

**BLOOD LEAD LEVELS IN FIRST GRADE SOUTH AFRICAN  
CHILDREN – A GEOGRAPHIC & TEMPORAL ANALYSIS**

**Angela Mathee**

**South African Medical Research Council**

**&**

**University of the Witwatersrand**

**June 2005**

## I - DECLARATION

I, Angela Mathee, declare that this thesis is my own work. It is being submitted for the degree of Doctor of Philosophy at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other University.

---

SIGNATURE

on the \_\_\_\_\_ day of \_\_\_\_\_, 2004.

## **II - DEDICATION**

This work is dedicated to my darling daughter Kiera Jordan – she and millions of other young South African children have an as yet unmet constitutional right to be protected from exposure to lead in the environment, and the associated consequences for their health, school performance and social well-being.

### III - PUBLICATIONS AND PRESENTATIONS THAT HAVE ARISEN FROM THIS THESIS TO DATE

**Mathee A**, Röllin H, von Schirnding Y, Levin J, Naik I. Reductions in Blood Lead Levels among School Children following the Introduction of Unleaded Petrol in South Africa. *Environmental Research*, 2006, 100(3): 319-22.

Montgomery M, **Mathee A**. A preliminary study of residential paint lead concentrations in an African city; Johannesburg. *Environmental Research*, 2005, 98(3): 279-83.

**Mathee A**, von Schirnding Y, Montgomery M, Röllin H. Lead poisoning in South African children: the hazard is at home. *Reviews in Environmental Health*, 2004, 19(3-4): 347-361.

von Schirnding YER, **Mathee A**, Kibel M, Robertson P, Strauss N, Blignaut R. A study of pediatric blood lead levels in a lead mining area in South Africa. *Environmental Research*, 2003, 93(3): 259-263.

**Mathee A**, Röllin H, Ditlopo NN, Theodorou P. Childhood lead exposure in South Africa. *South African Medical Journal*, 2003, 93(5): 313.

Harper CC, **Mathee A**, von Schirnding Y, De Rosa CT, Falk H. The health impact of environmental pollutants: a special focus on lead exposure in South Africa. *International Journal of Hygiene and Environmental Health*. 2003, 206(4-5):315-22.

**Mathee A**, von Schirnding YER, Levine J, Ismail A, Huntley R, Cantrell A. A survey of blood lead levels amongst young Johannesburg school children, *Environmental Research* 2002 90(3):181-4.

von Schirnding Y, **Mathee A**, Robertson P, Strauss N, Kibel M. A study of the distribution of blood lead levels in school children in selected Cape Peninsula suburbs subsequent to reductions in petrol lead. *South African Medical Journal*, 2001, 91(10): 870 -873.

## **REPORTS**

**Mathee A** & von Schirnding YER. The risk of environmental lead exposure amongst South African children. A Medical Research Council/World Health Organization Report prepared for the World Summit on Sustainable Development - 2002.

## IV - ABSTRACT

Lead is a toxic heavy metal that has been extensively used in modern society, causing widespread environmental contamination, even in isolated parts of the world. There is now overwhelming evidence associating lead exposure with wide-ranging health effects, including reductions in intelligence scores, hyperactivity, shortened concentration spans, poor school performance, violent/aggressive behaviour, hearing loss, delayed onset of puberty, anaemia, and in severe cases, coma and death. In recent years consensus has been reached in respect of the absence of a threshold of safety for key health effects associated with lead exposure, and the permanent and irreversible nature of many of the health and social consequences of exposure to lead.

The public health problem of environmental lead exposure has been widely investigated in developed countries such as the United States of America where, since the 1970s, policies and interventions have been followed by significant reductions in blood lead levels amongst children. In developing countries, and in African countries in particular, there is a relative dearth of information on the sources, mechanisms of exposure and blood lead distributions in children, and little action has been taken to protect children against lead poisoning.

This study was undertaken to determine the current distribution of blood lead concentrations, and associated risk factors, amongst selected groups of first grade school children in the South African urban settings of Cape Town, Johannesburg

and Kimberley, a lead mining town (Aggeneys) and two rural towns in the Northern Cape province. A further objective of the study was to compare blood lead distributions determined in the current study with the findings of similar studies undertaken prior to the introduction in 1996 of unleaded petrol in South Africa.

The results show that over the past decade, blood lead concentrations amongst first grade school children have declined considerably, but that large proportions of children, especially those living or attending school in impoverished areas, continue to have intolerably high blood lead concentrations, within a range that puts them at risk of detrimental health and social outcomes. The major sources of exposure to lead in the samples studied were leaded petrol, lead-based paint used to decorate homes and schools, lead solder used in “cottage industries” and other home-based lead-related activities, as well as the transfer of lead particles from lead-related work settings into homes. Recommendations for policy and relevant interventions for the South African context are discussed.

## **V - ACKNOWLEDGEMENTS**

Many friends and colleagues, directly or indirectly, have contributed to my decision to undertake and complete this study. During the early 1990s Yasmin von Schirnding introduced me to the problem of lead poisoning in South African children and to the field of children's environmental health. For this, and for her friendship, I am enormously appreciative.

I wish to thank Professor William Pick, Professor Emeritus at the School of Public Health of the University of the Witwatersrand, for agreeing to serve as a supervisor of this project, and for his insightful and thought-provoking comments on the various drafts of this work.

I am enormously grateful to Jonathan Levin for his patient guidance and assistance with the statistical analyses.

Ina Naik and Penny Theodorou of the National Institute for Occupational Health, and Francois Wewers (Peninsula Technikon) conducted the analyses of blood, soil, dust, paint and water samples. They are thanked most sincerely for their assistance in this regard.

I would like to warmly thank the many brave and beautiful children who participated in the study, as well as their parents, principals, teachers and school governing bodies for their support. The support and assistance of the Gauteng (Member of

Executive Committee (MEC) Ignatius Jacobs and John van Rooyen), Northern Cape and Western Cape Departments of Education is gratefully acknowledged.

I am indebted to my colleagues and friends at the South African Medical Research Council: Halina Röllin, Brendon Barnes, Kebitsamang (KB) Moiloa, Mirriam Mogotsi, Rochelle Spadoni, Rajeshree Naidoo, Liz Thomas, Pam Cerff and Romilla Maharaj for their support and assistance in the course of this work, and in general. Thanks also to Mary Montgomery for her work on the follow-up investigations of Johannesburg subjects with the highest blood lead concentrations and the city-wide study of residential paint lead concentrations in Johannesburg.

Without generous financial assistance from the South African Medical Research Council, the United States Environmental Protection Agency, the Northern Cape Department of Health and Engen Petroleum Limited, completion of the study would not have been possible.

A final note of thanks goes to members of my family, especially my mother and Aunt Sally, whose ongoing support and love is my motivation. My immediate family - Kiera and Colin – are the nucleus of my life - you are wonderful and I love you.

## VI - LIST OF CONTENTS

I - DECLARATION .....	2
II - DEDICATION .....	3
III - PUBLICATIONS AND PRESENTATIONS THAT HAVE ARISEN FROM THIS THESIS TO DATE .....	4
IV - ABSTRACT.....	6
V - ACKNOWLEDGEMENTS.....	8
VI - LIST OF CONTENTS .....	10
VII - LIST OF FIGURES .....	15
VIII - LIST OF TABLES .....	16
IX - LIST OF ACRONYMS & ABBREVIATIONS.....	18
CHAPTER 1 - INTRODUCTION .....	19
1.1    PROBLEM STATEMENT .....	19
1.2    PURPOSE OF THE STUDY.....	21
1.3    ORGANIZATION OF THE THESIS.....	22
1.4    SOURCES OF EXPOSURE TO LEAD .....	23
1.4.1 <i>Sources of Lead Exposure in Antiquity</i> .....	24
1.4.2 <i>Contemporary Sources of Lead</i> .....	25
1.4.3 <i>Sources of Exposure to Lead in South Africa</i> .....	26
1.5    PATHWAYS & ROUTES OF LEAD EXPOSURE IN CHILDREN .....	32
1.6    HIGH RISK GROUPS.....	32
1.6.1 <i>Children</i> .....	32

1.6.2	<i>Foetal Exposure</i> .....	33
1.6.3	<i>The Role of Poverty</i> .....	33
1.6.4	<i>Para-occupational Exposure</i> .....	34
1.7	HEALTH & SOCIAL OUTCOMES OF CHILDHOOD LEAD EXPOSURE .	34
1.7.1	<i>Death from Lead Poisoning</i> .....	35
1.7.2	<i>Haematological Effects</i> .....	35
1.7.3	<i>Neurological Effects</i> .....	36
1.7.4	<i>Hearing Loss</i> .....	39
1.7.5	<i>Delayed Onset of Puberty</i> .....	39
1.8	THE EPIDEMIOLOGY OF CHILDHOOD LEAD EXPOSURE IN SOUTH AFRICA (1970s & 1990s) .....	40
1.8.1	<i>Severe Lead Poisoning</i> .....	40
1.8.2	<i>Newborns</i> .....	40
1.8.3	<i>Pre-school Children</i> .....	42
1.8.4	<i>Young School Children</i> .....	42
1.8.5	<i>Blood Lead Levels in a Lead Mining Town (Aggeneys)</i> .....	46
1.9	MOTIVATION FOR THE STUDY .....	48
1.10	STUDY OBJECTIVES.....	49
	<b>CHAPTER 2 - METHODS</b> .....	<b>50</b>
2.1	STUDY DESIGN .....	50
2.2	STUDY AREAS AND POPULATION .....	50
2.2.1	<i>Study Areas</i> .....	50
2.2.2	<i>Study Sample</i> .....	52
2.3	MEASUREMENT OF BLOOD LEAD CONCENTRATIONS.....	53

2.4	ADMINISTRATION OF QUESTIONNAIRES.....	54
2.5	MEASUREMENT OF ENVIRONMENTAL LEAD CONCENTRATIONS ...	55
2.6	HOME ASSESSMENTS (JOHANNESBURG) .....	55
2.7	RESIDENTIAL PAINT SAMPLING (JOHANNESBURG).....	56
2.8	LEAD CONCENTRATIONS IN PAINTED CHILDREN'S TOYS .....	57
2.9	ETHICAL CONSIDERATIONS AND APPROVALS.....	58
2.10	DATA PROCESSING AND ANALYSIS .....	59
2.11	RESPONSE RATE .....	61
<b>CHAPTER 3 - RESULTS .....</b>		<b>63</b>
3.1	DESCRIPTION OF THE STUDY SAMPLE.....	63
3.1.1	<i>Rural Northern Cape (Pella &amp; Onseepkans)</i> .....	63
3.1.2	<i>Aggeneys (Lead Mining Town)</i> .....	68
3.1.3	<i>Kimberley</i> .....	69
3.1.4	<i>Cape Town &amp; Johannesburg</i> .....	71
3.2	BLOOD LEAD DISTRIBUTIONS.....	72
3.2.1	<i>The Total Sample</i> .....	72
3.2.2	<i>Rural Northern Cape (Onseepkans &amp; Pella)</i> .....	73
3.2.3	<i>The Lead Mining Town of Aggeneys</i> .....	75
3.2.4	<i>Kimberley</i> .....	76
3.2.5	<i>Cape Town</i> .....	78
3.2.6	<i>Johannesburg</i> .....	81
3.3	RISK FACTORS FOR ELEVATED BLOOD LEAD LEVELS .....	84
3.3.1	<i>Rural Northern Cape (Pella &amp; Onseepkans)</i> .....	84
3.3.2	<i>Kimberley</i> .....	86

3.3.3	<i>Cape Town</i> .....	88
3.3.4	<i>Johannesburg</i> .....	90
3.3.5	<i>The Total Sample</i> .....	91
3.4	GEOGRAPHICAL COMPARISON OF BLOOD LEAD DISTRIBUTIONS IN SELECTED STUDY SITES .....	94
3.5	TEMPORAL ANALYSES .....	97
3.5.1	<i>Aggeneys &amp; Pella</i> .....	97
3.5.2	<i>Cape Town</i> .....	99
3.5.3	<i>Johannesburg</i> .....	102
3.6	SUPPLEMENTARY INVESTIGATIONS .....	104
3.6.1	<i>School Environmental Sampling</i> .....	105
3.6.2	<i>Case Study of Subject with Elevated Blood Lead Concentration</i> ....	107
3.6.3	<i>Home Investigations - Johannesburg Children With Elevated Blood Lead Levels</i> .....	108
3.6.4	<i>Study of Residential Paint Lead Concentrations - Johannesburg</i> ...	110
3.5.5	<i>Analysis Of Lead Content of Off-The-Shelf Paint Samples</i> .....	112
3.5.6	<i>Lead Content of Paint on Children's Toys and Coloured Pencils</i> ....	113
<b>CHAPTER 4: DISCUSSION</b> .....		<b>115</b>
4.1	GEOGRAPHICAL VARIATIONS IN BLOOD LEAD DISTRIBUTIONS ....	115
4.1.1	<i>Rural Areas</i> .....	115
4.1.2	<i>Lead Mining Town</i> .....	116
4.1.3	<i>Urban Areas</i> .....	119
4.2	TEMPORAL CHANGES IN BLOOD LEAD DISTRIBUTIONS .....	121
4.3	RISK FACTORS FOR ELEVATED BLOOD LEAD CONCENTRATIONS	124

4.3.1	<i>Age</i> .....	125
4.3.2	<i>Socio-economic Status/Population Group</i> .....	125
4.3.3	<i>Housing Conditions</i> .....	126
4.3.3	<i>Lead in Petrol</i> .....	127
4.3.4	<i>Lead in Paint</i> .....	127
4.3.5	<i>Plumbing</i> .....	131
4.3.6	<i>Informal Sector Exposure to Lead</i> .....	131
4.4	STRENGTHS AND WEAKNESSES OF THE THESIS .....	132
4.4.1	<i>Weaknesses</i> .....	132
4.4.2	<i>Strengths</i> .....	136
4.5	OBSTACLES TO THE PREVENTION OF LEAD EXPOSURE & POISONING .....	136
4.6	ETHICAL OBLIGATIONS .....	139
4.7	IMPLEMENTATION OF POLICIES AND PROGRAMMES .....	140
	<b>CHAPTER 5 - CONCLUSIONS</b> .....	<b>141</b>
5.1	SUMMARY OF THE OBJECTIVES AND FINDINGS .....	141
5.2	ORIGINALITY/NEW INSIGHTS .....	142
5.3	POLICY IMPLICATIONS .....	143
5.4	FUTURE RESEARCH NEEDS .....	143
5.5	THE WAY FORWARD .....	144
	<b>LIST OF REFERENCES</b> .....	<b>147</b>
	<b>ANNEXURE 1 - THE QUESTIONNAIRE</b> .....	<b>164</b>
	<b>ANNEXURE 2 - SUBJECT INFORMATION SHEET</b> .....	<b>183</b>
	<b>ANNEXURE 3 - CONSENT FORM</b> .....	<b>184</b>

## VII - LIST OF FIGURES

Figure 1. MONTHLY AIR LEAD LEVELS IN JOHANNESBURG.....	31
Figure 2. BLOOD LEAD DISTRIBUTION (TOTAL SAMPLE).....	73
Figure 3. BLOOD LEAD DISTRIBUTION IN PELLA & ONSEEPKANS .....	74
Figure 4. BLOOD LEAD DISTRIBUTION IN PELLA VERSUS ONSEEPKANS .....	75
Figure 5. BLOOD LEAD DISTRIBUTION IN AGGENEYS .....	76
Figure 6. BLOOD LEAD DISTRIBUTION IN KIMBERLEY .....	77
Figure 7. BLOOD LEAD DISTRIBUTION IN CAPE TOWN.....	78
Figure 8. BLOOD LEAD LEVEL BY SCHOOL: CAPE TOWN, 2002.....	81
Figure 9. BLOOD LEAD DISTRIBUTION IN JOHANNESBURG, 2002.....	82
Figure 10. BOX PLOTS FOR BLOOD LEAD DISTRIBUTION BY STUDY SITE.....	94
Figure 11. BLOOD LEAD DISTRIBUTIONS IN URBAN VERSUS RURAL SITES .....	96
Figure 12. BLOOD LEAD DISTRIBUTION IN AGGENEYS – 1991 VERSUS 2003 .....	97
Figure 13. BLOOD LEAD DISTRIUBTION IN PELLA – 1991 VERSUS 2003.....	98
Figure 14. BLOOD LEAD DISTRIBUTION IN WOODSTOCK – 1991 VERSUS 2002.....	99

## VIII - LIST OF TABLES

Table 1. THE STUDY SAMPLE.....	52
Table 2. RESPONSE RATES BY STUDY AREA.....	62
Table 3. PROFILES OF THE STUDY SAMPLES (BY SITE).....	64
Table 4. HOUSEHOLD INCOME DISTRIBUTION IN THE NORTHERN CAPE .....	65
Table 5. REPORTED ILL HEALTH CONDITIONS BY STUDY SITE .....	66
Table 6. PREVALENCE OF PICA AND MOUTHING BEHAVIOUR .....	67
Table 7. LEVELS OF AWARENESS OF LEAD HAZARDS .....	68
Table 8. BLOOD LEAD LEVELS PELLA & ONSEEPKANS ( $\mu\text{g}/\text{dl}$ ).....	74
Table 9. BLOOD LEAD LEVELS BY SCHOOL – KIMBERLEY ( $\mu\text{g}/\text{dl}$ ).....	77
Table 10. MEAN BLOOD LEAD LEVELS BY AREA - CAPE TOWN ( $\mu\text{g}/\text{dl}$ ) .....	79
Table 11. BLOOD LEAD LEVELS BY SCHOOL (CAPE TOWN) ( $\mu\text{g}/\text{dl}$ ).....	80
Table 12. SUBJECTS WITH ELEVATED BLOOD LEAD LEVELS BY AREA – JOHANNESBURG .....	82
Table 13. BLOOD LEAD CONCENTRATIONS BY SCHOOL - JOHANNESBURG .....	83
Table 14. RISK FACTORS FOR ELEVATED BLOOD LEAD LEVELS BY STUDY SITE	85
Table 15. ANALYSES OF THE ADJUSTED (FINAL LINEAR REGRESSION MODELS) ASSOCIATIONS BETWEEN RISK FACTORS AND ELEVATED BLOOD LEAD LEVELS: KIMBERLEY, CAPE TOWN, JOHANNESBURG AND THE TOTAL SAMPLE.....	93
Table 16. COMPARISON OF BLOOD LEAD LEVELS IN KIMBERLEY, CAPE TOWN & JOHANNESBURG ( $\mu\text{g}/\text{dl}$ ).....	95
Table 17. BLOOD LEAD LEVELS BY TOWN – RURAL NORTHERN CAPE ( $\mu\text{g}/\text{dl}$ ).....	96
Table 18. COMPARISON OF BLOOD LEAD LEVELS IN URBAN & RURAL SITES ( $\mu\text{g}/\text{dl}$ ).....	97

<b>Table 19. COMPARISON OF BLOOD LEAD LEVELS IN CAPE TOWN – 1991 &amp; 2002</b>	<b>101</b>
<b>Table 20. MEAN SCHOOL BLOOD LEAD LEVELS (<math>\mu\text{g}/\text{dl}</math>) (CAPE TOWN 1991 &amp; 2002)</b>	<b>102</b>
<b>Table 21. COMPARISON OF BLOOD LEAD LEVELS BY SCHOOL IN JOHANNESBURG (1995 &amp; 2002)</b>	<b>103</b>
<b>Table 22. LEAD CONCENTRATIONS IN SCHOOL WATER SAMPLES</b>	<b>105</b>
<b>Table 23. LEAD CONCENTRATIONS IN SCHOOL SOIL SAMPLES</b>	<b>106</b>
<b>Table 24. LEAD CONCENTRATIONS IN SCHOOL PAINT SAMPLES</b>	<b>106</b>
<b>Table 25. LEAD CONCENTRATIONS IN HOME ENVIRONMENTAL SAMPLES</b>	<b>109</b>
<b>Table 26. PREVALENCE OF RISK FACTORS FOR ELEVATED BLOOD LEAD LEVELS*</b>	<b>110</b>
<b>Table 27. PAINT LEAD CONCENTRATIONS IN GREATER JOHANNESBURG (<math>\mu\text{g}/\text{g}</math>)</b>	<b>112</b>
<b>Table 28. RESULTS OF LEAD CONTENT ANALYSIS OF PAINTED, WOODEN CHILDREN’S TOYS</b>	<b>113</b>
<b>Table 29. LEAD CONCENTRATIONS IN PAINT COATING COLOURED PENCILS</b>	<b>114</b>

## IX - LIST OF ACRONYMS & ABBREVIATIONS

<b>ALA</b>	<b><math>\delta</math>-aminolevulinic acid</b>
<b>ALAD</b>	<b><math>\delta</math>-aminolevulinic acid dehydratase</b>
<b>BTT</b>	<b>Birth to Twenty</b>
<b>CDC</b>	<b>Centers for Disease Control</b>
<b>EDTA</b>	<b>Ethylenediaminetetraacetic acid</b>
<b>EPA</b>	<b>Environmental Protection Agency (USA)</b>
<b>HUD</b>	<b>Department of Housing &amp; Urban Development (USA)</b>
<b>HPCSA</b>	<b>Health Professions Council of South Africa</b>
<b>LOAEL</b>	<b>Lowest Observed Adverse Effect Level</b>
<b>MRC</b>	<b>Medical Research Council</b>
<b><math>\mu\text{g}/\text{dl}</math></b>	<b>Micrograms/decilitre</b>
<b><math>\mu\text{g}/\text{g}</math></b>	<b>Micrograms/gram</b>
<b>NIOH</b>	<b>National Institute of Occupational Health</b>
<b>ppm</b>	<b>parts per million</b>
<b>SAPMA</b>	<b>South African Paint Manufacturers' Association</b>
<b>USA</b>	<b>United States of America</b>
<b>WHO</b>	<b>World Health Organization</b>
<b>ZPP</b>	<b>zinc protoporphyrin</b>

## CHAPTER 1 - INTRODUCTION

### 1.1 PROBLEM STATEMENT

Lead is a heavy metal with many useful properties, including malleability and anti-corrosiveness, which has resulted in its mining and use for millennia. During the past century in particular, the anthropogenic use of lead escalated dramatically, for example as additives in petrol and paint, leading to widespread environmental contamination and elevated lead exposure amongst children around the globe. Childhood lead exposure, and the associated health and social effects, has been described as a public health “catastrophe” (Landrigan 2002), and currently constitutes a priority environmental health concern in many countries.

