" value .1298

PLAY STREET (Q D12)

		Cases	Controls
Yes	No.	17	14
	%	63	39
No	No.	9	22
	%	33	61
Don't Know	No.	1	0
	%	4	0

Statistical Test : L.R. Chi-Square
"p" value .0816

CRECHE (Q D13)

		Cases	Controls
Yes	No.	11	19
	%	41	53
No	No.	16	17
	%	59	47

Statistical Test : Fishers Exact Test
"p" value .4461

CARETAKER (Q D14)

		Cases	Controls
Mother	No.	16	29
	%	61	81
Father	No.	1	0
	%	4	0
Aunt	No.	3	1
	%	11	3
Sister	No.	1	1
	%	4	3
Grandmother	No.	3	3
	%	11	8
Grandfather	No.	2	0
	%	8	0
Cousin	No.	0	1
	%	0	3

Statistical Test : L.R. Chi-Square
"p" value .5236

BOOKS (Q D16)

		Cases	Controls
Yes	No.	10	31
	%	46	86
No	No.	12	5
	%	54	14

Statistical Test : Fishers Exact Test
"p" value .0023

TOYS (Q D16)

		Cases	Controls
Yes	No.	18	29
	%	69	83
No	No.	8	6
	%	31	17

Statistical Test : Fishers Exact Test
"p" value .02346

PLAY TOYS (Q D17)

		Cases	Controls
Yes	No.	9	19
	%	38	59
No	No.	15	13
	%	62	41

Statistical Test : Fishers Exact Test
"p" value .1765

PLAY (Q D18)

		Cases	Controls
Plays mainly alone	No.	5	5
	%	21	14
Plays mainly with others	No.	16	26
	%	67	72
Plays alone and with others	No.	3	5
	%	12	14

Statistical Test : L.R. Chi-Square
"p" value .7806

BEDTIME (Q D19)

	Cases	Controls
No.	25	35
Mean	7.69	8.24
Std. Dev.	1.4	.49
Median	8.00	8.00

Statistical Test : Kruskal-Wallis
"p" value .0057

OWN ROOM (Q D20)

		Cases	Controls
Yes	No.	0	5
	%	0	14
No	No.	26	30
	%	100	86

Statistical Test : Fishers Exact Test
"p" value .0656

OWN BED (Q D20)

		Cases	Controls
Yes	No.	12	26
	%	46	72
No	No.	14	10
	%	54	28

Statistical Test : Fishers Exact Test
"p" value .0634

OUTINGS (Q D21)

		Cases	Controls
Yes	No.	24	36
	%	96	100
No	No.	1	0
	%	4	0

Statistical Test : Fishers Exact Test
"p" value .4098

TRIPS (Q D22)

		Cases	Controls
Yes	No.	7	20
	%	28	57
No	No.	18	15
	%	72	43

Statistical Test : Fishers Exact Test
"p" value .0358

MUSEUM (Q D23)

		Cases	Controls
Visited museum with family	No.	3	8
	%	12	22
Visited museum with school	No.	17	11
	%	68	31
Visited museum	No.	4	11
	%	16	31
Did not visit museum	No.	1	6
	%	4	17

Statistical Test : L.R. Chi-Square
"p" value .0447

SCHOOLWORK (Q D24)

		Cases	Controls
Excellent	No.	0	8
	%	0	22
Good	No.	10	15
	%	37	42
Average	No.	7	9
	%	26	25
Poor	No.	9	4
	%	33	11
Don't know	No	1	0
	%	4	0

Statistical Test : L.R Chi-Square
"p" value .0269

GRADE ONE REPEAT (Q D25)

		Cases	Controls
Yes	No.	16	31
	%	59	86
No	No.	11	5
	%	41	14

Statistical Test : Fishers Exact Test
"p" value .0207

ACTIVE (Q D26)

		Cases	Controls
Active	No.	15	22
	%	62	63
Quiet	No.	5	9
	%	21	26
Both of the above	No.	2	3
	%	8	9
Don't know	No.	2	1
	%	8	3

Statistical Test : L.R Chi-Square
"p" value .8336

MORE ACTIVE THAN NORMAL (Q D27)

		Cases	Controls
Yes	No.	11	9
	%	41	26
No	No.	12	25
	%	44	71
Don't know	No.	4	1
	%	15	3

Statistical Test : L.R Chi-Square
"p" value .0702

PUNISHMENT (Q D28)

		Cases	Controls
Yes	No.	8	7
	%	36	20
No	No.	14	28
	%	64	80

Statistical Test : Fishers Exact Test
"p" value .2217

NUMBER PEOPLE (Q E1)