Lead interferes with neuronal migration, cell proliferation and synapse formation during critical periods of early vulnerability (Landrigan 2002). Young children, especially those aged two to four years, and the foetus, have been established to be particularly vulnerable to lead-related health effects, including reductions in intelligence quotients, behavioural abnormalities such as hyperactivity and shortened concentration spans, hearing loss and poor school performance (Bellinger et al 1987, Canfield et al 2003, Needleman & Bellinger 1991, Needleman et al 1996, Schwarz & Otto 1987, Schwarz & Otto 1991). Lead related health effects such as the delayed onset of puberty, hypertension and infertility, have also been established in adolescence and adult life (Wu et al 2003). There is now widespread consensus among scientists in relation to the absence of a threshold of

safety for lead exposure, and the irreversible nature of many of the health and social effects associated with lead exposure (Rogan & Ware 2003, Needleman & Bellinger 1991).

While an extensive body of knowledge exists in respect of childhood lead exposure in developed countries, there is a dearth of data regarding lead exposure and the distribution of childhood blood lead levels in developing countries in general, and in African children in particular (Nriagu et al 1996, Tong et al 2000). Studies of blood lead concentrations in first grade school children undertaken in the Cape Peninsula during the mid-1980s and early 1990s indicated that alarmingly high proportions of young South Africans were exposed to environmental lead (von Schirnding et al 2001, von Schirnding et al 1991a). Similarly, a study undertaken in Johannesburg in 1995, showed that the mean blood lead concentration amongst first grade school children was 12 µg/dl, and that 78% of subjects had blood lead concentrations equaling or exceeding the international action level of 10 µg/dl (Mathee et al 2002). A study of cord blood lead concentrations undertaken in Soweto/Johannesburg during 1990 revealed that exposure to lead occurred during the foetal stage, with cord blood lead measurements ranging up to 20 µg/dl (Mathee et al 1996).

Lead exposure reduction measures have been implemented in many developed countries. The removal of lead from petrol in the United States of America (USA) in particular, was associated with a 90% decline in mean blood lead levels amongst children (Pirkle et al 1994). Other measures implemented include the development of blood lead standards for children, public awareness campaigns, environmental

and personal hygiene interventions, secondary prevention measures, banning the use of lead in paint for residential use, and decontamination of housing and schools.

Since no national blood lead surveillance programme is in place, and no nationwide screening study has been undertaken, there is a paucity of information on children's current blood lead distributions across South Africa. Similarly, little is known about current risk factors for elevated blood lead levels. There is also limited awareness of the problem of childhood lead exposure in South Africa in the general public, as well as within important sectors such as Health, Education, Social Development, Minerals & Energy, Trade & Industry, Labour and Housing, and consequently limited action has been taken to reduce childhood lead exposure in the country.

Unleaded petrol was introduced in South Africa in 1996, with the potential to significantly reduce children's exposure to environmental lead. At the time of the study (2002/3) leaded petrol constituted around 30% of the petrol market share in the country. The impact of the introduction of unleaded petrol in South Africa on childhood blood lead distributions has not yet been evaluated.

## **1.2 PURPOSE OF THE STUDY**

The purpose of this study was to determine the current distribution of blood lead levels, and associated risk factors, in first grade school children in selected suburbs in the cities of Cape Town, Johannesburg and Kimberley, as well as in

three smaller rural towns of the Northern Cape, namely Aggeneys (a lead mining town), Pella and Onseepkans. A second objective of the study was to compare current blood lead concentrations in Cape Town, Johannesburg, Aggeneys and Pella, with those measured in similar studies undertaken prior to the introduction of unleaded petrol.

### **1.3 ORGANIZATION OF THE THESIS**

In the introductory chapter (Chapter One), information will be given on the key sources and mechanisms of childhood exposure to lead, with particular emphasis on the South African situation. Through a review of the scientific literature, the health and social effects of lead exposure, especially during childhood, will be discussed. The chapter will conclude with an outline of published information on blood lead distributions and associated risk factors for elevated blood lead levels among South African children.

A chapter on Methods (Chapter Two) will describe the study design, study population, study sites, measurement instruments, data collection, the approach to laboratory and data analysis and ethical aspects of the study.

Chapter Three on Results will describe the key findings of the current study, and will include profiles of the study populations, data on blood lead distributions and associated risk factors in Cape Town, Johannesburg, Kimberley, Aggeneys and Pella/Onseepkans.

For Cape Town, Johannesburg, Aggeneys and Pella, blood lead distributions in the current study will be compared with those determined in similar studies undertaken prior to the introduction of unleaded petrol.

The results of supplementary investigations into the sources and risk factors in the 20 study subjects with the highest blood lead concentrations in the Johannesburg phase of the study, a case study of the follow-up home investigation and hospitalization of the subject with the highest blood lead concentration (more than four times as high as the internationally accepted action level of 10 µg/dl), results of analyses of the lead content of paint samples collected from homes within randomly selected suburbs across the City of Johannesburg, analyses of the lead concentrations in paint samples obtained from paint stores in Johannesburg and Cape Town and lead content analyses of painted children's toys will be included in this chapter.

In the final chapter (Chapter Four), headed "Discussion and Conclusions", the key study findings will be discussed in relation to policy options and recommendations for action. The study limitations are also outlined.

#### **1.4 SOURCES OF EXPOSURE TO LEAD**

Lead is a non-biodegradable, malleable, non-corrosive heavy metal, which occurs naturally in the earth's crust. Although both natural and anthropogenic processes are responsible for the distribution of lead through the environment, anthropogenic processes are predominant in this regard (Budd et al 2004). Its widespread use,

especially during the past century, has resulted in lead becoming a ubiquitous environmental contaminant, particularly in urban areas.

Long-range transportation has resulted in elevated lead concentrations in settings as remote as Greenland, as well as the Arctic and Antarctic circles (Piomelli et al 1980, Poole et al 1980). The natural background level of lead in blood is estimated to be 0.016 µg/dl. The current, internationally accepted, action level for lead in blood (10 µg/dl) exceeds the background level by a factor of 600. Even remote populations have been shown to have blood lead levels 50 times as high as the background level (Tong et al 2000).

#### **1.4.1 Sources of Lead Exposure in Antiquity**

Lead has been known, mined and used for thousands of years. Properties such as resistance to corrosion, density, low fusion point and ductility, lead to widespread mining and use of the metal in the ancient cultures of Greece, Rome, Egypt, Babylon, Assyria, China and India, as well as during the middle ages. The first known lead object, a statue found in Turkey, dates from 6500 B.C. Historical records illustrate the use of lead in the time of ancient Rome (Woolley 1984) in, for example, plumbing, building, shipping, construction of fishing nets and weights, welding, pigments and paints, wine-making, coin manufacture, glassware, roofing, guttering, lining of aqueducts, sewage pits, piping, writing (lead pencils, sheets and tablets), pottery, vases, utensils, the preservation of foodstuffs using lead salts, condiments, wine, cosmetics, birth control, the manufacture of chastity belts, medicinal drugs and hair dye (Nriagu et al 1983).

### **1.4.2 Contemporary Sources of Lead**

The use of lead escalated dramatically around the time of the industrial revolution. With the advent of motor vehicles early in the 20th century, there was a substantial escalation in global environmental lead contamination, due to the widespread use of lead in petrol (Tong et al 2000) as an “anti-knocking” agent.

Lead has been used as a fixing agent for pigments added to paints. Even in areas where the addition of lead to paint has been discontinued, peeling or weathering of old lead-based paint from dwellings, school buildings and other infrastructure may contribute to elevated lead concentrations in dust and soil. Removal of lead-based paint from surfaces by burning/torching, scraping or sanding has been found to result, at least temporarily, in higher levels of exposure for families residing in affected homes. Improper practices in the removal of lead-based paint have the potential to contribute to highly elevated levels of lead exposure and blood lead concentrations in household members, especially young children (Jacobs et al 2003).

Work undertaken in “cottage industries”, for example motor vehicle repairs, jewellery manufacture, spray painting and repairs to electrical appliances using lead solder, may predispose household members to elevated levels of exposure (Counter et al 2000). Lead particles may also be transported from work on the skin, clothing and motor vehicles of workers in lead-related industries such as lead mining and smelting, motor vehicle repair workshops, radiator repair workshops, battery manufacturing plants, leaded glass manufacturers and other activities

(Chiaradia 1997, Gulson et al 1994). In this way home environments may become contaminated, increasing lead exposure risks in affected households.

Lead may leach from lead crystal decanters and glasses into the liquids they contain, especially if these are acidic as in the case of wine. Lead is present in tobacco at concentrations of approximately 2.5 to 12.2 µg/g (WHO 1977), and may be found in illicit alcoholic beverages produced in the informal sector (sometimes referred to in South Africa as “concoctions”). Lead has also been detected in some herbal or traditional medications (Dwivedi & Dey 2002). The use of lead ammunition has resulted in exposure to lead dust generated during gun or rifle discharge, from the ingestion of lead pellets (Greensher et al 1974) or from pellets embedded in human organs or tissues during shooting incidents (McQuirter et al 2004, Cao et al 2003, Dillman et al 1979, Greensher et al 1974). Engaging in hobbies in which it is used may also result in higher levels of lead exposure, including pottery-making, stained glass manufacture, glassblowing and screen printing (Landrigan et al 1980).

Uses for lead continue to be found in modern society. Currently it is known that lead is used in thousands of products and processes, including, in recent times, in electronic equipment, television sets, computers and mobile telephones (Tong et al 2000).

### **1.4.3 Sources of Exposure to Lead in South Africa**

Children in developing countries may be at particular risk of exposure to lead, involving multiple sources and higher levels of exposure than observed in

developed countries (Nriagu et al 1996). African children, in particular, may be at high risk as a consequence of a paucity of information available to the public on the sources and mechanisms of exposure to lead in children, as well as children's blood lead levels, ongoing use of leaded petrol and other products to which lead has been added, inadequate regulatory frameworks, weak enforcement of existing legislation, high levels of poverty & inequity, poor housing conditions and extensive malnutrition.

Lead was first discovered in South Africa in 1782, and a lead smelter erected in 1893 in the former Transvaal (a province of South Africa prior to the democratic era) (Snodgrass 1986). South Africa is currently among the ten largest producers of lead in the world (Dzioubinski & Chipman 1999). The country has two major lead producing mines, and four secondary (but no primary) lead smelters (Joseph & Verwey 2001).

Batteries account for more than 80% of lead consumption in South Africa, but other uses of lead in the country are of particular importance in terms of childhood exposure and public health, including petrol additives, lead-based paint, lead solder used in "cottage industries" and other informal sector activities, and para-occupational exposure (from adults working in lead-related industries).

### ***Lead in Petrol***

The addition of lead to petrol to reduce "engine knock" and improve engine efficiency, commenced during the 1920s in the United States of America. In the following decades, motor vehicle exhaust emissions of lead particles contributed to

widespread environmental lead contamination, making elevated lead exposure one of the priority global environmental health concerns in modern times (Chiaradia et al 1997).

At varying concentrations, lead has been added to petrol in South Africa since the 1920s. Until 1986 the maximum permitted lead content in petrol in South Africa was 0.836 g/litre - amongst the highest concentrations ever used anywhere in the world. In 1986 the maximum permissible petrol lead concentration was reduced to 0.6 g/litre. In 1989 the level was again reduced, to 0.4 g/litre, which is currently the maximum permissible petrol lead concentration in the country. In 1996, unleaded petrol (maximum lead concentration of 0.013 g/litre) was made available for the first time in South Africa.

By 2002, unleaded petrol constituted around 30% of the market share of petrol in South Africa (Moldan A, Director: South African Petroleum Industries Association, personal communication). In 2002 the South African parliament decided that the use of lead in petrol would be phased out by 2006 (RSA 2003).

### ***Lead in Paint***

Lead compounds have been added to paint all over the world because of their resistance to corrosion and protective properties. Lead based paint was first recognized as a source of childhood lead poisoning in Australia in 1904 (Gibson 1904). Since then numerous studies have demonstrated that children suffer neurological damage after ingesting even small amounts of lead based paint (Needleman & Gatsonis 1990; Needleman & Bellinger 1991). Following the

promulgation of International Labour Organisation Convention 13 on prohibition of the use of lead-based paint, numerous countries adopted international and national laws that limit or ban the use of lead based paint (ILO 1921).

The use of “white lead” in paint in South Africa was abolished late in the 1940s. Other forms of lead however, continued to be used in paint. In the mid-1970s, a voluntary agreement to limit the addition of lead to paint was reached among members of the South African Paint Manufacturers’ Association (SAPMA) (Hayes M, Director: South African Paint Manufacturers’ Association). Little is known however, about the extent of adherence to the voluntary agreement amongst SAPMA members, or about lead use practices amongst non-members. In any event, the peeling or weathering of old lead-based paint from walls, doors and windowsills of homes, schools and other buildings, remains a major public health concern. In 1979 a nation-wide survey conducted by the Department of Health revealed that about 20% of interior walls with leaded paintwork contained between 5% and 13% of acid leachable lead (Department of Health 1979). More recently (2001) a survey of paint samples taken from the classrooms of ten Johannesburg inner city primary schools showed that half had lead concentrations which equalled or exceeded the standard of 5 000 µg/g set by the USA Department of Housing and Urban Development (HUD) (Mathee & Röllin, unpublished data).

### ***Lead in Water***

During the first decades of the twentieth century lead plumbing and lead-lined water tanks were widely used in South Africa. Concentrations of lead in domestic water supplies, and human exposure to lead, were consequently also high. In 1917

for example, lead concentrations ranging from 0.5 to 7.6 parts per million (ppm) were measured in household water samples in a study undertaken in Cape Town. In a study of circumpulpal dentine of teeth taken from 28 exhumed bodies which had been buried in a Cape Town cemetery between 1863 and 1922, the mean lead level was 304 µg/g, with individual results ranging from 55 to 764 µg/g (Grobler *et al* 1996).

By the 1930s, lead poisoning was regarded as a serious enough concern in Cape Town to warrant the institution of a special investigation, which in turn led to the establishment of water treatment plants and the city-wide replacement of lead water storage tanks and plumbing with copper pipes. By the 1970s however, during the *apartheid* era demolition of the inner city Cape Town suburb of District Six, houses with lead plumbing were still found (Retief-Steyn 1976).

Available data indicate that levels of lead in reticulated drinking water in South Africa are currently relatively low. For example, during the period 2001/2002, all Johannesburg household samples tested by the Johannesburg Water Agency were below the detection limits for lead (Rimmer R, Head: Scientific Services, Johannesburg Water Agency, 2002, personal communication).

### ***Lead in Air***

Lead in the atmosphere is routinely monitored at several locations in South Africa, especially in major municipal areas. In the past, annual average air lead levels recorded at major urban centres in South Africa frequently exceeded the World

Health Organization guideline of  $0.5 \mu\text{g}/\text{m}^3$ . In Johannesburg for example, the annual air lead level measured in 1996 was  $0.86 \mu\text{g}/\text{m}^3$  (see Figure 1).

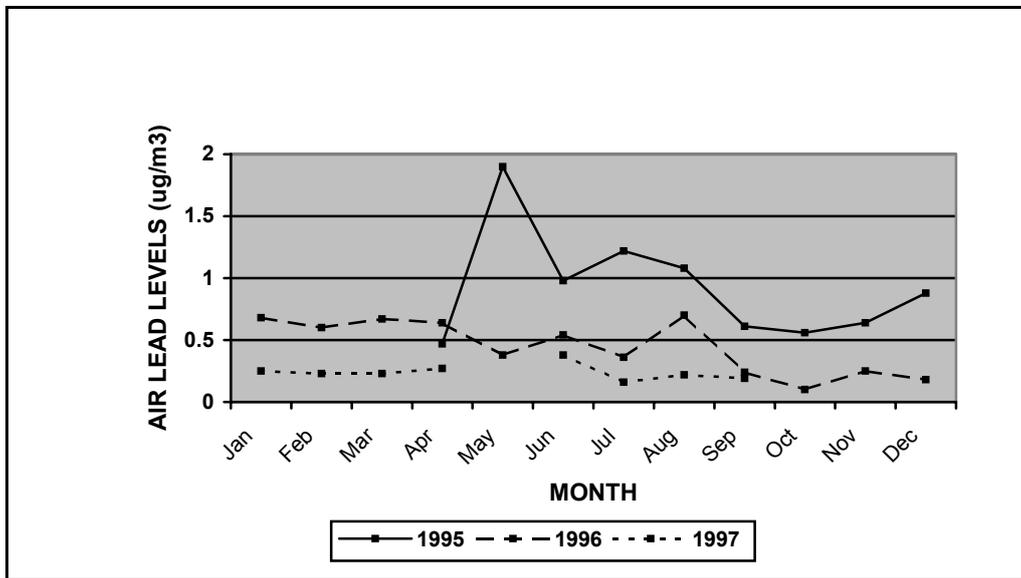


Figure 1. MONTHLY AIR LEAD LEVELS IN JOHANNESBURG

Since 1996 however, air lead levels in Johannesburg, as well as in other major cities in the country, appear to have been decreasing. Nevertheless, in certain areas, fallout leading to high soil lead concentrations remains a major environmental and public health concern. In a recent preliminary investigation of the lead content of soil from the playgrounds of nine Johannesburg inner city schools, soil lead concentrations ranged up to  $767 \mu\text{g}/\text{g}$ , with four schools having levels exceeding the USA HUD standard for lead in soil of  $80 \mu\text{g}/\text{g}$  (Mathee & Röllin, unpublished data).

## **1.5 PATHWAYS & ROUTES OF LEAD EXPOSURE IN CHILDREN**

Human exposure to lead may occur through direct dermal absorption, respiration or ingestion. Ingestion of lead-rich dust or soil, through the hand-to-mouth pathway, is by far the predominant route of exposure in children. Appreciable amounts of lead may be ingested during normal childhood developmental play and mouthing behaviour. Exposure may also occur to the lead contained in food, water and air.

Children with a pica tendency (the ingestion of non-food items), for example for paint, soil and cigarette butts, are at particular risk of having highly elevated blood lead concentrations. Lead poisoning and deaths from the ingestion of lead-based paint continues to be a contemporary public health concern (Mathee et al 2003, CDC 2001, Lanphear & Roghman 1997).

## **1.6 HIGH RISK GROUPS**

### **1.6.1 Children**

Children are innately susceptible to environmental hazards. Young children are particularly vulnerable to lead exposure, and the associated health and social effects. Children's nervous systems are rapidly developing, and therefore more susceptible to lead-induced disruption. Children also have a natural propensity to explore their living environments through touching and tasting. Mouthing of fingers, toys and other non-food objects is common in early childhood, and constitutes an important mechanism of exposure to lead. The risk of lead exposure is particularly high around the ages of two to four years, when exploratory behaviour is at a peak.

Some children have a pica tendency - the ingestion of non-food items such as paint chips and soil. Relative to adults, children absorb more ingested lead from their digestive systems (McCabe 1979).

### **1.6.2 Foetal Exposure**

The placenta constitutes a weak barrier to the transfer of lead from mother to foetus during pregnancy, with the potential for lead exposure during the foetal stage. Maternal and foetal blood lead concentrations are usually highly correlated (Raghunath et al 2000).

Lead absorbed by girls during childhood, and stored in the skeletal system, may subsequently be released during pregnancy, and cross the placental barrier, particularly in cases of calcium deficiency. Thus, even in the absence of an obvious source of contemporary exposure to lead in pregnant women, the risk of foetal lead exposure remains, with the potential for lead-related health and developmental effects to occur before birth (Gomaa et al 2002).

### **1.6.3 The Role of Poverty**

In contrast to Roman times, when elevated lead exposure was a particular concern amongst the wealthy classes, in the present time elevated lead levels are principally a problem among socially and economically deprived children. Poor people are more likely to live in substandard housing or reside near industrial centres and heavily trafficked areas. In lead-related industries, low-income workers are more likely to be exposed to lead, and to transport lead particles from the work environment into their homes on their hair, skin, clothing and motor vehicles. A

greater likelihood of nutritional deprivation in poor communities also increases their susceptibility to lead exposure (since lead competes with calcium for absorption) and lead-related ill health effects (Goyer 1995, Tong et al 2000).

#### **1.6.4 Para-occupational Exposure**

Children living in homes in which adults are involved in lead-related occupations, for example motor vehicle repairs, lead mining and battery manufacture, may be at high risk of lead exposure. Of particular concern are children in whose homes lead is used, in relation to either hobbies such as leaded glass, pottery or jewellery manufacture, or “cottage industries” such as repairs to electrical appliances or motor vehicles.

### **1.7 HEALTH & SOCIAL OUTCOMES OF CHILDHOOD LEAD EXPOSURE**

In recent decades researchers, using epidemiological studies of increasingly sophisticated design, and conducted in a variety of populations and settings around the world, have been able to discern a wide range of lead-related health and social effects at progressively lower blood lead concentrations. Several of these effects have been demonstrated to be persistent, even following interventions to reduce blood lead concentrations (Tong et al 1998, Tong 1998).

Over the past decade, 10 µg/dl of lead in blood has been internationally adopted as an “action” level (CDC 1991). In recent years however, studies have been reported in which significant associations between health effects and blood lead concentrations as low as 3 µg/dl have been demonstrated (Rogan & Ware 2003,

Canfield et al 2003, Schwarz and Otto 1991). These findings have contributed to current scientific consensus regarding the absence of a threshold of safety in relation to blood lead levels. In addition, the findings have implications for the future use of 10 µg/dl as an action level for lead in blood (Rogan & Ware 2003).

### **1.7.1 Death from Lead Poisoning**

Lead concentrations in excess of 100 µg/dl have been associated with death in children (Chisolm & Harrison 1957, Chisolm & Harrison 1956, Harris 1976).

### **1.7.2 Haematological Effects**

Lead inhibits the activity of δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, which are involved in haeme biosynthesis. As a consequence of these changes haeme synthesis is decreased, even at very low concentrations of blood lead (Hernberg & Nikkanen 1970). Decreased haeme synthesis is associated with increased urinary porphyrins, coproporphyrin and δ-aminolevulinic acid (ALA), as well as increased zinc protoporphyrins (ZPP). Increased urinary coproporphyrin has long been used as an indicator of excessive exposure to lead (EPA 2001).

General population studies have indicated that ALAD activity is inhibited at very low blood lead concentrations (Chisolm et al 1985). A study of Saudi Arabian boys found a negative association between blood lead levels and haematological values when the study group was sub-divided into “high” (>15 µg/dl) and “normal” (<15 µg/dl) lead exposure groups (Ahmed et al 1989).

### **1.7.3 Neurological Effects**

Excessive exposure to lead is associated with a range of neurological effects, the most severe of which is encephalopathy. Lead encephalopathy occurs primarily in children at blood lead levels of 80 µg/dl or higher (Needleman 1988), when a wide variety of signs and symptoms may be observed, including tremors and twitching, convulsions, paralysis, muscle soreness, fatigue, weakness, joint pain, a lack of coordination, visual abnormalities, loss of appetite, weight loss, constipation, vomiting, diarrhoea, abdominal pain, high blood pressure, agitation, coma, hallucinations, lethargy and malaise, irritability, headache, sleeping difficulty, confusion and death (Needleman 1988).

In the 1970s and early 1980s a number of studies were undertaken that showed a consistent pattern of poor performance on IQ or other psychometric tests among children with high lead exposure, than did reference groups, even in the absence of overt signs or symptoms of lead poisoning (de la Burde & Choate 1975, Ernhart et al 1981, Kotok et al 1977). For example, a mean Stanford-Binet IQ decrement of 5 points, fine motor dysfunction and altered behaviour profiles were found in pre-school children with pica for paint and plaster, and elevated blood lead levels, compared with matched controls who did not engage in pica (de la Burde & Choate 1975). Rummo et al (1979) observed hyperactivity and a decrement of around 16 IQ points on the McCarthy General Cognitive Index among children who had previously had encephalopathy and whose average maximum blood lead level at the time of encephalopathy was 88 µg/dl. Asymptomatic children with average blood lead levels of 68 µg/dl had an average decrement of 5 IQ points.

Despite methodological problems associated with a number of the studies outlined above, including a failure to take account of important confounding factors and misclassification of subjects into study and control groups, a consistent pattern nevertheless emerged of lower IQ scores and other neuropsychological deficits amongst children exposed to higher lead levels, even in the absence of obvious symptoms of lead exposure.

Numerous studies have also been undertaken that looked at the neurological impacts in children within the general population who had relatively low blood lead concentrations. Needleman and colleagues (1979) undertook a study in which associations between tooth dentine lead values and neurobehavioural effects, including IQ deficits, were examined in 6 to 7 year olds. An IQ deficit of approximately 4 points, poorer scores on tests of auditory and verbal processing, attention deficits and teachers' behaviour rating were achieved by those with dentine lead levels > 20 to 30 ppm (corresponding to blood lead values of 30 to 50 µg/dl), compared with those having lower tooth dentin lead levels (< 10 ppm). Re-examination 11 years later of a sub-set of this group showed that impaired neurobehavioural functions continued to be related to tooth dentine lead levels at age 6 to 7 years. Higher lead levels were also associated with lower standing in class, increased absenteeism, lower grammatical reasoning scores, lower vocabulary, poorer hand-eye coordination and longer reaction times (Needleman & Gatsonis 1990).

Wang et al (1989) reported a significant dose-effect relationship between blood lead levels and neuropsychological performance in 6 to 14 year old children (blood

lead levels ranging from 10 to 30 µg/dl) residing near a battery factory in Shanghai, China. The study estimated that an increase of 10 µg/dl of blood lead resulted in a lowering of verbal IQ by 8 points, performance IQ by 7 points and full-scale IQ by 9 points.

A significant increase in behavioural problems (inattention and hyperactivity) associated with increased blood lead levels was found in a group of 579 New Zealand children, whose blood lead levels ranged from 4 to 50 µg/dl (mean = 11.1 µg/dl) (Silva et al 1988).

A longitudinal study of 249 children associated elevated cord blood lead concentrations with decrements in measures of intelligence at the ages of 6, 12, 18 and 24 months. There was a 4.8 point difference in IQ scores between infants with cord blood lead concentrations less than 3 µg/dl compared with those having cord blood lead concentrations equaling or exceeding 10 µg/dl (Bellinger et al 1987).

In a prospective study of 172 children from the ages of 6 months to 5 years, an inverse, statistically significant relationship was found between blood lead concentration and intelligence at the ages of 3 and 5 years. The decline in intelligence was sharpest amongst those whose blood lead concentrations remained below 10 µg/dl. IQ declined by 7.4 points as lifetime average blood lead concentrations increased from 1 to 10 µg/dl (Canfield et al 2003).

#### **1.7.4 Hearing Loss**

Data from the National Health and Nutrition Examination Survey II (NHANES II) conducted in the USA were used to examine the relationship between blood lead level and hearing threshold. The probability of elevated hearing thresholds at 500, 1000, 2000 and 4000 Hz increased significantly ( $p < 0.0001$ ) with increasing blood lead concentrations, for both ears (Schwartz & Otto 1987).

Data from the Hispanic Health and Nutrition Survey relating to 3 545 subjects aged 6 to 19 years were used to confirm the relationship between elevated blood lead levels and hearing impairment. Lead levels were associated with an increased risk of hearing thresholds that were elevated above the standard reference level at all four frequencies (500 Hz, 1 000 Hz, 2 000 Hz, and 4 000 Hz). Lead was also associated with hearing thresholds when they were treated as a continuous outcome. The relationships appeared to continue at blood lead levels less than 10  $\mu\text{g}/\text{dl}$ . An increase in blood lead from 6 to 18  $\mu\text{g}/\text{dl}$  was associated with a 2-dB loss in hearing at all frequencies (Schwartz & Otto 1991).

#### **1.7.5 Delayed Onset of Puberty**

A cross-sectional analytical study undertaken in the USA associated elevated blood lead levels (3  $\mu\text{g}/\text{dl}$  compared with 1  $\mu\text{g}/\text{dl}$ ) in African American and Mexican American girls with significant delays in the onset of puberty. For white girls the relationship between measures of puberty and blood lead concentration was in the same direction as for African American and Mexican American girls, but not statistically significant. The authors suggested that the research findings be confirmed in further studies of a prospective design (Selevan et al 2003).

## **1.8 THE EPIDEMIOLOGY OF CHILDHOOD LEAD EXPOSURE IN SOUTH AFRICA (1970s & 1990s)**

### **1.8.1 Severe Lead Poisoning**

While it is likely that considerable under-reporting occurs in the country, from time to time there have been reports of overt lead poisoning amongst South African children. In 1974 for example, six children were admitted to the Frere Hospital in East London with severe lead encephalopathy. The blood lead levels of four of the children ranged from 106 to 290 µg/dl (blood lead levels of the remaining two children were unknown). Initial misdiagnoses occurred in four of the six cases. At least two of the six children are known to have subsequently died (Harris 1976).

In 1993 Rees and Schneider reported on the case of a 3-year old Soweto child who had been admitted to hospital with signs of severe lead poisoning and a blood lead level in excess of 100 µg/dl. Further investigation determined blood lead concentrations of 78 µg/dl and 96 µg/dl in two other children living in the same house. Damaged car batteries were found in the yard, and it was determined that household members had been removing lead plates from old car batteries for subsequent sale.

### **1.8.2 Newborns**

Under the umbrella of the Birth-To-Twenty (BTT) birth cohort study initiated in 1990, cord blood samples were obtained from a sample of 1 270 infants. Structured questionnaires administered ante-natally were used to obtain

information related to a wide range of socio-environmental factors, including maternal and paternal educational status, economic status, marital status, home language, housing quality, the presence of dust, crowding, fuel usage practices, transport use, the main water source as well as tobacco use and alcohol consumption. Further information related to, for example, the sex of the baby, birth weight, head circumference, length, gestational age and the presence of congenital abnormalities, were obtained from birth records at the time of delivery (Mathee et al 1996).

Due to clotting, 389 samples were discarded, and blood lead measurements carried out on 881 samples. The blood lead distribution ranged from 2 to 20 ug/dl, with the mean equalling 5.9 ug/dl. Following transformation of the positively skewed blood lead distribution, univariate regression analyses were carried out on the log-normally distributed data to examine the relationship between individual variables and blood lead concentrations.

A number of social and environmental factors were significantly related to cord blood lead levels, including, for example, levels of crowding and the presence or presence of a separate kitchen for cooking purposes only (Mathee 1995).

Maternal and cord blood lead levels were examined in a study undertaken at the King Edward VIII hospital in Durban. Amongst 21 study subjects, the mean maternal blood lead level was  $21.75 \pm 8.35$   $\mu\text{g/dl}$ , whilst the mean cord blood lead level was  $15.53 + 4.80$   $\mu\text{g/dl}$ . Amongst both maternal and cord blood samples, lead levels exceeded 10  $\mu\text{g/dl}$  in 95% of cases. Five percent of cord blood lead levels

exceeded 25 µg/dl. Maternal and cord blood lead levels were positively correlated (Chetty et al 1993).

### **1.8.3 Pre-school Children**

In a study of 293 Cape Town pre-school children aged 4 to 6 years reported in 1986, the mean blood lead concentration was 16 µg/dl (ranging from 2 to 49 µg/dl). Around 85% of children had blood lead levels equalling or exceeding 10 µg/dl, and 4.4% had blood lead levels  $\geq 30$  µg/dl. Children with elevated blood lead levels tended to live in inner city suburbs, and in homes close to a busy road. Appreciable lead loads were determined in paint, soil and dust samples taken from the homes of children with the highest and lowest blood lead levels. Mouthing behaviour was most pronounced amongst children with the highest blood lead concentrations (Deveaux et al 1986).

### **1.8.4 Young School Children**

The first indications of high blood lead levels among Cape school children emerged from a screening study undertaken by von Schirnding and colleagues in 1982, at a time when the maximum permissible petrol lead concentration in South Africa was 0.836 g/litre. Among 1 234 Coloured first and second grade children attending schools in the Cape Peninsula, it was found that the average blood lead level of children from urban industrial areas was twice as high as among children from suburban areas (von Schirnding et al 1986). At a school situated in the urban area of Woodstock (close to the central business district of Cape Town), the average blood lead level was 22 µg/dl, while at a control school in Hout Bay, the level was 11 µg/dl. Seventeen percent of children from the Woodstock school had blood lead

levels greater than, or equal to 30 µg/dl, whilst no children from Hout Bay had blood lead levels in this range. Pilot investigations suggested no obvious lead source, such as lead plumbing or water with a high lead content, in the homes of children with the highest blood lead levels. There was however evidence of behavioural abnormalities in children with high lead levels (von Schirnding et al 1984).

Following the screening study a cross-sectional, analytical study of first grade Woodstock school children was carried out, together with a nested case-control study to determine sources of lead exposure in the home environment among children with high blood lead levels, as compared to children with low concentration levels (von Schirnding et al 1991a, von Schirnding et al 1991b).

In the cross-sectional study, the median blood lead level for all children living in the inner city study area of Woodstock was found to equal 16 µg/dl. A statistically significant difference existed in the blood lead concentrations of White and Coloured children, with the former having a mean level of 12 µg/dl and the latter a mean of 18 µg/dl. Among Coloured pupils 13% had blood lead levels greater than or equal to 25 µg/dl; however no White pupils had blood lead levels in this range.

Considerable variation in the blood lead concentrations of pupils also existed between schools, with median blood lead levels ranging from 8 to 21 µg/dl. There was a strong association between raised blood lead levels and the proximity of schools to busy roads. There remained a statistically significant association between blood lead levels and school, when socio-economic factors were taken

into account. Among the Coloured schools blood lead levels of pupils attending schools in the immediate proximity of heavy traffic density averaged between 18 and 21 µg/dl; at schools further away median blood lead levels averaged around 13 µg/dl. Other factors found to be associated with children's blood lead levels included the state of their housing, certain cultural factors, and home language (von Schirnding et al 1991b).

From the nested case control study conducted subsequently, it emerged that certain physical and social characteristics of the child's home environment were important, as well as factors relating to behavioural characteristics of children. Sources of lead were found in the homes of both cases and controls, but were more accessible in the homes of cases than of controls. The homes of cases were in a more dilapidated state than those of the controls, with more flaking lead paint and more lead rich dust, and were also in considerable need of attention as far as overall domestic hygiene was concerned. Lead levels in water, air and street dust were not found to vary significantly between cases and controls, nor were there significant differences with respect to the overall nutritional status or reported dietary habits. However the homes of cases were found to be more crowded than those of the controls, and the mother's level of schooling, as well as the total family income was lower among cases than controls. Significant differences between the cases and controls existed with respect to a previous history of pica-related behaviour. Although most children were not reported to have routinely ingested non-food items, and pica for paint was not a significant factor, more cases than controls were observed to eat items such as plaster, cement, soil, sticks and matchsticks. Significant differences were also found between cases and controls

with respect to generalized mouthing activities of children, these being much more pronounced in cases than controls.

A repeat blood lead survey was carried out in 1991 in which blood lead levels of children in the same inner city areas (as in the 1984 study) were measured, as well as of children living in other suburbs of the Cape Peninsula (von Schirnding et al 2001). This was subsequent to petrol lead levels having been reduced to 0.4 g/litre in South Africa. The results showed that in both 1984, when the first cross-sectional analytical study was carried out, and 1991, median blood lead levels of 16  $\mu\text{g}/\text{dl}$  were measured in the inner city Woodstock area. In 1991, more than 90% of children continued to have blood lead levels equalling or exceeding the international level of concern of 10  $\mu\text{g}/\text{dl}$ .

A cross-sectional analytical survey of the blood lead levels of first grade school children conducted in 1995 showed that blood lead concentrations in Johannesburg were similiarly high. Seven schools in three Johannesburg areas of relatively low socio-economic status were selected for inclusion in the study. The areas sampled included the inner city (3 schools), the densely populated township of Alexandra, which is located to the north of central Johannesburg and comprises both formal and informal housing developments (2 schools), and the formal townships of Westbury and Newclare to the west of central Johannesburg (2 schools).

Blood lead levels for the total sample of 433 children, ranged from 6 to 26  $\mu\text{g}/\text{dl}$ , with the mean level equalling 12  $\mu\text{g}/\text{dl}$ . No statistically significant differences in

mean blood lead levels by area, or amongst the seven schools, were determined. The blood lead levels of 78% of children were or exceeded 10  $\mu\text{g}/\text{dl}$ . A number of risk factors and potential outcomes were associated with elevated blood lead levels. These included the mother having only a primary school education, the presence of smokers in the home, and regular consumption of canned foods. In addition, elevated blood lead levels were associated with the respondent's perception that the child's schoolwork was poor, and that the child was overactive (Mathee et al 2002).

In a study of the blood lead distribution amongst more than 1 200 children from KwaZulu-Natal, the mean blood lead level amongst children from an informal settlement in Durban was 10  $\mu\text{g}/\text{dl}$ . Five percent of children had blood lead levels  $\geq 25$   $\mu\text{g}/\text{dl}$ . Risk factors for elevated blood lead levels included distance from tarred roads, overcrowding, household hygiene habits, and the use of solid fuels as a domestic energy source (Nriagu et al 1997).

### **1.8.5 Blood Lead Levels in a Lead Mining Town (Aggeneys)**

A study was undertaken at a primary school in the lead mining town of Aggeneys, and in the comparison, non-mining town of Pella, located around 40 kilometres away. Eighty six children from Aggeneys aged between 6 and 10 years and 68 children from Pella were studied. Questionnaires were administered to a randomly selected sub-sample of children's parents (36 children from Pella and 49 children from Aggeneys), and information collected on socio-demography, housing conditions, children's behaviour (for example play sites, hand-to-mouth activity,

pica), environmental and personal hygiene, and occupants' work history (von Schirnding et al 2003).

Statistically significant differences in blood lead distributions between the two communities were found ( $p = 0.0001$ ). Blood lead levels in Aggeneys averaged around  $16 \mu\text{g}/\text{dl}$ , with 66% of children having blood lead levels  $\geq 15 \mu\text{g}/\text{dl}$ . In Pella, the mean blood lead level was  $13 \mu\text{g}/\text{dl}$ , with 35%  $\geq 15 \mu\text{g}/\text{dl}$ . Blood lead levels in Aggeneys ranged from 9 to  $27 \mu\text{g}/\text{dl}$ , and at Pella from 6 to  $22 \mu\text{g}/\text{dl}$ . Blood lead levels were not related to age or gender, and males and females respectively showed significant differences in blood lead concentration between the communities. Aggeneys children were slightly taller and heavier than children from Pella. In general, the impoverished community in Pella lived in small houses (many of them make-shift), which were more dilapidated and densely populated than those of people living in the more affluent Aggeneys. More parents of children living in Aggeneys had a high school education (64% versus 43% in Pella) and more Aggeneys fathers had post school qualifications (25% versus 0 in Pella).

Further analyses of blood lead levels among children in the mining town of Aggeneys were conducted, comparing those with blood lead levels less than  $18 \mu\text{g}/\text{dl}$ , to those greater than or equal to  $18 \mu\text{g}/\text{dl}$  ( $18 \mu\text{g}/\text{dl}$  formed a mid-point within the distribution of blood lead levels in the sample). This revealed that within Aggeneys, more "low" blood lead children than "high" blood lead children had a father with a post-school qualification, and that children who had failed a grade at school, had higher blood lead levels than other children. It was also found that children of fathers/male guardians who showered or bathed at home immediately

upon returning from work, tended to have lower blood lead levels than those who did not. Two-thirds of fathers of high blood lead children showered at work, compared to 41% of fathers of low blood lead children. A higher percentage of the former group had their clothes washed at work rather than at home, and none of the former group showered immediately upon coming home, whereas 19% of the latter group did so.

The mean blood lead levels of children in both communities tested were higher than would be expected in a rural setting. Blood lead levels at Aggeneys in particular, which averaged around 16  $\mu\text{g}/\text{dl}$ , were comparable to blood lead levels of Cape urban (inner-city) “Coloured”<sup>1</sup> children of a similar age (18  $\mu\text{g}/\text{dl}$ ).

## **1.9 MOTIVATION FOR THE STUDY**

The current study was undertaken to address the paucity of information in South Africa about the blood lead concentrations of first grade school children. This project has been of particular importance in light of the absence of a national blood lead surveillance programme in the country. Data and findings generated through the implementation of this study will help to inform national policy proposals in relation to the prevention of childhood lead poisoning.

---

<sup>1</sup> A classification adopted during South Africa’s apartheid era that refers to people of mixed racial background. Use of the term “Coloured” in this work does not imply acceptance or approval.

## 1.10 STUDY OBJECTIVES

The objectives of the study were to:

- Determine the blood lead distributions of first grade South African school children in key settings in the country (large cities – Cape Town & Johannesburg, a medium-sized city - Kimberley, small rural towns – Pella & Onseepkans, and a lead mining town - Aggeneys);
- Identify the risk factors for elevated blood lead concentrations in children;
- Compare blood lead distributions determined in this study, with those determined in similar studies conducted prior to the introduction of unleaded petrol, in a sub-set of the study areas.