	Cases	Controls
No.	27	36
Mean	9	7
Std. Dev.	4.2	3.0
Median	9	6

Statistical Test : Kruskal-Wallis
"p" value .0198

NUMBER CHILDREN (Q E2)

	Cases	Controls
No.	27	36
Mean	4	3
Std. Dev.	2.3	1.7
Median	3	3

Statistical Test : Kruskal-Wallis
"p" value .3439

NUMBER SIBLINGS (Q E3)

	Cases	Controls
No.	27	35
Mean	3	2
Std. Dev.	1.53	1.42
Median	2	2

Statistical Test : Kruskal-Wallis
"p" value .4030

MOTHER (Q E4)

		Cases	Controls
Yes	No.	26	34
	%	96	94
No	No.	1	2
	%	4	6

Statistical Test : Fishers Exact Test
"p" value 1.0000

FATHER (Q E4)

		Cases	Controls
Yes	No.	17	29
	%	63	81
No	No.	10	7
	%	37	19

Statistical Test : Fishers Exact Test
"p" value .1555

FEMALE GUARDIAN (Q E4)

		Cases	Controls
Yes	No.	1	3
	%	4	8
No	No.	26	33
	%	96	92

Statistical Test : Fishers Exact Test
"p" value .6288

MALE GUARDIAN (Q E4)

		Cases	Controls
Yes	No.	2	2
	%	7	6
No	No.	25	34
	%	93	94

Statistical Test : Fishers Exact Test
"p" value 1.0000

HOBBIES (Q E6)

		Cases	Controls
Spray painting &/ or panel beating	No.	1	3
	%	4	9
Other motor repairs	No.	2	2
	%	8	6
Other mechanical work	No.	1	3
	%	4	9
Other hobbies	No.	6	4
	%	25	12
No hobbies	No.	14	20
	%	58	62

Statistical Test : L.R Chi-Square
"p" value .7371

RESIDENCE PRESENT ADDRESS (Q F1)

		Cases	Controls
Less than 6 months	No.	2	2
	%	7	6
6 months - 1 year	No.	0	2
	%	0	6
1-2 years	No.	0	2
	%	0	6
More than 2 years	No.	25	30
	%	93	83

Statistical Test : L.R Chi-Square
"p" value .5428

HOME OWNERSHIP (Q F2)

		Cases	Controls
Yes	No.	7	13
	%	26	36
No	No.	20	23
	%	74	64

Statistical Test : Fishers Exact Test
"p" value .4262

AGE HOME (YEARS) (Q F3)

	Cases	Controls
No.	26	33
Mean	65	51
Std. Dev.	21.73	28.28
Median	50	50

Statistical Test : Kruskal-Wallis
"p" value .0080

NUMBER ROOMS (Q F4)

	Cases	Controls
No.	27	36
Mean	3	4
Std. Dev.	1.41	1.48
Median	3	3

Statistical Test : Kruskal-Wallis
"p" value .3754

NUMBER SLEEPING ROOMS (Q F5)

	Cases	Controls
No.	27	36
Mean	2	3
Std. Dev.	1.12	1.11
Median	2	2

Statistical Test : Kruskal-Wallis
"p" value 0.8207

CANDLES (Q F6)

		Cases	Controls
Yes	No.	1	0
	%	4	0
No	No.	26	36
	%	96	100

Statistical Test : Fishers Exact Test
"p" value .4286

COAL (Q F6)

		Cases	Controls
Yes	No.	1	0
	%	4	0
No	No.	25	36
	%	97	100

Statistical Test : Fishers Exact Test
"p" value .4194

WOOD (Q F6)

		Cases	Controls
Yes	No.	2	1
	%	7	3
No	No.	25	35
	%	93	97

Statistical Test : Fishers Exact Test
"p" value .5717

GAS (Q F6)

		Cases	Controls
Yes	No.	8	8
	%	30	22
No	No.	19	28
	%	70	78

Statistical Test : Fishers Exact Test
"p" value .5665

PARAFFIN (Q F6)

		Cases	Controls
Yes	No.	5	1
	%	19	3
No	No.	22	35
	%	82	97

Statistical Test : Fishers Exact Test
"p" value .0758

ELECTRICITY (Q F6)

		Cases	Controls
Yes	No.	21	35
	%	78	97
No	No.	6	1
	%	22	3

Statistical Test : Fishers Exact Test
"p" value .0360

NUMBER SMOKERS (Q F7)

	Cases	Controls
No.	24	36
Mean	2	1
Std. Dev.	1.97	1.45
Median	2	1

Statistical Test : Kruskal-Wallis
"p" value .0416

RENOVATING ACTIVITIES (PAST YEAR) (Q F8)