## **Chapter 2 - METHODS**

### **2.1 STUDY DESIGN**

Cross-sectional, analytical studies were conducted to determine the blood lead distributions, and associated environmental and other risk factors for elevated levels of lead in children's blood, in selected areas in South Africa. The data generated were used to conduct geographical comparisons, and for some of the study areas, temporal comparisons with the findings of similar studies undertaken prior to the introduction of unleaded petrol.

### **2.2 STUDY AREAS AND POPULATION**

#### **2.2.1 Study Areas**

The study was undertaken in selected suburbs of the large South African cities of Cape Town and Johannesburg, the medium-sized City of Kimberley (population estimate - 211 000), two rural towns in the Northern Cape Province (Pella and Onseepkans) and the lead mining town of Aggeneys (also in the Northern Cape province).

Primary schools formed the principal unit of sampling. A total of 29 primary schools from the five areas were studied. In Cape Town and Johannesburg the schools included in the study were those that had been studied prior to the introduction of unleaded petrol. These schools had previously been selected to reflect the

situation in close proximity to, and further away from, heavily trafficked roads. In Cape Town, for example, six schools were located in the busy inner city suburb of Woodstock, while three schools were in Hout Bay, a peri-urban suburb associated with lower traffic densities. One school, previously randomly selected, from Mitchell's Plain was also included in the study. Timeous approval could not be obtained from the headmaster of one inner city school that had been included in the earlier (1991) study, and was consequently excluded. Ultimately eleven schools were included in the current Cape Town phase of the study, compared with twelve in 1991.

In Johannesburg, three new schools were added to the seven studied in 1995, resulting in a total of ten Johannesburg schools being included in the current study. The schools previously included were located in Alexandra (two schools), Westbury (two schools) and the Johannesburg inner city area (three schools). The additional schools were in Soweto (two schools) and Riverlea Extension 1 (one school).

In the City of Kimberley, four schools of similar socio-economic status, two of which were close to, and two relatively further away from a busy road, were selected for inclusion in the study.

The mining town of Aggeneys, in which the blood lead concentrations of primary school children had been measured during 1991, is situated around 110 km north-east of Springbok in the Northern Cape Province. South Africa's largest deposits of lead occur in the region. The Black Mountain Broken Hill deposit is characterized

by high lead-to-zinc ratios. The town of Aggeneys developed around the mine, and is fully electrified, with a reticulated water supply from the Orange River.

The community of Pella is situated about 40km to the north-east of Aggeneys, close to the Orange River. There was no electricity or reticulated water inside homes, but piped water from the Orange River was accessed through outside taps. The only primary schools in Aggeneys and Pella were included in the study, and in Onseepkans both primary schools were included. The schools in Pella and Aggeneys had previously (1991) been studied.

## 2.2.2 Study Sample

The study sample comprised first grade children attending the selected primary schools, for whom written, informed parental consent had been obtained, and who were present and willing to participate on the day selected for fieldwork at the school concerned. As can be seen from Table 1, a total of 1 287 children from 29 selected primary schools participated in the cross-sectional component of the study.

**Table 1. THE STUDY SAMPLE**

<b>STUDY AREA</b>	<b>NUMBER OF SCHOOLS</b>	<b>NUMBER OF SUBJECTS IN THE SAMPLE</b>
<b>CAPE TOWN</b>	11	429
Woodstock	7	239
Mitchell's Plain	1	95
Hout Bay	3	95
<b>JOHANNESBURG</b>	10	382
Alexandra	2	89
Westbury/Riverlea	3	142
Soweto	2	56
Inner City	3	96
<b>KIMBERLEY</b>	4	356
<b>RURAL NORTHERN CAPE</b>	4	119
Aggeneys	1	21
Pella	1	55
Onseepkans	2	43
<b>TOTAL</b>	<b>29</b>	<b>1 287</b>

### **2.3 MEASUREMENT OF BLOOD LEAD CONCENTRATIONS**

Samples of approximately seven millilitres of venous blood were collected from participating children into sterile EDTA-containing test tubes (BD Vacutainer System) that had previously been determined to be free of trace metals. All blood samples were collected by professionally trained nurses, registered with the Health Professions Council of South Africa (HPCSA). Disposable, sterile blood sampling equipment and aseptic sampling techniques were used throughout. Collected samples were stored under refrigeration, and transferred to the analytical laboratory in Johannesburg as soon as was possible after sampling.

Blood lead analyses were carried out in the laboratories of the National Institute for Occupational Health (NIOH) in Johannesburg. The NIOH participates in national and international quality control programmes (Röllin et al 1988). Lead concentrations were measured using a flameless atomic absorption method of addition (Model Perkin-Elmer Analyst 300 with HGA 850) adapted from Baily (1979).

With each batch of samples a reagent blank and set of working standards were run simultaneously. The coefficient of variation in blood lead samples was 5.8%. The limit of detection for lead in blood was 0.1 µg/dl.

All medical waste, for example used swabs, was collected into red plastic bags and marked appropriately. Sharp objects such as needles, were disposed of into

approved “sharps” containers, and sealed according to the manufacturer’s instructions. Red plastic bags and sharps containers were despatched for incineration by approved service providers.

## **2.4 ADMINISTRATION OF QUESTIONNAIRES**

Structured questionnaires (Annexure 1) were designed to obtain information about the health of the child, household socio-economic status, housing type and other potential risk factors for elevated blood lead levels. Questionnaires were distributed to the homes of study children in advance, and self-administered by parents or guardians. Parents were requested to return completed questionnaires to the school on the following day.

For the rural areas of the Northern Cape (Aggeneys, Pella and Onseepkans), questionnaires were translated into Afrikaans, which was the predominant or only language spoken, by professional translators from the University of the Witwatersrand, and back-translated for the purposes of accuracy. Respondents were provided with the contact details for the research team, and were encouraged to call in the event of any uncertainty about the questionnaire.

Potential observer bias was controlled through the administration of a standardized questionnaire that had been pre-tested to identify and address variations in interpretation. Wherever possible, missing information was obtained through telephonic follow-up with respondents.

## **2.5 MEASUREMENT OF ENVIRONMENTAL LEAD CONCENTRATIONS**

In order to obtain an indication of environmental lead concentrations, samples of paint, soil, dust and water were taken from the grounds of the study schools, and analyzed for lead content. Dust samples were collected by swiping a marked floor area of one squared foot, using Johnson & Johnson wet wipes. Swiping was conducted using vertical and then horizontal motions across the marked area. Wipe samples were double-bagged in Ziploc bags and marked with a unique identification code.

Paint samples, approximately the size of a business card, were collected from the walls of a randomly selected grade one classroom at each school, using a standard paint scraper obtainable from hardware stores, and collected into a Ziploc bag.

Composite soil samples, taken over an area of approximately one squared metre, on or near the main school playground, were collected using a plastic teaspoon, into standard sample jars. Approximately five scoops of soil were included in each soil sample.

## **2.6 HOME ASSESSMENTS (JOHANNESBURG)**

In Johannesburg, visits were undertaken to the homes of the twenty subjects with the highest blood lead concentrations, in order to further investigate potential sources of lead exposure within the home setting. Floor and windowsill dust, paint

and soil samples were collected for lead content analysis, and semi-structured interviews were conducted with one or both parents, to obtain details of lead-related hobbies and “cottage industries”, as well as the potential for transfer of lead into the home by adults who worked in lead-related settings or occupations.

## **2.7 RESIDENTIAL PAINT SAMPLING (JOHANNESBURG)**

Using a database obtained from the Planning Department of the Johannesburg Metropolitan Municipality, sixty Johannesburg suburbs, stratified by date of proclamation, were randomly selected for inclusion in the study. Thus twenty suburbs were selected for each of the periods (a) 1901 to 1947 (when the use of white lead is thought to have been discontinued), (b) 1948 to 1978 (around the time when the voluntary industry agreement to limit the addition of lead to paint was reached) and (c) 1979 to the present day (during which time it was expected that the use of lead-based paint had been voluntarily restricted by paint manufacturers).

Around four dwellings from each of the selected suburbs were included in the study. Through drive-by exercises dwellings that appeared to be in a state of degeneration, or on which peeling/flaking paint was observed, were selected for inclusion in the survey. Informed verbal consent was sought from a senior member of the household present at the time of the visit, and where approval was given, paint chip samples were collected from the interior and exterior surfaces of the dwellings for lead content analysis.

The paint chip collection methodologies adopted in this study were consistent with the guidelines of the United States Environmental Protection Agency (EPA 2001) and the United States Department of Housing and Urban Development “Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing” (HUD 1995). A stainless steel paint scraper was used to remove all layers of paint from the surface, while avoiding the removal of layers of substrate. Single surface rather than composite samples were collected. The paint chip samples were 5 to 8 squared centimeters in size (2 to 3 squared inches). At least one paint sample from an interior or exterior wall, impact/friction surface or chewable surface was obtained from each dwelling. Wherever possible, both interior and exterior samples were collected. Levels of lead in paint are reported in  $\mu\text{g/g}$ . Paint lead concentrations were assessed against standards of the USA EPA, which stipulates that “lead based paint is paint that contains lead levels equal to or greater than 5 000  $\mu\text{g/g}$ ” (US EPA 2001).

## **2.8 LEAD CONCENTRATIONS IN PAINTED CHILDREN’S TOYS**

As part of a preliminary scanning process, painted wooden toys and coloured pencils were purchased from toy stores, craft markets and stationery stores in Johannesburg. Paint samples were collected from the purchased toys and the lead content analyzed by the NIOH.

## **2.9 ETHICAL CONSIDERATIONS AND APPROVALS**

Clearance for the study was obtained from the Committee for Ethical Research on Human Subjects of the University of the Witwatersrand (Certificate number M-01-15-16). Permission for the study was obtained from the provincial Departments of Education of Gauteng, the Western Cape and the Northern Cape. Consent was also obtained from all school principals, who in turn obtained consent from the school governing bodies on behalf of the researchers.

Parents of potential study subjects were informed about the study through a subject information sheet (Annexure 2), which also included information about their right of refusal at any time, as well as their right of access to the results of blood lead analyses relating to their child.

Written parental consent was obtained for all children participating in the study. For those study subjects whose parents did not sign the consent form (Annexure 3), but who personally or telephonically communicated to researchers their willingness to have their child participate in the study, oral consent was recorded by the researcher in the presence of a witness, or taken telephonically and repeated to a witness, both of whom subsequently signed the consent form.

On the day of fieldwork, all children were asked about their personal willingness to participate in the study. Those not wishing to participate, as well as those who were deemed to be overly anxious, were excluded.

On completion of the study, a lead fact sheet was designed and delivered to the study schools for distribution to the parents of the study children.

## **2.10 DATA PROCESSING AND ANALYSIS**

Responses recorded on the returned questionnaires were screened and a coding sheet developed. Responses that were unclearly recorded, or that did not make sense, were followed up through telephonic communication with the respondents.

Data entry was undertaken by the Biostatistics Unit of the Medical Research Council. All data were “double punched” by two separate individuals, to reduce the possibility of processing errors. Outliers and anomalies were cross-checked with the raw data by members of the research team, and corrected as necessary.

Statistical analyses were carried out in consultation with the Biostatistics Unit of the South African Medical Research Council, to examine the relationships between individual variables and blood lead concentrations. However the analyses for this thesis were all carried out by the author.

The method of analysis needed to take into account the study design, in other words that children were clustered into schools, which were the sampling units. The actual lead level was regarded as the response variable and considered to be continuous. Thus linear regression for continuous explanatory variables and analysis of variance for discrete explanatory variables were used to examine

relationships with lead levels. Robust standard error calculations were used to take into account the clustering. Analyses were carried out using the survey regressions facilities of the Statistical Package STATA release 7.0 (2001).

To find the most important risk factors for high lead levels, stepwise multiple regression models were fitted; again using robust standard error calculations. This was done separately for each city/site and then combined over all sites in order to compare the levels between sites.

In order to decide which variables to include in the final multiple regression models, a backward elimination approach similar to that recommended by Vittinghoff et al (2005) was followed. In the terminology of Vittinghoff et al the inferential goal in this study was to identify the most important predictors of an outcome. Thus variables were included in the final model (a) if they met a liberal backward selection criterion, namely using a probability for removal level of 0.15 and (b) if they played a confounding role in that their inclusion altered the magnitude of the effect of any predictor of primary interest by 10% or more.

Lead levels were compared between years (Johannesburg, Cape Town, Aggeneys and Pella) and cities (and areas within cities), allowing for interaction between year and city and year and area, adjusting for confounders found to be important (for example socio-economic status, sex and age) using a multiple regression analysis of covariance approach.

The lead levels were compared between years using an aggregated approach; in other words using the mean lead level for each school, and then basing the comparison on a two way analysis of variance with terms for school and year, weighted by the number of children in each school in each year.

## **2.11 RESPONSE RATE**

Parents of 27% of the total number of 2 216 eligible children refused to give permission for their participation. From interviews with class teachers and school principals, it appeared that the main reasons for non-consent were fears of infection, especially with the Human Immuno-deficiency Virus (HIV), as well as fears that HIV testing would covertly be conducted on the blood samples taken. A further 328 children were excluded because of absenteeism on the day of fieldwork, personal refusal by children to participate or perceptions by the study team that potential study children were overly anxious. The final response proportion was 58%.

Table 2 gives a breakdown of the response rate by the various study areas. As can be seen, consent rates were highest in the rural towns of Pella and Onseepkans, but were lower in the more developed town of Aggeneys. The lowest response rate was found in Johannesburg. Overall, 73% of parents responded positively to the request to have their children participate in the study. In total, blood samples were taken from 58% of the potential participants.

Table 2. RESPONSE RATES BY STUDY AREA

STUDY AREA	MAXIMUM POTENTIAL PARTICIPANTS	# WITH PARENTAL CONSENT	% WITH CONSENT	# WITH BLOOD SAMPLES SUCCESSFULLY OBTAINED	% WITH BLOOD SAMPLES SUCCESSFULLY TAKEN
<b>PELLA</b>	72	68	94%	55	76%
<b>ONSEEPKANS</b>	49	47	96%	43	88%
<b>AGGENEYS</b>	34	28	82%	21	62%
<b>KIMBERLEY</b>	574	428	75%	356	61%
<b>CAPE TOWN</b>	710	603	85%	429	60%
Woodstock	326	292	90%	182	56%
Mitchell's Plain	130	110	85%	94	72%
Hout Bay	254	201	79%	152	60%
<b>JOHANNESBURG</b>	777	441	57%	383	49%
Alexandra	176	104	59%	89	51%
Westbury/Riverlea	246	168	68%	142	58%
Soweto	116	62	53%	56	48%
Inner City	239	107	45%	96	40%
<b>TOTAL</b>	2 216	1 615	73%	1 287	58%

## **Chapter 3 - RESULTS**

### **3.1 DESCRIPTION OF THE STUDY SAMPLE**

Blood samples were successfully obtained from a total of 1 287 first grade school children from study sites in Cape Town (429), Johannesburg (383), Kimberley (356), Onseepkans (43), Pella (55) and Aggeneys (21).

Given the degree of population variability, as well as the likely variation in the sources of exposure to lead across the five study sites, profiles for each of the study groups will be presented separately; Onseepkans & Pella (rural Northern Cape), Aggeneys (the lead mining town), Kimberley (a medium-sized city), while Cape Town and Johannesburg are regarded as large cities with different climatic & geographic profiles.

#### **3.1.1 Rural Northern Cape (Pella & Onseepkans)**

A total of 98 school children from Pella and Onseepkans, two remote rural towns in the Northern Cape, participated in the study. Pella and Onseepkans were selected to provide an indication of the “background” levels of lead exposure, against which lead exposure in the urban areas and the mining town might be measured. Onseepkans is located along the banks of the Orange River, which forms a border between South African and Namibia, while Pella is located around 8 kilometres away from the Orange River. A profile for the study sample drawn from the two

towns is given in Table 3, which also provides profiles for the remaining four study samples (Aggeneys, Kimberley, Cape Town and Johannesburg).

**Table 3. PROFILES OF THE STUDY SAMPLES (BY SITE)**

	PELLA/ONSEEPKANS	AGGENEYS	KIMBERLEY	CAPE TOWN	JOHANNESBURG
<b>N</b>	98	21	355	429	383
<b>% females</b>	50%	60%	45%	50%	48%
<b>Median Age</b>	6.7 years (range 6.7 – 8.3 years)	7.2 years (range 6.8 – 8.2 years)	7.2 years (range 5.8 – 11.2 years)	7 years (range 5 to 11 years)	7 years (range 5 to 12 years)
<b>Home Language</b>					
African	0%	5%	29%	32%	51%
English	1%	5%	20%	44%	20%
Afrikaans	99%	90%	51%	22%	21%
<b>Population Group</b>					
Black African	0%	5%	50%	37%	65%
Coloured	100%	74%	48%	57%	33%
White	0%	14%	<1%	5%	<1%
<b>Mean number of people per dwelling</b>	6.5 people (range: 2-15)	6 people (range: 2-16)	5 people (range: 2 to 20)	5 people (range: 2 to 20)	5 people (range: 2 to 31)
<b>Maternal Education</b>					
No Schooling	9%	5%	7%	2%	7%
Primary School	21%	10%	6%	31%	7%
High School	68%	80%	73%	60%	80%
Tertiary Education	2%	5%	14%	7%	6%
<b>Paternal Education</b>					
No Schooling	13%	0%	7%	2%	10%
Primary School	13%	5%	7%	34%	9%
High School	74%	84%	72%	57%	72%
Tertiary Education	0%	11%	15%	7%	9%
<b>Has indoor water supply</b>	11%	100%	58%	76%	66%
<b>Has outdoor toilet</b>	91%	5%	59%	32%	51%
<b>Housing Type</b>					
Free-standing dwelling	90%	95%	87%	58%	61%
Flat/apartment	2%	0%	3%	17%	20%
Backyard dwelling	2%	5%	1%	7%	7%
Informal dwelling	5%	0%	9%	18%	11%
<b>% of dwellings <math>\geq</math>25 years old</b>	27%	33%	33%	25%	30%
<b>% of dwellings in need of major repairs</b>	57%	29%	33%	<b>31%</b>	<b>39%</b>
<b>Electricity used for Cooking</b>	44%	100%	70%	89%	88%
<b>Paint peeling from interior surfaces</b>	26%	48%	26%	28%	35%
<b>Paint peeling from exterior surfaces</b>	27%	43%	31%	25%	30%
<b>Child has pica for paint</b>	4%	0%	4%	3%	4%
<b>Dwelling within 1 block of busy road</b>	0%	52%	56%	35%	53%
<b>Someone smokes at home</b>	69%	48%	44%	52%	52%
<b>Child travels to school by privately owned motor vehicle</b>	0%	5%	36%	26%	6%
<b>Child attended a crèche/nursery school</b>	92%	90%	83%	88%	70%
<b>Schoolwork perceived to be average/poor</b>	67%	29%	60%	39%	47%
<b>% children repeating grade one</b>	7%	0%	8%	3%	10%

As can be seen from Table 3, the Onseepkans/Pella sample was relatively impoverished and infrastructure within the towns was relatively under-developed. For example, only 11% of dwellings had an indoor water supply, only 44% of households used electricity for cooking and 91% used an outdoor toilet. Many dwellings appeared to be in a state of degradation, with 57% reported to be in need of major repairs.

The household income distribution for Pella and Onseepkans (as well as for Aggeneys and Kimberley) is given in Table 4. As shown, a large proportion of households in Onseepkans and Pella were earning under R1000.00 per month. Household incomes in Pella/Onseepkans were considerably lower than that of their counterparts in Kimberley or Aggeneys.

**Table 4. HOUSEHOLD INCOME DISTRIBUTION IN THE NORTHERN CAPE**

	R0 – R1000	R1001 – R3000	R3001 – R5000	R5001 – R8000	R8001 – R10 000	> R10 000
<b>Aggeneys</b>	0%	52%	24%	14%	10%	0%
<b>Pella</b>	57%	37%	2%	4%	0%	0%
<b>Onseepkans</b>	85%	15%	0%	0%	0%	0%
<b>Kimberley</b>	47%	28%	15%	6%	2%	1%

Local roads were unpaved and 69% of the study children's homes were described as very or slightly dusty. In order to save money or to generate an income, motor vehicle repair work was regularly undertaken at the homes of 8% of children, while repairs to electrical appliances (which may also involve the use of lead solder) were undertaken in 8% of homes, and spray painting was undertaken at the home of 1% of subjects. Motor vehicle ownership levels were relatively low, with only 18% of households having ownership of one or more cars.

Most study subjects (90%) were reported to be well at the time of the study, while 9% were reported to be unwell and 1% of the respondents were unaware of the health status of the child. Most children (92%) were reported to normally have a good appetite. Table 5 gives the reported prevalence in Onseepkans/Pella as well as the other study sites, of selected ill health symptoms in children at the time of the study. Home remedies had been administered to 54% of children to treat illness or to improve their health.

Levels of smoking in Onseepkans/Pella were the highest among all five samples, with 69% of children living in a house with one or more smokers.

**Table 5. REPORTED ILL HEALTH CONDITIONS BY STUDY SITE**

<b>ILL HEALTH SYMPTOMS</b>	<b>PELLA &amp; ONSEEPKANS</b>	<b>AGGENEYS</b>	<b>KIMBERLEY</b>	<b>CAPE TOWN</b>	<b>JOHANNESBURG</b>
Abdominal pains	14%	44%	29%	10%	13%
Convulsions	1%	0%	3%	1%	1%
Anaemia	1%	0%	0%	2%	0%
Tiredness	2%	11%	8%	3%	2%
Headache	15%	44%	47%	13%	19%
Constipation	5%	11%	20%	4%	4%
Diarrhoea	3%	22%	4%	3%	2%
Vomiting	5%	0%	14%	4%	5%
Inability to concentrate	10%	0%	21%	5%	12%
Irritability	5%	Inc	4%	6%	4%
Weakness	4%	Inc	9%	3%	2%
Colds & Flu	47%	Inc	Inc	Inc	47%
Chest problems	9%	Inc	Inc	Inc	9%

\*inc information not collected.

Table 6 gives information on mouthing behaviour and pica, a known risk factor for elevated blood lead concentrations. Pica for soil, sticks and matchsticks were of particular concern in Pella and Onseepkans.

**Table 6. PREVALENCE OF PICA AND MOUTHING BEHAVIOUR**

	<b>PELLA &amp; ONSEEPKANS</b>	<b>AGGENEYS</b>	<b>KIMBERLEY</b>	<b>CAPE TOWN</b>	<b>J'BURG</b>
<b>Sucking of fingers or nail chewing</b>	29%	35%	29%	24%	21%
<b>Lifetime observation of pica for:</b>					
<b>Paint</b>	4%	0%	4%	3%	4%
<b>Cement</b>	1%	0%	2%	3%	4%
<b>Soil</b>	15%	15%	19%	9%	16%
<b>Sticks</b>	14%	14%	28%	18%	27%
<b>Matchsticks</b>	11%	0%	24%	14%	21%
<b>Cigarette ends</b>	5%	5%	7%	8%	8%
<b>% of children ingesting non-food items at time of study</b>	4%	0%	15%	9%	15%

When asked, 36% of parents thought their children were more active than other children, while 13% described their children as “over-active”.

Table 7 gives information about parental awareness of lead hazards and levels of personal/domestic hygiene behaviours that might lead to reduced exposure to lead in the home environment. As can be seen, levels of awareness of lead as a health hazard were particularly low in Onseepkans, as well as in Pella, compared with Aggeneys.

**Table 7. LEVELS OF AWARENESS OF LEAD HAZARDS**

	PELLA	ONSEEPKANS	AGGENEYS	KIMBERLEY
<b>Proportion who thought exposure to lead could harm health</b>	39%	10%	67%	48%
<b>Proportion who damp dust</b>		77%	80%	65%
<b>Proportion who mop/damp sweep</b>		4%	21%	18%

Dry (rather than damp) dusting was practiced in 22% of Onseepkans/Pella homes, while in 96% dry sweeping was practiced.

### **3.1.2 Aggeneys (Lead Mining Town)**

A total of 21 first grade children from the only school in the lead mining town of Aggeneys were studied, where the population is predominantly Afrikaans-speaking. Relative to Pella and Onseepkans infrastructure, initiated by the mining company, is well developed in Aggeneys. On average Aggeneys homes were larger ( $p = 0.018$ ), with higher levels of access to basic environmental health services such as water, sanitation and electricity supplies, than homes in Onseepkans/Pella (see Table 3). In some respects however, there was evidence of housing degradation in Aggeneys. Paint, for example, was reported to be peeling from indoor and exterior surfaces in 48% and 43% of children's homes respectively, and the homes of 29% of study subjects were reported to be in need of major repairs.

Household income in the Aggeneys sample was high relative to that in the Pella/Onseepkans, as well as Kimberley samples (Table 4). In some homes work was undertaken, or hobbies practiced, that may have exposed children to lead in the home environment. Motor vehicle repair work, for example, was regularly undertaken at the homes of 14% of children, while electrical appliances were regularly repaired in the homes of a similar proportion of study subjects. No home use of lead solder, however, was reported.

Most study subjects (90%) were reported to be well at the time of the study, and 86% were reported to normally have a good appetite. The prevalence of ill health symptoms in Aggeneys children is given in Table 5, while Table 6 outlines the prevalence of pica and mouthing behaviour in the sample. As shown, levels of pica in Aggeneys were comparable with that determined in Pella and Onseepkans. When asked, 48% of parents thought their child was more active than other children, while 15% described their children as “over-active”.

Compared to Onseepkans/Pella, as well as Kimberley, a high proportion of Aggeneys respondents were aware of the link between lead exposure and detrimental health outcomes (Table 7). The higher level of lead awareness in Aggeneys may have been due to lead awareness campaigns targeted at mine, as part of the occupational hygiene programme that is in place at the mine.

### **3.1.3 Kimberley**

A total of 356 first grade children from four schools participated in the Kimberley study. Descriptive information about the Kimberley sample is given in Table 3,

while the household income distribution, relative to that in Aggeneys and Pella/Onseepkans, is given in Table 4. In Kimberley, a higher proportion of children spoke an African language or English, compared with their counterparts in Aggeneys or Pella/Onseepkans. Relative to Pella/Onseepkans and Aggeneys (where most children were described as Coloured), a higher proportion of the Kimberley sample identified their children as being Black African or White. Relative to Aggeneys and Pella/Onseepkans, a higher proportion of both mothers and fathers in the Kimberley reported having a tertiary education. Nonetheless, nearly half of the households in the Kimberley sample were earning less than R1 000.00 per month, which is a considerably higher proportion for this income category than in Aggeneys, but lower than in Pella/Onseepkans.

One-third of homes were reported to be in need of major repairs. Motor vehicle repair work was regularly undertaken at the homes of 10% of children, while spray painting was undertaken at the homes of 4%. A relatively high proportion (20%) of households were involved with fixing electrical appliances at home (which is likely to have involved the use of lead solder), and 6% of respondents reported that they made use of lead solder in the home environment.

Most study subjects (89%) were reported to be well at the time of the study. Table 5 outlines the prevalence of selected ill health symptoms in the study sample. Home remedies had been administered to 44% of children to treat illness or to improve their health. Most children (84%) were reported to normally have a good appetite.

### **3.1.4 Cape Town & Johannesburg**

Descriptive information on the Cape Town and Johannesburg samples are given in Tables 3, 5 and 6. The Cape Town and Johannesburg samples were similar in terms of mean age and household density, but more children in the Cape Town sample came from English-speaking homes and were described as Coloured. A similar proportion of Cape Town and Johannesburg mothers and fathers had a tertiary education, but in terms of secondary education, there was a higher level of achievement among both mothers and fathers in Johannesburg compared with Cape Town.

In respect of housing infrastructure more Cape Town than Johannesburg children had access to an indoor water supply, but fewer made use of an outdoor toilet. More Johannesburg relative to Cape Town dwellings were older than 25 years and similarly, more Johannesburg dwellings were reported to be in need of major repairs. In Cape Town the mean age of dwellings was 19.6 years, while the median number of rooms per dwelling was three, with up to fifteen rooms in a dwelling.

Similar proportions of households in Cape Town and Johannesburg made use of electricity for cooking, and the proportion of children who lived in dwellings with smokers was 52% for both Johannesburg and Cape Town. More Johannesburg respondents reported peeling paint from the interior and exterior walls of their homes than their counterparts in Cape Town.

Motor vehicle repair work to generate an income was regularly undertaken in the home environments of 7% of Cape Town study subjects, while in 3% of homes

spray painting was regularly done. In Johannesburg on the other hand, motor vehicle repair work was regularly undertaken at the homes of 12% of children, while spray painting was undertaken at the homes of 4%.

## **3.2 BLOOD LEAD DISTRIBUTIONS**

### **3.2.1 The Total Sample**

Blood samples were collected from a total of 1 287 first grade children from schools in Pella/Onseepkans, Aggeneys (the lead mining town), Kimberley, Cape Town and Johannesburg. The blood lead distribution for the total sample is given in Figure 2. The mean and median blood lead concentrations for the total sample was 7.4 and 6.9  $\mu\text{g}/\text{dl}$  respectively. Individual blood lead concentrations ranged from 1.0 to 44.4  $\mu\text{g}/\text{dl}$ . The blood lead distribution was positively skewed, with the five highest blood lead measurements equaling 20.4, 22.0, 22.6, 24.5 and 44.4  $\mu\text{g}/\text{dl}$ . Seventeen percent of the blood lead concentrations was or exceeded the internationally accepted action level of 10  $\mu\text{g}/\text{dl}$ , while 78% of measurements equalled or exceeded 5  $\mu\text{g}/\text{dl}$ .

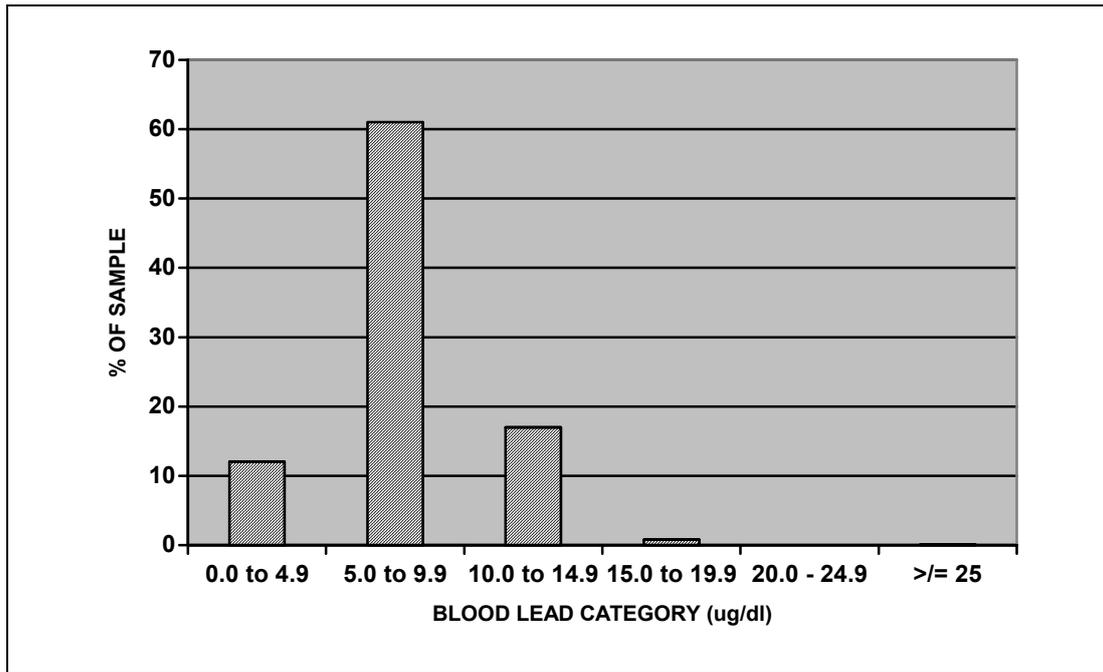
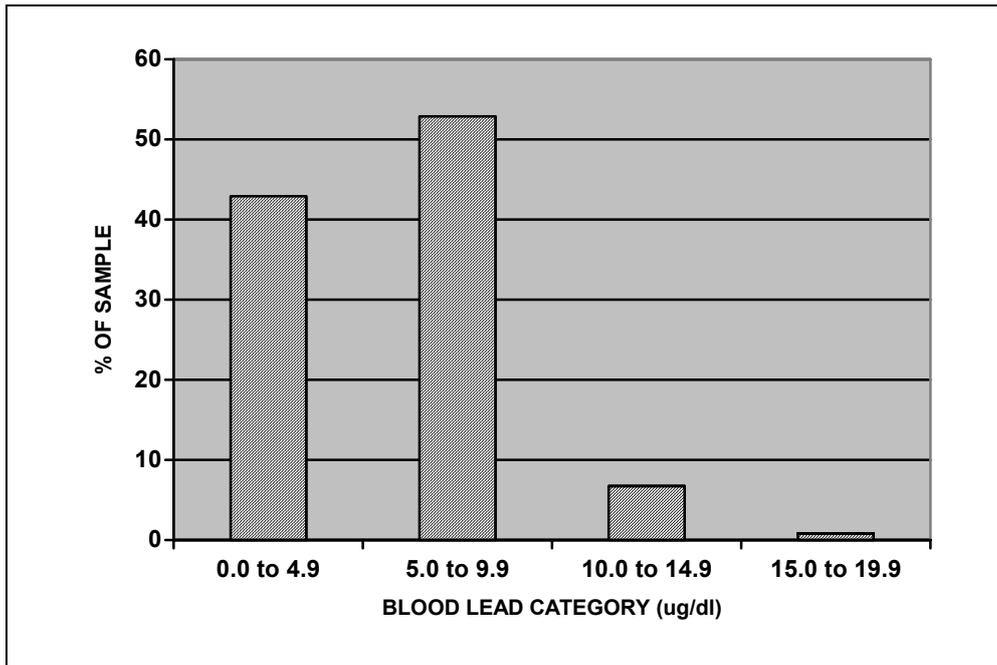


Figure 2. BLOOD LEAD DISTRIBUTION (TOTAL SAMPLE)

### 3.2.2 Rural Northern Cape (Onseepkans & Pella)

The blood lead distribution amongst the 98 study subjects from the remote rural Northern Cape towns of Pella and Onseepkans is given in Figure 3. Blood lead concentrations ranged from 2.4 to 17.1 µg/dl. The mean and median blood lead concentrations respectively were 6.1 and 5.5 µg/dl. Around 7.6% of the sample had blood lead concentrations equaling or exceeding 10 µg/dl, while in Onseepkans and Pella respectively 60% and 53% (56% in total) of blood lead concentrations was or exceeded 5 µg/dl.



**Figure 3. BLOOD LEAD DISTRIBUTION IN PELLA & ONSEEPKANS**

In Table 8 the blood lead distribution for Pella is compared with that for Onseepkans. As can be seen, the blood lead distributions in the two towns were similar, with the mean concentrations in both Pella (sd = 2.58) and Onseepkans (sd = 1.99) equaling 5.7  $\mu\text{g}/\text{dl}$ , while the median concentrations were 5.1  $\mu\text{g}/\text{dl}$  and 5.7  $\mu\text{g}/\text{dl}$  respectively.

**Table 8. BLOOD LEAD LEVELS PELLA & ONSEEPKANS ( $\mu\text{g}/\text{dl}$ )**

	<b>N</b>	<b>Median</b>	<b>Mean</b>	<b>sd</b>	<b>% <math>\geq 10</math> <math>\mu\text{g}/\text{dl}</math></b>	<b>Min.</b>	<b>Max.</b>
Pella	55	5.1	5.7	2.58	9	2.5	17.1
Onseepkans	43	5.7	5.7	1.99	2	2.4	12.1
<b>TOTAL</b>	<b>119</b>	<b>5.5</b>	<b>6.1</b>	<b>2.54</b>	<b>7.6</b>	<b>2.4</b>	<b>17.1</b>

As can be seen from Table 8 (& Figure 4), in Pella and Onseepkans respectively the proportion of study subjects with elevated blood lead ( $\geq 10$   $\mu\text{g}/\text{dl}$ )

concentrations was 9% and 2%. Using 5 µg/dl as a cut-off point, 53% and 60% of Pella and Onseepkans children respectively had high blood lead concentrations.

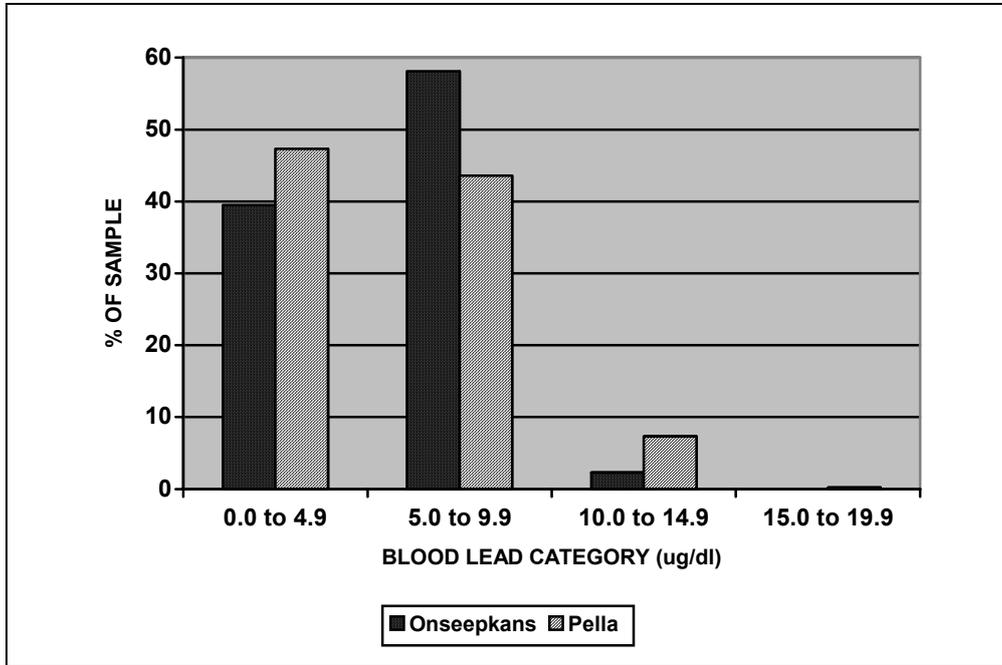
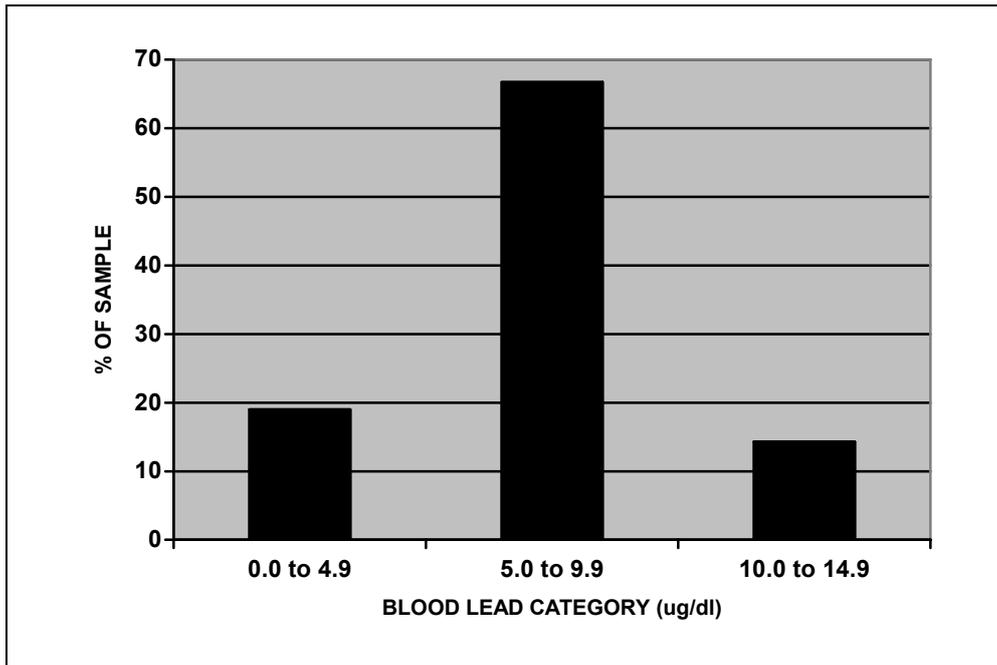


Figure 4. BLOOD LEAD DISTRIBUTION IN PELLA VERSUS ONSEEPKANS

### 3.2.3 The Lead Mining Town of Aggeneys

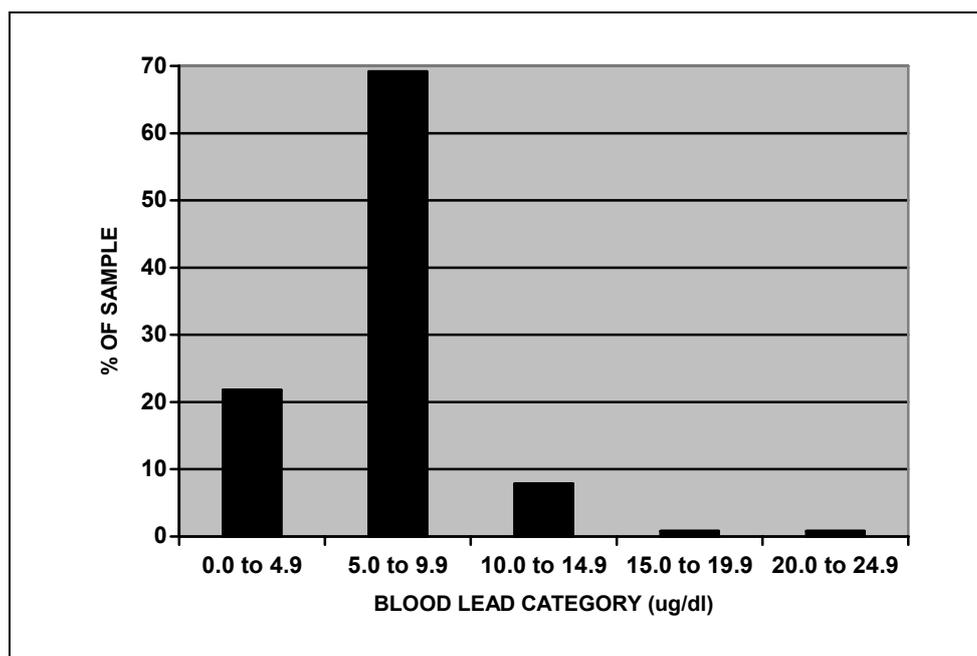
The blood lead distribution for 21 first grade subjects attending the only primary school in the lead mining town of Aggeneys is given in Figure 5. As can be seen, blood lead concentrations ranged from 2.8 to 13.4 µg/dl. The mean and median blood lead concentrations respectively was 7.8 (sd = 2.88) and 7.9 µg/dl. Just over 14% of the Aggeneys sample had blood lead levels equal to or greater than 10 µg/dl, while 81% had blood lead levels equaling or exceeding 5 µg/dl.