		Cases	Controls
Painting only	No.	5	5
	%	21	16
Painting & scraping	No.	4	4
	%	17	12
Other renovating activities	No.	5	2
	%	21	6
No renovating	No.	10	21
	%	42	66

Statistical Test : L.R. Chi-Square
"p" value .2677

RENOVATING ACTIVITIES (PREVIOUS YEAR) (Q F8)

		Cases	Controls
Painting only	No.	3	5
	%	12	15
Painting & scraping	No.	6	4
	%	25	12
Other renovating activities	No.	3	0
	%	12	0
No renovating	No.	12	24
	%	50	73

Statistical Test : L.R. Chi-Square
"p" value .1128

CAR (Q F9)

		Cases	Controls
Yes	No.	13	25
	%	52	69
No	No.	12	11
	%	48	31

Statistical Test : Fishers Exact Test
"p" value .1896

GARAGE (Q F10)

		Cases	Controls
Street	No.	10	18
	%	100	78
Garage	No.	0	5
	%	0	22

Statistical Test : Fishers Exact Test
"p" value .2911

MOTOR REPAIRS (Q F11)

		Cases	Controls
Yes	No.	5	5
	%	28	18
Motor repairs other than body work	No.	2	3
	%	11	11
No	No.	11	20
	%	61	71

Statistical Test : L.R. Chi-Square
"p" value .7125

RENT (RANDS/MONTH) (Q G1)

	Cases	Controls
No.	19	21
Mean	47	69
Std. Dev.	25	37
Median	33	60

Statistical Test : Kruskal-Wallis
"p" value .0448

MOTHER'S SCHOOLING (YEARS COMPLETED) (Q G2)

	Cases	Controls
No.	26	36
Mean	6	7
Std. Dev.	1.94	1.76
Median	6	7

Statistical Test : Kruskal-Wallis
"p" value .0313

MOTHER'S INCOME (RANDS/MONTH) (Q G4)

	Cases	Controls
No.	8	16
Mean	307	371
Std. Dev.	116	313
Median	280	280

Statistical Test : Kruskal-Wallis
"p" value .8782

FATHER'S SCHOOLING (YEARS COMPLETED) (Q G5)

	Cases	Controls
No.	21	33
Mean	7	8
Std. Dev.	2.20	1.79
Median	7	8

Statistical Test : Kruskal-Wallis
"p" value .2249

FATHER'S INCOME (RANDS/MONTH) (Q G7)

	Cases	Controls
No.	19	30
Mean	592	842
Std. Dev.	427	871
Median	476	600

Statistical Test : Kruskal-Wallis
"p" value .1651

TOTAL FAMILY INCOME (RANDS/MONTH) (Q G8)

	Cases	Controls
No.	21	32
Mean	648	986
Std. Dev.	438	896
Median	580	750

Statistical Test : Kruskal-Wallis
"p" value .0819

NUMBER OF DEPENDANTS (Q G9)

	Cases	Controls
No.	22	27
Mean	5	5
Std. Dev.	1.21	1.09
Median	5	5

Statistical Test : Kruskal-Wallis
"p" value .5648

TOTAL FAMILY INCOME (RANDS/MONTH), RELATIVE TO NO. OF DEPENDANTS (Q G9)

	Cases	Controls
No.	16	25
Mean	152	252
Std. Dev.	99	192
Median	133	203

Statistical Test : Mann-Whitney
"p" value .0383

NUMBER DEPENDANTS UNDER TWELVE (Q G10)

	Cases	Controls
No.	22	29
Mean	2	2
Std. Dev.	.89	1.01
Median	2	3

Statistical Test : Kruskal-Wallis
"p" value .8098

SOCIAL CLASS

	Cases	Controls
Category 1	14	29
	56	91
Categories 2,3	11	3
	44	9

Statistical Test : Fishers Exact
"p" value .0044

OCCUPATIONAL DENSITY

	Cases	Controls
No.	24	33
Mean	189	125
Std. Dev.	106	48
Median	159	100

Statistical Test : Mann-Whitney
"p" value .0069

APPENDIX E 4a

INSIDE WALL PAINT LEAD CONCENTRATIONS (ppm)

	Cases	Controls
No.	14	13
Mean	10191	5503
Std. Dev.	23082	7827
Median	3293	1449

APPENDIX E 4b

INSIDE WINDOWSILL PAINT LEAD CONCENTRATIONS (ppm)