**Figure 5. BLOOD LEAD DISTRIBUTION IN AGGENEYS**

### **3.2.4 Kimberley**

Blood lead levels in the Kimberley sample of 355 first grade children from four Kimberley schools ranged from 2.1 to 22.6  $\mu\text{g}/\text{dl}$ , with the mean and median values respectively equaling 7.1 (sd 2.72) and 6.7  $\mu\text{g}/\text{dl}$ . The four highest blood lead concentrations measured in the Kimberley sample was 22.2, 22.0, 20.4 and 17.7  $\mu\text{g}/\text{dl}$ . The overall distribution of blood lead levels for the total Kimberley sample is given in Figure 6. The proportion of study subjects with blood lead levels equaling or exceeding 10  $\mu\text{g}/\text{dl}$  was 10%, while the proportion equaling or exceeding 5  $\mu\text{g}/\text{dl}$  was 79%.



**Figure 6. BLOOD LEAD DISTRIBUTION IN KIMBERLEY**

The mean and median blood lead concentrations by school in Kimberley are given in Table 9. Although the mean blood lead level measured at School 1 (Flamingo Primary school) (6.7 µg/dl) appeared lower than at the other schools studied, this difference did not reach statistical significance ( $p = 0.4161$ ). The proportion of subjects at individual schools with blood lead levels equaling or exceeding 10 µg/dl ranged from 6.8 to 13.7%.

**Table 9. BLOOD LEAD LEVELS BY SCHOOL – KIMBERLEY (µg/dl)**

	<b>N</b>	<b>Median</b>	<b>Mean</b>	<b>Sd</b>	<b>% <math>\geq</math> 10 µg/dl</b>	<b>Min.</b>	<b>Max.</b>
1. Flamingo	95	5.8	6.7	3.38	8.4	2.1	22.6
2. Olympic	95	6.7	7.2	2.99	13.7	3.2	20.4
3. Beacon	91	7.0	7.2	2.16	8.8	3.2	17.7
4. Endeavour	74	7.2	7.2	1.91	6.8	2.6	12.8
<b>TOTAL</b>	<b>355</b>	<b>6.7</b>	<b>7.1</b>	<b>2.72</b>	<b>9.6</b>	<b>2.1</b>	<b>22.6</b>

### 3.2.5 Cape Town

The distribution of blood lead concentrations amongst children in the Cape Town sample is given in Figure 7. Blood lead levels ranged from 1.0 to 24.5 µg/dl, with the mean for the total sample equaling 6.4 µg/dl, while the median was 6.1 µg/dl. The four highest blood lead concentrations in the Cape Town sample was 24.5, 18.7, 16.0 and 15.7 µg/dl.

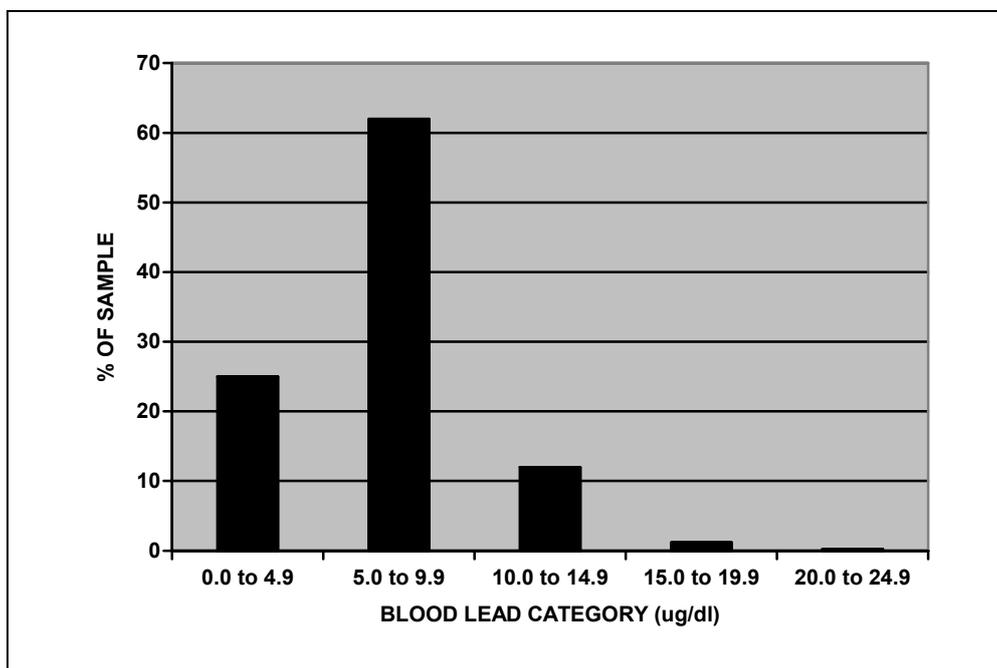


Figure 7. BLOOD LEAD DISTRIBUTION IN CAPE TOWN

The blood lead concentrations of 7% of the Cape Town sample exceeded the internationally accepted action level of 10 µg/dl, while 52% had blood lead levels  $\geq$  5 µg/dl. The blood lead concentration of 1% of the study sample was or exceeded 15 µg/dl.

There were significant differences in blood lead levels across the three suburbs of Woodstock, Mitchell's Plain and Hout Bay included in the Cape Town phase of the

study (see Table 10). The mean blood lead level amongst children attending Hout Bay schools (4.8 µg/dl) was significantly lower than amongst their counterparts attending schools in either Mitchell's Plain (6.9 µg/dl) or Woodstock (6.9 µg/dl) ( $p < 0.0001$ ). The proportion of subjects with blood lead levels equaling or exceeding 10 µg/dl was similarly lower in Hout Bay (3%) compared with either Woodstock (10%) or Mitchell's Plain (9%).

**Table 10. MEAN BLOOD LEAD LEVELS BY AREA - CAPE TOWN (µg/dl)**

	<b>N</b>	<b>Median</b>	<b>Mean</b>	<b>Sd</b>	<b>% ≥ 5 µg/dl</b>	<b>% ≥ 10 µg/dl</b>	<b>Min.</b>	<b>Max.</b>
Woodstock	182	6.8	7.3	2.88	56	10	1.0	18.7
Mitchell's Plain	94	6.4	6.9	3.11	64	9	2.0	24.5
Hout Bay	152	5.0	5.2	2.30	38	3	1.4	15.7
<b>TOTAL</b>	<b>428</b>	<b>6.1</b>	<b>6.4</b>	<b>2.90</b>	<b>52</b>	<b>7</b>	<b>1.0</b>	<b>24.5</b>

As can be seen from Figure 8, mean school blood lead concentrations varied considerably in Cape Town, ranging from 3.3 to 8.1 µg/dl ( $p = < 0.0001$ ). The lowest mean school blood lead concentrations were recorded in Hout Bay schools, whilst the highest mean concentrations were recorded in Woodstock.

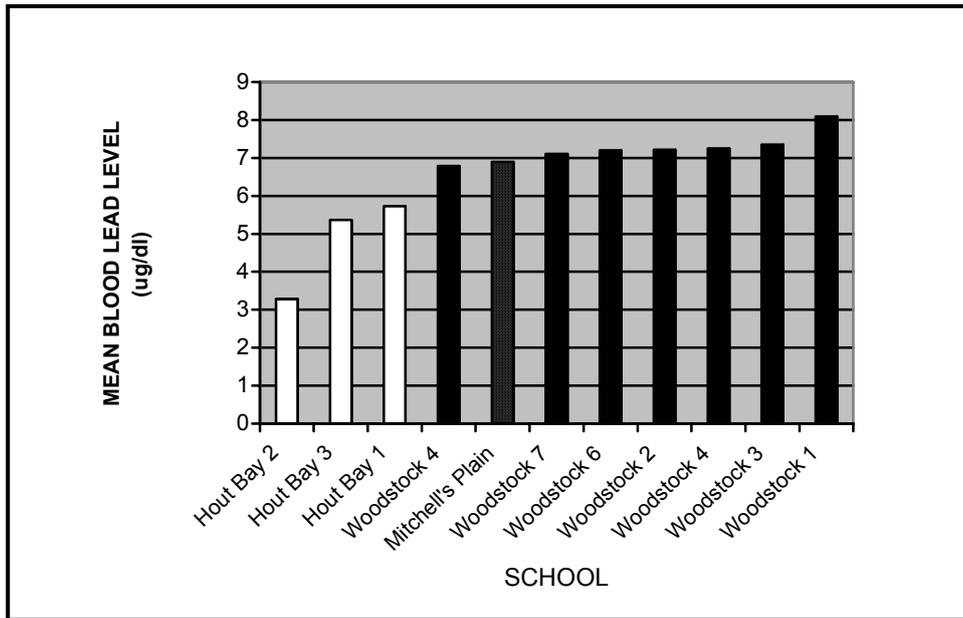


Figure 8. MEAN BLOOD LEAD LEVELS BY SCHOOL IN CAPE TOWN - 2002

Table 11 gives further information on the blood lead distribution by school.

Table 11. BLOOD LEAD LEVELS BY SCHOOL (CAPE TOWN) ( $\mu\text{g}/\text{dl}$ )

	n	Median	Mean	Sd	% $\geq 10 \mu\text{g}/\text{dl}$	% $\geq 5 \mu\text{g}/\text{dl}$	Min	Max
<b>WOODSTOCK</b>								
Woodstock 1	17	6.7	8.1	3.7	24%	88%	3.8	18.7
Woodstock 2	38	7.0	7.2	3.2	24%	74%	1.0	15.7
Woodstock 3	35	7.5	7.4	2.9	14%	71%	2.9	14.2
Woodstock 4	19	6.3	7.3	3.1	21%	79%	3.4	14.7
Woodstock 5	24	6.8	6.8	2.5	4%	79%	1.0	14.2
Woodstock 6	20	7.2	7.2	1.5	0%	85%	4.7	9.5
Woodstock 7	29	6.5	7.1	2.8	17%	83%	2.8	16.0
<b>MITCHELL'S PLAIN</b>								
Mitchell's Plain 1	94	6.4	6.9	3.1	12%	73%	2.0	24.5
<b>HOUT BAY</b>								
Hout Bay 1	58	5.3	5.7	2.3	2%	53%	2.5	15.7
Hout Bay 2	24	2.7	3.3	1.9	0%	21%	1.4	8.3
Hout Bay 3	70	5.1	5.4	2.2	4%	59%	1.4	11.6

As shown, the proportion of subjects with elevated blood lead concentrations at the level of the school ranged from 0 to 24%. Hout Bay schools recorded the lowest proportions of children with elevated blood lead concentrations, while those schools with the highest proportions of children with high blood lead levels were located in Woodstock.

### 3.2.6 Johannesburg

The blood lead distribution for the Johannesburg sample is given in Figure 9. The mean blood lead concentration for all children in the Johannesburg component of the study was 9.1 µg/dl, with individual results ranging from 1.1 to 44.4 µg/dl (sd = 3.59). The median blood lead concentration was 8.9 µg/dl. The five highest blood lead measurements were 44.4, 18.1, 17.6, 17.6, and 16.4 µg/dl.

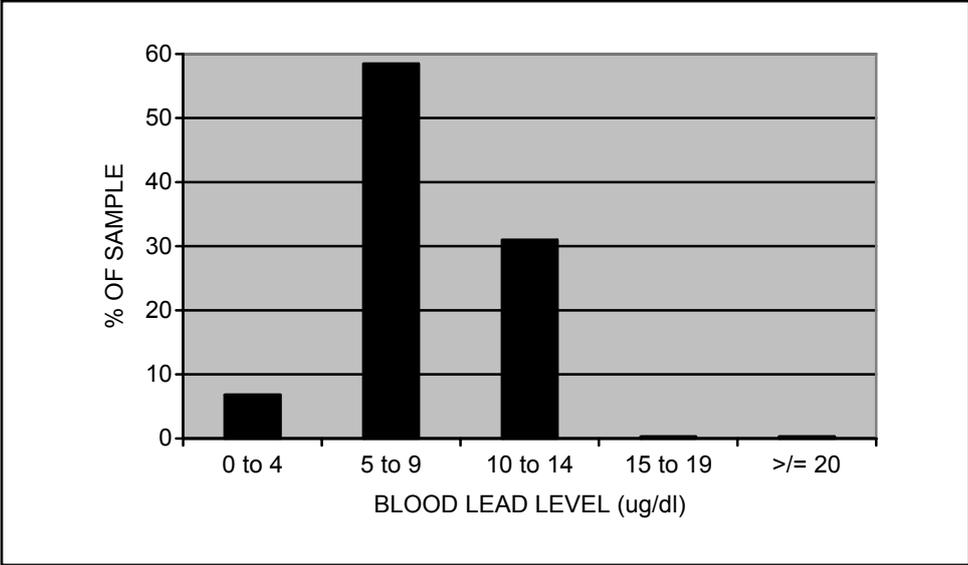


Figure 9. BLOOD LEAD DISTRIBUTION IN JOHANNESBURG, 2002

The study subject with the excessively high blood lead concentration of 44.4 µg/dl was investigated further. The findings are reported in a subsequent section.

However, for the purposes of further statistical analyses, this result was regarded as an outlier, and excluded.

The proportion of children with blood lead levels equal to or exceeding 10 µg/dl (the internationally accepted action level) was 35%, while the blood lead concentrations of 93% of the sample was equal to or exceeded 5 µg/dl, and 3% was equal to or exceeded 15 µg/dl.

As was the case for Cape Town, blood lead distributions varied significantly by area in the Johannesburg phase of the study, with the highest levels being determined in Alexandra and Westbury, and the lowest concentrations amongst children attending inner city schools (see Table 12). In all areas however, at least one-fifth of the study subjects had blood lead concentrations within the range of concern, putting them at risk of associated ill health and social effects.

**Table 12. SUBJECTS WITH ELEVATED BLOOD LEAD LEVELS BY AREA – JOHANNESBURG**

	N	Median	mean	SD	% $\geq$ 10µg/dl	Min	Max
Alexandra	89	10.4	10.4	2.89	53	3.2	17.6
Westbury/Riverlea	142	9.0	9.6	2.98	37	3.0	44.4
Soweto	56	7.6	8.2	2.45	21	3.5	13.7
Inner city	96	7.6	8.0	3.34	21	1.1	18.1
<b>TOTAL SAMPLE</b>	<b>383</b>	<b>8.9</b>	<b>9.1</b>	<b>3.59</b>	<b>35</b>	<b>1.1</b>	<b>44.4</b>

Blood lead concentrations also varied significantly by school ( $p = <0.0001$ ) in the Johannesburg sample. As can be seen from Table 13, the mean blood lead level at individual schools ranged from 6.9 µg/dl at a school in Soweto to 11.3 µg/dl at a

school in Alexandra. Median blood lead levels ranged from 6.8 µg/dl to 11.1 µg/dl. The proportion of subjects at the study schools with blood lead levels equal to or exceeding 10 µg/dl varied from 3% at a school in Soweto to 76% at a school in Alexandra.

**Table 13. BLOOD LEAD CONCENTRATIONS BY SCHOOL - JOHANNESBURG**

	<b>N</b>	<b>Median</b>	<b>Mean</b>	<b>SD</b>	<b>Min</b>	<b>Max</b>	<b>% &gt;10µ/dl</b>	<b>% &gt; 5µg/dl</b>
<b>ALEXANDRA</b>								
School 1	52	9.3	9.6	3.29	3.2	17.6	37	90
School 2	37	11.1	11.3	1.81	8.1	16.3	76	100
<b>WESTBURY</b>								
School 3	44	8.3	8.8	2.83	4.6	16.3	30	95
School 4	38	8.9	9.3	3.13	5.1	17.6	31	100
School 5	58	9.6	10.3	5.40	3.0	44.4	48	97
<b>SOWETO</b>								
School 6	31	7.0	6.9	1.56	3.5	10.2	3	90
School 7	25	9.8	9.9	2.34	5.5	13.7	58	100
<b>INNER CITY</b>								
School 8	42	6.8	7.4	2.90	3.5	15.1	14	83
School 9	27	7.8	8.0	3.30	2.7	15.7	22	100
School 10	27	8.4	8.8	3.92	1.1	18.1	30	100
<b>TOTAL</b>	<b>382</b>	<b>8.9</b>	<b>9.1</b>	<b>3.10</b>	<b>1.1</b>	<b>44.4</b>	<b>35</b>	<b>93</b>

Within each of the four Johannesburg areas selected for study, the highest school mean blood lead concentration was recorded at the school located closest to a busy road. For example, the mean blood lead concentration at a roadside school in Soweto was 9.9 µg/dl, which was considerably higher than at a school located further away (6.9 µg/dl). Four of the five schools with the highest mean blood lead concentrations were located on busy roads, while the fifth school was within one block of a heavily trafficked road.

### **3.3 RISK FACTORS FOR ELEVATED BLOOD LEAD LEVELS**

Mean blood lead levels were related to various population characteristics and known or suspected environmental risk factors for elevated blood lead concentrations.

#### **3.3.1 Rural Northern Cape (Pella & Onseepkans)**

As seen from Table 14, the mean blood lead level amongst boys and girls respectively were 5.8 and 5.6 µg/dl. This difference was not statistically significant ( $p = 0.629$ ). Blood lead concentrations were not significantly associated with age.

There was no association between housing type and blood lead concentration, nor was blood lead level associated with water source, location of toilet facilities, cooking fuel, the age of the house or the type of plumbing. However, children who lived in relatively crowded conditions tended to have higher blood lead concentrations ( $p = 0.034$ ) than other children. Exposure to environmental tobacco smoke was not significantly associated with elevated blood lead levels.

**Table 14. RISK FACTORS FOR ELEVATED BLOOD LEAD LEVELS BY STUDY SITE**

<b>RISK FACTOR</b>	<b>PELLA/ ONSEEPKANS</b>	<b>KIMBERLEY</b>	<b>CAPE TOWN</b>	<b>JOHANNESBURG</b>	<b>TOTAL SAMPLE</b>
<b>Sex</b>	0.629 <sup>1</sup>	0.5495	0.105	0.055	0.066
Male	5.8 <sup>2</sup>	7.2	6.7	9.5	7.5
Female	5.6 <sup>2</sup>	7.0	6.2	8.6	7.2
<b>Age</b>	0.912	0.293	0.0032 Each additional year associated with a reduction of 0.4 µg/dl in blood lead level	0.4949	0.014 Each additional year associated with a reduction of 0.4 µg/dl in blood lead level
<b>Population Group</b>	Analysis not undertaken due to small numbers of White and African Black study subjects	0.2247	0.0168	0.4006	<0.0001
Black African		6.8	6.0	8.9	7.5
Coloured		7.4	7.0	9.3	7.4
White		-	3.7	7.9	4.7
<b>Housing Type</b>	0.434	0.006	0.003	0.057	0.579
Informal	4.9	7.0	5.6	8.9	7.4
Other	5.8	7.5	6.7	10.4	7.2
<b>Age of House</b>	0.420	0.012	0.0713	0.1044	0.041
0 – 25 years	5.8	6.9	6.1	9.3	7.2
26 to 50 years	6.3	7.3	6.8	8.6	7.5
>50 years	4.5	9.6	7.3	7.4	7.7
<b>Number of People Living at Home</b>	0.034 Each additional person increases blood lead concentration by 0.2 µg/dl	0.096	0.0576 Each additional person increases blood lead concentration by 0.2 µg/dl	0.9649	0.002 Each additional persons increases blood lead concentration by 0.11 µg/dl
<b>Toilet</b>	0.388	0.0002	0.0694	0.4837	0.556
Indoors	5.0	6.5	6.7	8.8	7.4
Outdoors	5.8	7.5	5.8	9.3	7.3
<b>Water Source</b>	0.582	0.0003	0.1272	0.3318	0.876
Indoors	6.1	6.6	6.6	8.8	7.4
Outdoors	5.7	7.7	5.8	9.4	7.3
<b>Plumbing</b>	0.689	0.8139	0.0738	0.1239	0.001
Metal	5.4	6.9	6.6	8.9	7.6
Other	5.7	7.0	6.3	9.4	6.9
<b>Live near a Busy Road</b>	No heavily trafficked roads	0.9658	0.017	0.845	0.007
Yes		7.1	6.8	9.1	7.6
No		7.1	6.1	9.1	7.1
<b>Peeling Paint Indoors</b>	0.716	0.0103	0.6620	0.0154	<0.001
Yes	5.7	7.8	6.6	9.6	8.0
No	5.8	6.7	6.4	8.7	7.0
<b>No of Cars Owned by Household Members</b>	0.706	0.745	0.8585	0.3877	0.001 Each additional car increases blood lead level by 0.4 µg/dl
<b>Maternal Education</b>	0.459	0.253	0.1364	0.0054	<0.0001
No schooling	5.8	7.6	6.5	10.2	6.7
Primary School	6.1	7.1	6.9	9.2	6.9
High School	5.9	6.7	5.9	8.6	6.3
Tertiary	5.2	7.4	6.7	8.7	8.5
<b>Paternal Education</b>	0.826	0.282	0.600	0.4184	<0.0001
No schooling	5.4	7.5	6.3	9.8	6.5
Primary School	6.0	7.2	6.7	9.0	7.0
High School	5.3	6.7	6.2	8.9	6.4
Tertiary	5.7	7.3	6.6	9.0	8.1
<b>Paternal Occupation</b>	0.309	0.447	0.6209	0.1667	<0.0001
Professional	-	6.9	5.9	9.5	7.1
Skilled services	5.2	6.7	6.3	8.8	7.2
Unskilled	5.3	7.4	7.0	8.6	7.5
Armed forces	6.5	7.4	6.2	9.6	8.3
Unemployed	5.9	7.1	6.6	8.8	7.1
<b>Attended a Creche</b>	0.143	0.0077	0.8797	0.0083	<0.0001
Yes	5.6	6.9	6.4	8.7	7.1
No	6.9	8.0	6.5	9.9	8.6
<b>Mode of Travel to School</b>	0.210	0.3288	0.179	0.0320	<0.0001
Walk	5.6	7.5	6.6	9.3	7.7
Public transport	6.7	6.7	6.6	7.9	7.1
Private car	-	6.8	6.0	8.4	6.6
<b>Pica for Paint</b>	0.848	0.0072	0.152	0.0227	<0.0001
Yes	5.5	9.2	4.9	11.7	9.2
No	5.7	6.9	6.5	8.8	7.2
<b>Pica for Sand</b>	0.312	0.0080	0.910	0.0330	<0.0001
Yes	6.5	7.9	6.5	9.8	8.4
No	5.6	6.8	6.5	8.8	7.1
<b>Child sucks fingers</b>	0.328	0.0069	0.698	0.821	0.589
Yes	5.6	7.8	6.6	8.9	7.5
No	6.8	6.8	6.4	9.2	7.3
<b>Perception of Schoolwork</b>	0.622	0.3288	0.7025	0.0008	0.097
Good	5.5	6.8	6.3	8.5	7.2
Average/Poor	5.8	7.2	6.5	9.5	7.5

<sup>1</sup> P-value

<sup>2</sup> mean blood lead concentration

There was no statistically significant difference in blood lead levels by mode of transport used to travel to school. Blood lead levels were not higher amongst those repeating grade one, compared to those in grade one for the first time. There was no difference in the blood lead levels of children described as “over-active”, compared to others. There was also no difference in the blood lead concentrations of those who had attended a crèche compared with those who had not. Maternal education status was not associated with children’s blood lead levels ( $p=0.0025$ ).

### **3.3.2 Kimberley**

Although the mean blood lead concentration at one of the four Kimberley schools studied was lower ( $6.7 \mu\text{g/dl}$ ) than the mean concentration at the other four schools ( $7.2 \mu\text{g/dl}$ ), this difference did not reach statistical significance ( $p = 0.4161$ ). The mean blood lead levels amongst boys and girls respectively were  $7.2$  and  $7.0 \mu\text{g/dl}$ . This difference was not statistically significant ( $p = 0.5495$ ). Blood lead concentrations were not significantly associated with age, or population group ( $p = 0.2247$ ).

Blood lead levels were not higher amongst children living in formal housing relative to informal. However children who lived in older houses had higher blood lead concentrations than other children ( $p = 0.012$ ). Blood lead levels were also higher ( $7.5 \mu\text{g/dl}$ ) amongst those who made use of an outside toilet at home relative to those who used an indoor toilet ( $6.5 \mu\text{g/dl}$ ) ( $p = 0.0002$ ). Similarly, blood lead concentrations were higher amongst those who had an outdoor ( $7.7 \mu\text{g/dl}$ ) relative to an indoor ( $6.6 \mu\text{g/dl}$ ) water supply ( $p = 0.0003$ ). Peeling indoor paint was

associated with elevated blood lead concentrations ( $p = 0.0103$ ), as was exterior peeling paint ( $p = 0.0355$ ).

The number of residents in the dwelling was not associated with blood lead concentrations (0.096). Living near a busy road was not associated with elevated blood lead levels. There was a marginal association between the presence of a smoker in the home and blood lead concentrations ( $p = 0.0724$ ).

Blood lead levels were not associated with the mode of travel to school. Blood lead levels were not higher amongst those repeating grade one, compared to those in grade one for the first time. There was no difference in the blood lead levels of children described as more active or “over-active”, compared to others. Blood lead levels were lower amongst children who had attended a crèche (6.9  $\mu\text{g}/\text{dl}$ ) compared with those who had not (8.0  $\mu\text{g}/\text{dl}$ ) ( $p = 0.0077$ ). Children whose parents reported that they did not suck their fingers had lower blood lead concentrations (6.8  $\mu\text{g}/\text{dl}$ ) than those who were reported to suck their fingers (7.8  $\mu\text{g}/\text{dl}$ ) or whose parents were unaware of whether or not their children sucked their fingers (7.3  $\mu\text{g}/\text{dl}$ ) ( $p = 0.0069$ ). Those who had been observed to ingest paint chips had higher blood lead levels (9.2  $\mu\text{g}/\text{dl}$ ) than those who had not (6.9  $\mu\text{g}/\text{dl}$ ) ( $p = 0.0072$ ). Similarly, those who ingested soil had higher blood lead concentrations (7.9  $\mu\text{g}/\text{dl}$ ) than children who did not (6.8  $\mu\text{g}/\text{dl}$ ) ( $p = 0.008$ ).

After taking account of potential confounding factors such as maternal and paternal education and occupation, blood lead concentrations remained significantly

associated with dwelling type, the age of the house, water source, the presence of peeling paint indoors and mouthing behaviour (see Table 15).

### **3.3.3 Cape Town**

Mean blood lead concentrations varied significantly by school ( $p = <0.0001$ ), and ranged from 3.3 to 8.1  $\mu\text{g}/\text{dl}$ . The mean blood lead level amongst boys and girls respectively were 6.7 and 6.2  $\mu\text{g}/\text{dl}$ . This difference was not statistically significant. Older children had lower blood lead concentrations than younger children ( $p = 0.003$ ), with each additional year of age resulting in a reduction in blood lead concentration of 0.4  $\mu\text{g}/\text{dl}$ .

Looking at blood lead levels in relation to South Africa's apartheid-based population groups, the mean blood lead level amongst White children was significantly lower (3.7  $\mu\text{g}/\text{dl}$ ) than amongst their Coloured (7.0  $\mu\text{g}/\text{dl}$ ) or Black African (6.0  $\mu\text{g}/\text{dl}$ ) counterparts ( $p = 0.0168$ ). Amongst White, Coloured and Black African children respectively, 4%, 17% and 9% had blood lead levels equal to or exceeding 10  $\mu\text{g}/\text{dl}$ . Blood lead concentrations were not associated with maternal or paternal educational or occupational status.

In terms of housing conditions, children living in informal dwellings tended to have lower blood lead levels (5.6  $\mu\text{g}/\text{dl}$ ) than those living in formal structures (6.7  $\mu\text{g}/\text{dl}$ ) ( $p = 0.003$ ). Blood lead concentrations increased as the age of the house increased. Relative to those who lived in the newest dwellings (6.1  $\mu\text{g}/\text{dl}$ ), those whose parents could not recall the age of their dwelling had the highest blood lead concentrations (7.2  $\mu\text{g}/\text{dl}$ ) ( $p = 0.037$ ). There was a marginal association between

living in a home with an indoor toilet and higher blood lead levels ( $p = 0.0694$ ). Blood lead levels were not associated with water source. The type of fuel used for cooking was not associated with blood lead levels. Children who lived in houses with metallic plumbing had slightly higher blood lead levels than other children ( $p = 0.0738$ ). Blood lead levels were higher among children whose parents could not recall whether paint was peeling from the interior walls, doors or windowsills ( $8.3 \mu\text{g}/\text{dl}$ ) relative to those who reported that there was ( $6.6 \mu\text{g}/\text{dl}$ ) or was not ( $6.3 \mu\text{g}/\text{dl}$ ) peeling interior paint ( $p = 0.031$ ). Blood lead concentrations were not associated with peeling paint; either indoors ( $p = 0.6620$ ) or outdoors ( $p = 0.8810$ ). Blood lead concentrations were associated with household size, with each additional household member contributing  $0.2 \mu\text{g}/\text{dl}$  to blood lead concentration ( $p = 0.0576$ ). Children whose homes were located on or within one block of a busy road ( $6.8 \mu\text{g}/\text{dl}$ ) had higher blood lead levels than others ( $6.1 \mu\text{g}/\text{dl}$ ) ( $p = 0.017$ ).

Blood lead levels did not vary by mode of transport used to travel to school. Blood lead levels were not higher amongst those repeating grade one, compared to those in grade one for the first time. There was no difference in the blood lead levels of children described as more active or “over-active”, compared to others. There was no difference in the blood lead concentration of those who had attended a crèche compared with those who had not. Maternal or paternal educational or occupational status was not significantly associated with blood lead level.

After taking account of potential confounding factors in a multivariate regression model, the child's age, population group and crowding remained significantly associated with blood lead level (see Table 15).

### 3.3.4 Johannesburg

Blood lead concentrations differed significantly by school in Johannesburg ( $p < 0.0001$ ). The mean blood lead concentration among boys was 9.5  $\mu\text{g/dl}$ , and was higher than that amongst girls (8.6  $\mu\text{g/dl}$ ) ( $p = 0.055$ ). Blood lead level did not vary significantly by age. There was no statistically significant difference between the blood lead levels of Black African and Coloured children. Children's blood lead levels were associated with maternal (but not with paternal) educational ( $p = 0.0054$ ) and occupational ( $p = 0.0383$ ) status.

Children who lived in dwellings with peeling exterior ( $p = 0.0243$ ) or interior ( $p = 0.0154$ ) paint had higher blood lead concentrations than other children. Blood lead concentrations were higher amongst children who walked or cycled to school, relative to those who used public transport ( $p = 0.003$ ). Blood lead concentrations were not associated with the number of motor vehicles owned by household members.

The perceptions of respondents regarding children's schoolwork were significantly associated with blood lead concentrations ( $p = 0.0008$ ). Blood lead levels were significantly lower amongst children who had attended a creche/playschool prior to primary school (8.7  $\mu\text{g/dl}$ ), compared with those who had not (9.9  $\mu\text{g/dl}$ ) ( $p = 0.0083$ ). Blood lead levels were higher amongst children who had been observed to ingest paint chips ( $p = 0.0227$ ) or soil ( $p = 0.0330$ ), compared with those who had not.

After taking account of potential confounding factors in a multiple regression analysis, blood lead concentrations in Johannesburg children remained significantly associated with gender ( $p = <0.001$ ), attending a crèche or pre-school institution ( $p = 0.030$ ), the presence of peeling paint indoors ( $p = 0.010$ ) and pica for paint ( $p = 0.001$ ) (see Table 15).

### **3.3.5 The Total Sample**

In terms of the total sample of 1 287 children from all of the five study sites, blood lead levels were marginally associated with gender ( $p = 0.066$ ). Each additional year of age was associated with a reduction of 0.4  $\mu\text{g}/\text{dl}$  in blood lead concentration. Blood lead levels amongst White children were significantly lower than in their Black African or Coloured counterparts ( $p = <0.0001$ ). Both maternal ( $p = <0.0001$ ) and paternal educational status ( $p = <0.0001$ ) was associated with children's blood lead concentrations. Paternal, but not maternal, occupational status was linked to blood lead levels ( $p = <0.0001$ ).

Children living in older dwellings tended to have higher blood lead levels than other children ( $p = 0.041$ ). Crowding was associated with blood lead concentration; each additional person increased blood lead concentration by 0.11  $\mu\text{g}/\text{dl}$  ( $p = 0.002$ ). Children whose homes were fitted with metallic plumbing had higher blood lead levels than other children ( $p = 0.001$ ). Children who lived in houses with peeling interior ( $p = <0.001$ ) or exterior paint (0.072) had higher blood lead concentrations than other children. The number of cars owned by household members was

associated with blood lead levels; each additional car increased blood lead level by 0.4 µg/dl.

Children who had attended a crèche or pre-school institution had lower blood lead levels than other children ( $p = <0.0001$ ). Children who traveled to school by private car had lower blood lead concentrations than other children ( $p = <0.0001$ ). Children with pica for paint and sand were highly significantly associated with elevated blood lead concentrations ( $p = <0.0001$ ).

Taking account of potential confounding factors in a multiple regression analysis, blood lead levels in the total sample remained significantly associated with the study site, population group, crowding, the presence of peeling paint indoors, having attended a creche, pica for sand, and marginally associated with pica for paint (see Table 15).

**Table 15. ANALYSES OF THE ADJUSTED (FINAL LINEAR REGRESSION MODELS)  
ASSOCIATIONS BETWEEN RISK FACTORS AND ELEVATED BLOOD LEAD LEVELS:  
KIMBERLEY, CAPE TOWN, JOHANNESBURG AND THE TOTAL SAMPLE**

<b>Risk Factor</b>	<b>Adjusted Association</b>
<b>Kimberley</b>	
Dwelling Type	P = 0.006 (the blood lead levels of children who lived in backyard dwellings were 2.75 µg/dl higher than those who lived in free-standing dwellings)
Age of House	P = <0.001 (children living in dwellings >25 years old had higher blood lead levels than those living in dwellings <25 years old)
Water Source	P = 0.002 (the blood lead levels of children who used an indoor tap were 3.09 µg/dl lower than those who used other water sources)
Peeling paint indoors	P = 0.011 (the blood lead levels of those who lived in homes with peeling paint was 2.54 µg/dl higher than those who did not)
Child sucks fingers	P = 0.005 (the blood lead levels of children who did not suck their fingers were 2.78 µg/dl lower than those who did)
<b>Cape Town</b>	
Child's Age	P = 0.292 (with each additional year blood lead levels declined by 1.05 µg/dl)
Population Group	P <0.001 (The blood lead levels of Coloured children were 3.76 µg/dl higher than African Black children)
No of people living in house	P = 0.176 (each additional person contributed an increase of 1.35 µg/dl of lead in blood)
<b>Johannesburg</b>	
Sex	P = 0.001 (blood lead levels of girls were 3.18 µg/dl lower in girls than boys)
Peeling paint indoors	P = 0.010 (the blood lead levels of children who lived in homes with peeling paint were 2.58 µg/dl higher)
Attended a creche	P = 0.030 (the blood lead levels of children who did not attend a crèche were 2.17 µg/dl higher)
Pica for paint	P = <0.001 (the blood lead levels of children who had pica for paint were 5.52 µg/dl higher than those who did not)
<b>Total Sample</b>	
Study Site	P = <0.0001 (the blood lead levels of Johannesburg children were 5.69 µg/dl higher than Cape Town children) P = 0.040 (the blood lead levels of children from Onseepkans/Pella were 2.05 µg/dl lower than children from Cape Town)
Population Group	P = 0.002 (the blood lead levels of Coloured children were 3.04 µg/dl higher than that of Black African children) P = 0.002 (the blood lead levels of White children were 3.11 µg/dl lower than Black African children)
No of people living in dwelling	p = 0.008 (each additional person resulted in an increase of 2.63 µg/dl in the study child)
Peeling paint indoors	P = 0.004 (the blood lead levels of children who lived in homes with peeling paint were 2.89 µg/dl higher than those who did not)
Attended a crèche	P = 0.003 (the blood lead levels of children who had not attended a crèche were 3.02 µg/dl higher than those who had)
Pica for sand	P = 0.002 (the blood lead levels of children who had pica for sand were 3.06 µg/dl higher than those who did not)
Pica for paint	P = 0.081 (the blood lead levels of children with pica for paint were 1.75 µg/dl higher)

### 3.4 GEOGRAPHICAL COMPARISON OF BLOOD LEAD DISTRIBUTIONS IN SELECTED STUDY SITES

After taking account of the effect of clustering, mean blood lead levels varied significantly by study site ( $p < 0.001$ ) (see Figure 10). Blood lead levels were highest in the Johannesburg sample, where concentrations significantly exceeded those measured in Cape Town ( $p < 0.001$ ), Kimberley ( $p < 0.001$ ), Pella ( $p < 0.001$ ) and Onseepkans ( $p < 0.001$ ). Although the mean blood lead concentration in the Johannesburg sample was 1.4  $\mu\text{g}/\text{dl}$  higher than in Aggeneys, this difference did not reach statistical significance.

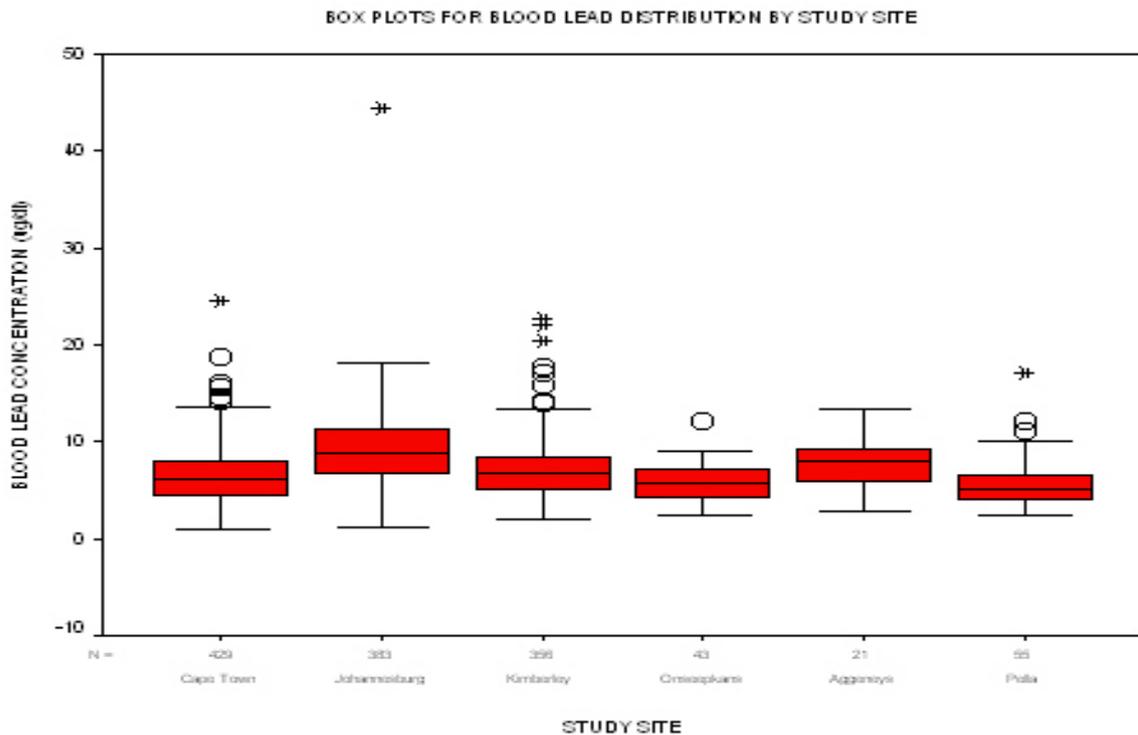


Figure 10. BOX PLOTS FOR BLOOD LEAD DISTRIBUTION BY STUDY SITE

Further information comparing blood lead distributions in Kimberley, Cape Town and Johannesburg is given in Table 16. As can be seen, the mean and median blood concentrations in Johannesburg, which equalled 9.1 and 8.9 µg/dl respectively, were significantly higher than in the other two urban centres (Cape Town and Kimberley).

**Table 16. COMPARISON OF BLOOD LEAD LEVELS IN KIMBERLEY, CAPE TOWN & JOHANNESBURG (µg/dl)**

	<b>N</b>	<b>Median</b>	<b>Mean</b>	<b>sd</b>	<b>% &gt; 10 µg/dl</b>	<b>Min.</b>	<b>Max.</b>
<b>KIMBERLEY</b>	355	6.7	7.1	2.72	10	2.1	22.6
<b>CAPE TOWN</b>	429	6.1	6.4	2.90	10	1.0	24.5
<b>JOHANNESBURG</b>	382	8.9	9.1	3.10	35	1.0	44.4
<b>TOTAL</b>	<b>1168</b>	<b>7.1</b>	<b>7.5</b>	<b>3.31</b>	<b>18</b>	<b>1.0</b>	<b>44.4</b>

The proportion of children with blood lead levels equal to or exceeding 10 µg/dl in Johannesburg (35%) was also higher than in Cape Town (10%) and Kimberley (10%).