	Cases	Controls
No.	9	9
Mean	33946	33943
Std. Dev.	34104	38370
Median	19782	15973

APPENDIX E 4c

INSIDE WALL AND WINDOWSILL PAINT LEAD CONCENTRATIONS (ppm)

	Cases	Controls
No.	17	17
Mean	17791	17426
Std. Dev.	23414	26714
Median	7721	5442

APPENDIX E 4d

OUTSIDE WALL PAINT LEAD CONCENTRATIONS (ppm)

	Cases	Controls
No.	8	9
Mean	7408	10298
Std. Dev.	9825	17119
Median	2809	3872

APPENDIX E 4e

INSIDE AND OUTSIDE WALL AND WINDOWSILL PAINT LEAD CONCENTRATIONS (ppm)

	Cases	Controls
No.	19	19
Mean	13791	15120
Std. Dev.	15836	22068
Median	7287	5442

APPENDIX E 4f

INSIDE WALL PAINT LEAD CONCENTRATIONS (ppm) (ALL CHILDREN)

No.	27
Mean	7934
Std. Dev.	17331
Median	2104

APPENDIX E 4g

INSIDE WINDOWSILL PAINT LEAD CONCENTRATIONS (ppm) (ALL CHILDREN)

No.	18
Mean	33944
Std. Dev.	35216
Median	16656

APPENDIX E 4h

INSIDE WALL AND WINDOWSILL PAINT LEAD CONCENTRATIONS (ppm) (ALL CHILDREN)

No.	34
Mean	17608
Std. Dev.	24735
Median	6581

APPENDIX E 4i

OUTSIDE WALL PAINT LEAD CONCENTRATIONS (ppm) (ALL CHILDREN)

No.	17
Mean	8938
Std. Dev.	13819
Median	3744

APPENDIX E 4j

INSIDE AND OUTSIDE WALL AND WINDOWSILL PAINT LEAD CONCENTRATIONS (ppm) (ALL CHILDREN)

No.	38
Mean	14455
Std. Dev.	18957
Median	6500

APPENDIX E 4k

LEAD CONCENTRATION (%) OF PAINT SAMPLES

	Cases	Controls
1 or more samples $\geq$ 1%		
No.	12	9
%	44	26
Stat. Test	Fishers Exact	
"p" value	.1766	

APPENDIX E 5a

HOUSE DUST LEAD CONCENTRATIONS (ppm)

	Cases	Controls
No.	20	28
Mean	2253	745
Std. Dev.	6569	777
Median	535	550

APPENDIX E 5b

HOUSE DUST LEAD LEVELS (ug/2sq.ft)
(ALL CHILDREN)

	Inside House (Bedroom, Main Living Room)	Outside House (Front Veranda)
No.	48	15
Mean	776	3967
Std. Dev.	3170	11593
Median	30	585

APPENDIX E 5c

HOUSE DUST LEAD CONCENTRATIONS (ppm)
(ALL CHILDREN)

	Inside House (Bedroom, Main Living Room)	Outside House (Front Veranda)
No.	48	16
Mean	1374	3004
Std. Dev.	4284	5908
Median	542	749

APPENDIX E 6a

STREET DUST LEAD LEVELS (ug/2sq.ft)

	Cases	Controls
No.	21	21
Mean	4573	11204
Std. Dev.	7788	21491
Median	1930	4140

APPENDIX E 6b

STREET DUST LEAD CONCENTRATIONS (ppm)

	Cases	Controls
No.	21	21
Mean	2699	1993
Std. Dev.	5902	2250
Median	1005	1086

APPENDIX E 7a

PERCENT EXPECTED WEIGHT FOR AGE

	Cases	Controls
No.	28	41
Mean	92	96
Std. Dev.	14	14
Stat. Test	Median	
"p" value	.3187	

APPENDIX E 7b

PERCENT EXPECTED HEIGHT FOR AGE

	Cases	Controls
No.	28	41
Median	97	99
Std. Dev.	5	3
Stat Test	Median	
"p" value	.0645	

APPENDIX E 7c

PERCENT EXPECTED WEIGHT FOR HEIGHT

	Cases	Controls
No.	28	41
Mean	97	97
Std. Dev.	8	11
Stat. Test	Median	
"p" Value	.2835	

APPENDIX E 7d

FREQUENCY <90 PERCENT EXPECTED HEIGHT FOR AGE

	Cases	Controls
No.	5	4
%	16	9
Stat. Test	Fishers Exact.	
"p" Value	.4770	

APPENDIX E 7e

FREQUENCY <80 PERCENT EXPECTED WEIGHT FOR AGE

	Cases	Controls
No.	8	7
%	25	16
Stat. Test	Fishers Exact	
"p" Value	.3850	