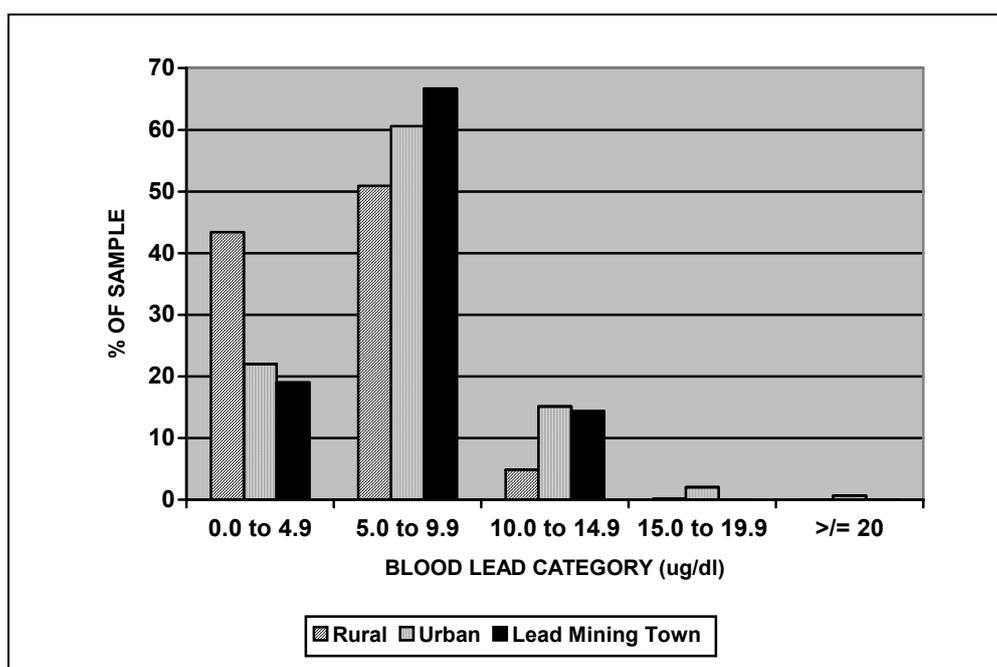
Despite the difference in population size between the two cities, there was no statistically significant difference in the mean blood lead levels of the Cape Town and Kimberley samples ( $p = 0.211$ ).

As expected the lowest blood lead levels were determined in the rural sites of Onseepkans & Pella. In Onseepkans ( $p = 0.064$ ) and Pella ( $p = 0.079$ ) blood lead levels were around 2 µg/dl lower than in the lead mining town of Aggeneys. In Table 17 it can be seen how the mean blood lead concentrations, and proportions of subjects with elevated blood lead levels varied across the three towns.

**Table 17. BLOOD LEAD LEVELS BY TOWN – RURAL NORTHERN CAPE (µg/dl)**

	N	Median	Mean	Sd	% > 10 µg/dl	Min.	max.
AGGENEYS	21	7.9	7.8	2.88	14.3	2.8	13.4
PELLA	55	5.1	5.7	2.58	9.1	2.5	17.1
ONSEEPKANS	43	5.7	5.7	1.99	2.3	2.4	12.1
<b>TOTAL</b>	<b>119</b>	<b>5.5</b>	<b>6.1</b>	<b>2.54</b>	<b>7.6</b>	<b>2.4</b>	<b>17.1</b>

In Figure 11 the blood lead distributions in the urban sites (Kimberley, Cape Town and Johannesburg) are compared with the rural sites of Pella and Onseepkans, as well as with, separately, Aggeneys.



**Figure 11. BLOOD LEAD DISTRIBUTIONS IN URBAN VERSUS RURAL SITES**

Table 18 gives further information comparing the blood lead distributions in the rural and urban samples. As can be seen the mean blood lead concentration of children in the rural sample was significantly lower than in either the urban ( $p = <0.0001$ ) or the lead mining ( $p = 0.0270$ ) samples. The mean blood lead

concentration in the Aggeneys (7.9 µg/dl) sample was higher even than the mean value of 6.4 µg/dl measured in the Cape Town sample, though lower than the value of 9.1 µg/dl determined amongst Johannesburg children in the study.

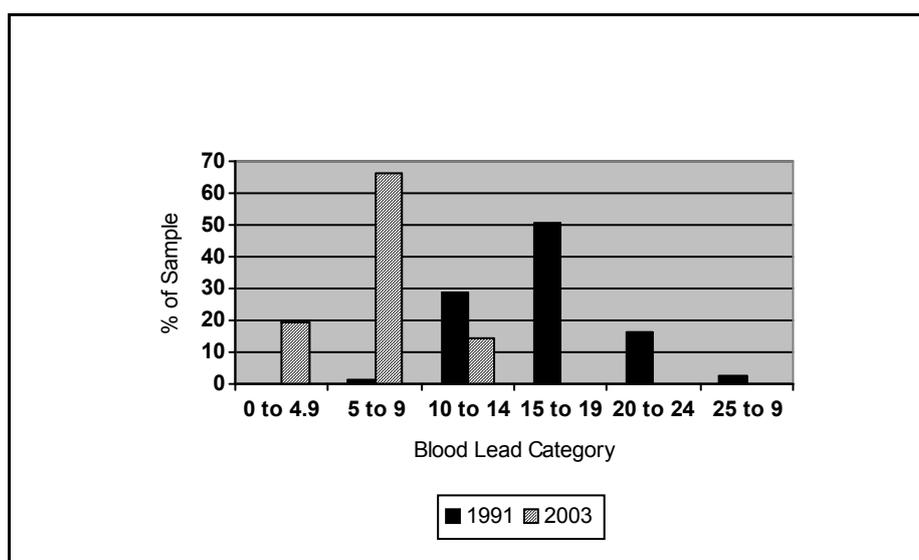
**Table 18. COMPARISON OF BLOOD LEAD LEVELS IN URBAN & RURAL SITES (µg/dl)**

	<b>N</b>	<b>Median</b>	<b>Mean</b>	<b>Sd</b>	<b>% &gt; 10 µg/dl</b>	<b>Min.</b>	<b>Max.</b>
<b>URBAN SAMPLE</b>	1166	7.1	7.5	3.31	18	1.0	44.4
<b>RURAL SAMPLE</b>	98	5.2	5.5	2.33	8	2.4	17.1
<b>LEAD MINING TOWN</b>	21	7.9	7.8	2.88	14	2.8	13.4
<b>TOTAL</b>	<b>1 285</b>	<b>7.4</b>	<b>6.9</b>	<b>3.27</b>	<b>17</b>	<b>1.0</b>	<b>44.4</b>

### 3.5 TEMPORAL ANALYSES

#### 3.5.1 Aggeneys & Pella

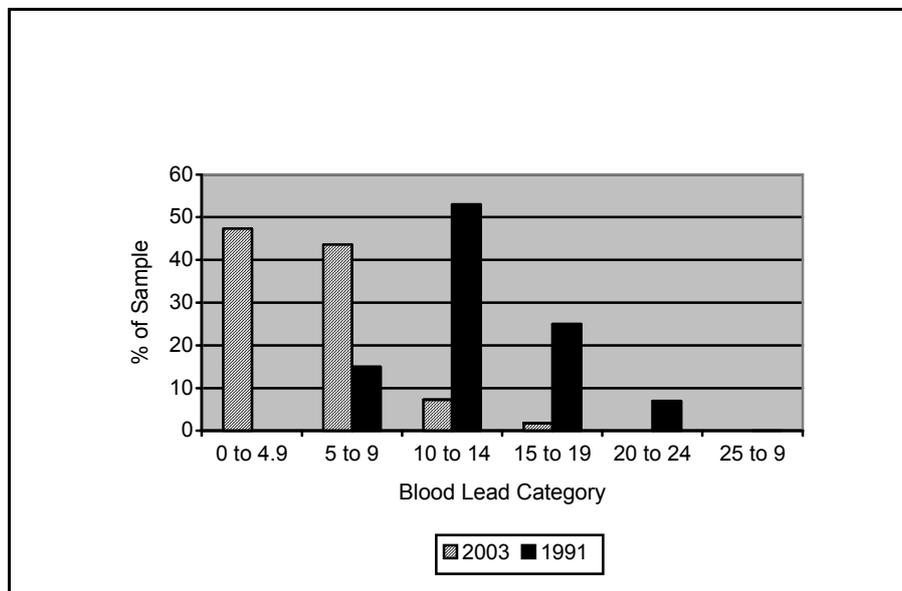
In Figure 12 the blood lead distribution in Aggeneys during 1991 is compared with that obtained during 2003.



**Figure 12. BLOOD LEAD DISTRIBUTION IN AGGENEYS – 1991 VERSUS 2003**

In 1991 blood lead levels in Aggeneys ranged from 9 to 27.5  $\mu\text{g}/\text{dl}$ , and averaged around 16  $\mu\text{g}/\text{dl}$ . Ninety-eight percent of the sample had blood lead levels equalling or exceeding 10  $\mu\text{g}/\text{dl}$ . In the current study blood lead concentrations in the Aggeneys sample ranged from 2.8 to 13.4  $\mu\text{g}/\text{dl}$ , with the mean and median levels respectively equalling 7.8 and 7.9  $\mu\text{g}/\text{dl}$ . The proportion of subjects with blood lead levels of 10  $\mu\text{g}/\text{dl}$  or higher in 2003 was 14%.

Figure 13 compares the blood lead distribution in Pella during 1991 with that obtained during 2003. In 1991 the mean blood lead level in Pella children was 13  $\mu\text{g}/\text{dl}$ , with 85% of subjects having blood lead levels equalling or exceeding 10  $\mu\text{g}/\text{dl}$ . Individual blood lead concentrations ranged from 6 to 22  $\mu\text{g}/\text{dl}$ .



**Figure 13. BLOOD LEAD DISTRIUBTION IN PELLA – 1991 VERSUS 2003**

### 3.5.2 Cape Town

In Figure 14 the blood lead distribution determined in the 1991 study in the Cape Town suburb of Woodstock is compared with that obtained in the same suburb during 2002. As can be seen, relative to 1991, the distribution for 2002 is distinctly shifted to the left.

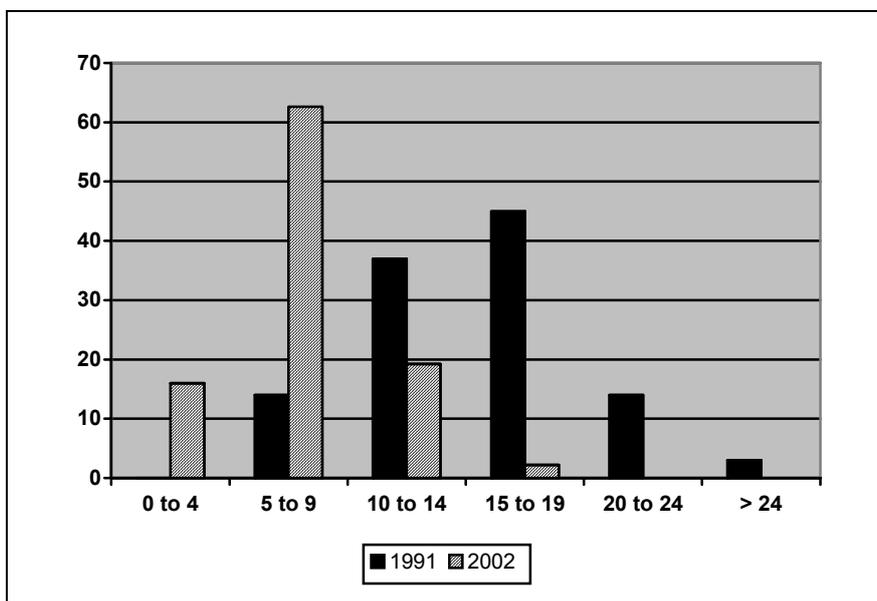


Figure 14. BLOOD LEAD DISTRIBUTION IN WOODSTOCK – 1991 VERSUS 2002

A further breakdown of the mean blood lead concentrations, and the proportions of children with elevated blood lead levels by area and population group, in 1991 and 2002, is included in Table 19. As can be seen, the proportion of children with elevated blood lead levels decreased substantially between 1991 and 2002. In Woodstock for example, the proportion of children with elevated blood lead levels decreased from 99% in 1991 to 12% in 2002. In Hout Bay, the proportion of children with high blood lead levels similarly decreased from 95% to 3%. In both 1991 and 2002 the mean blood lead concentration was lower in the relatively

sparsely populated and less trafficked suburb of Hout Bay, than in either Woodstock or Mitchell's Plain, though the margin of difference in 2002 was distinctly greater than in 1991.

**Table 19. COMPARISON OF BLOOD LEAD LEVELS IN CAPE TOWN – 1991 & 2002**

		1991		2002	
		Mean	% $\geq$ 10 $\mu$ g/dl	Mean	% $\geq$ 10 $\mu$ g/dl
AREA	Woodstock	16	99	6.9	12
	Mitchell's Plain	15	93	6.9	12
	Hout Bay	14	95	4.8	3
POPULATION GROUP	Black African	16	100	6.0	9
	Coloured	16	98	7.0	17
	White	14	96	3.5	4

Reductions in blood lead distributions were also observed by population group in 2002 relative to 1991. For example, as can be seen in Table 19, whereas 98% of Coloured children had elevated blood lead levels in 1991, this proportion was reduced to 22% in 2002.

The mean blood lead concentration in this group was also reduced from 16  $\mu$ g/dl in 1991 to 7  $\mu$ g/dl in 2002. Similarly, the proportion of Black African and White children with elevated blood lead concentrations declined from 100% to 9% and from 96% to 4% respectively between 1991 and 2002.

As shown in Table 20, which gives mean school blood lead concentrations for the eleven Cape Town schools included in both the 1991 and 2002 surveys, declines in blood lead concentrations were observed at all the study schools.

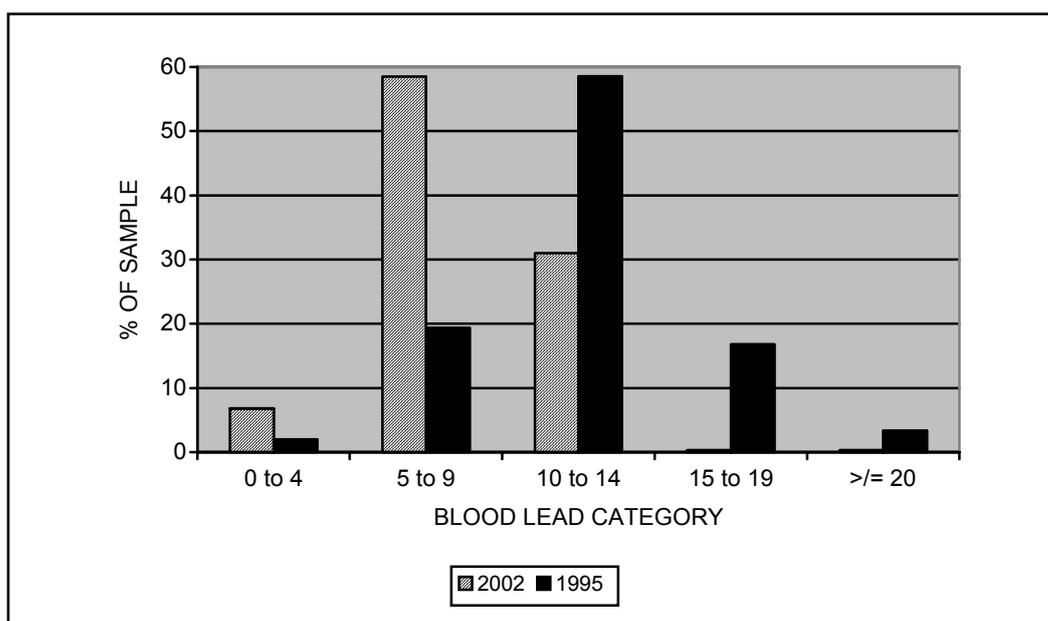
**Table 20. MEAN SCHOOL BLOOD LEAD LEVELS ( $\mu\text{g}/\text{dl}$ ) (CAPE TOWN 1991 & 2002)**

SUBURB	SCHOOL	1991		2002	
		LEAD LEVEL	N	LEAD LEVEL	N
Woodstock	7	13	43	8.1	17
	1	16	68	7.2	38
	4	16	14	7.1	29
	8	16	25	7.2	20
	10	17	19	7.3	19
	11	17	37	6.8	24
	2	19	37	7.4	35
Mitchell's Plain	9	15	104	6.9	94
Hout Bay	6	13	70	5.7	58
	3	14	25	3.3	24
	5	14	20	5.4	70

Adjusting for the effect of school, the lead level in the 1991 Cape Town survey was on average  $8.7 \mu\text{g}/\text{dl}$  higher than in the 2002 survey ( $p < 0.0001$ ) [95% confidence interval for the difference (7.57; 9.75)].

### 3.5.3 Johannesburg

In Figure 15 the blood lead distribution obtained during 1995, when the first survey was conducted in Johannesburg, is compared with that obtained during 2002. As was the case for Cape Town, a distinct shift to the left was observed in blood lead distributions in the latter relative to the earlier survey.



**Figure 15 BLOOD LEAD DISTRIBUTION IN JOHANNESBURG – 1995 & 2002**

Table 21 gives further details of the changes in blood lead levels by school in Johannesburg between 1995 and 2002. As can be seen, as was the case in Pella, Aggeneys and Cape Town, reductions in mean blood lead concentrations were observed in all Johannesburg schools.

**Table 21. COMPARISON OF BLOOD LEAD LEVELS BY SCHOOL IN JOHANNESBURG (1995 & 2002)**

SUBURB	SCHOOL	1995		2002	
		LEAD LEVEL	N	LEAD LEVEL	N
Inner city	21	10.8	32	7.4	42
	23	12.2	66	8.0	27
	22	11.9	43	8.8	27
Westbury	25	12.2	96	9.3	39
	24	11.3	94	10.3	59
Alexandra	27	13.2	32	9.6	52
	28	11.9	70	11.3	37

Adjusting for the effect of school and key confounding factors, there is overwhelming evidence ( $p = <0.0001$ ) of a decrease in blood lead concentrations in Johannesburg school children between 1995 and 2002. The average decrease in blood lead level was 2.79  $\mu\text{g}/\text{dl}$  with a 95% confidence interval of 1.87 - 3.88.

Reductions in children's blood lead concentrations were greater in Cape Town, where, on average, blood lead levels declined by 8.7  $\mu\text{g}/\text{dl}$  between 1991 and 2002, compared with Johannesburg, where blood lead levels declined by only 2.5  $\mu\text{g}/\text{dl}$  over the period between 1995 and 2002.

### **3.6 SUPPLEMENTARY INVESTIGATIONS**

A number of supplementary investigations were conducted to further examine some of the key findings to emerge from the blood lead analyses and the questionnaire data. These included:

- a preliminary survey of lead concentration in samples of soil, dust and paint collected from the study schools,
- a case study of the subject with the highest blood lead concentration (a girl from the suburb of Westbury in Johannesburg),
- an investigation of potential sources of lead in the homes of the twenty Johannesburg subjects with the highest blood lead concentrations,
- a survey of paint lead concentrations in selected homes from a randomly selected sample of Johannesburg suburbs, stratified by age of proclamation, and

- an analysis of the lead concentrations in paint samples purchased from paint stores.

### 3.6.1 School Environmental Sampling

In order to obtain a preliminary indication of the environmental sources of lead in the school environment, samples of water, soil and paint were collected from the grounds of the study schools and analyzed for their lead content.

#### **Water**

The guideline level of the World Health Organization (WHO) for the concentration of lead in water equals 10 µg/litre. As can be seen from Table 22 the lead concentrations measured in samples of water taken from all of the study schools were within the WHO guideline levels.

**Table 22. LEAD CONCENTRATIONS IN SCHOOL WATER SAMPLES**

<b>AREA</b>	<b>N</b>	<b>RANGE (µg/g)</b>	<b>% ≥ 10 µg/litre</b>
<b>Rural Northern Cape</b>	4	0.2 – 1.2	0%
<b>Johannesburg</b>	10	0.7 – 3.5	0%
<b>Cape Town</b>	11	0.0 – 0.5	0%
<b>Kimberley</b>	4	0.0 – 0.2	0%
<b>ALL AREAS</b>	<b>26</b>	<b>0.0 – 1.2</b>	<b>0%</b>

#### **Soil**

Soil samples were collected from the main playground at each of the study schools. The Department of Housing and Urban Development in the United States of America has adopted a standard of 400 ppm for lead in soil in areas where

children play most of the time. As can be seen from Table 23, the lead concentrations in all of the soil samples collected were below 400 ppm.

**Table 23. LEAD CONCENTRATIONS IN SCHOOL SOIL SAMPLES**

<b>AREA</b>	<b>N</b>	<b>RANGE (µg/g)</b>	<b>% ≥ 400 µg/g</b>
<b>Rural Northern Cape</b>	4	2.8 – 35.2	0%
<b>Kimberley</b>	4	5.1 – 231.1	0%
<b>Cape Town</b>	11	1.5 – 198.0	0%
<b>Johannesburg</b>	10	35.8 – 231.1	0%
<b>ALL AREAS</b>	29	1.37 – 231.1	0%

### **Paint**

As part of ageing, degradation or normal weathering processes, or in the case of flaking or peeling, lead particles may be released from lead-based paint used to decorate homes, schools and other buildings. In this study the lead based paint standard of the Department of Housing and Urban Development of the United States of America (5000 µg/g) was used as a threshold value. The results of analyses of the lead content of paint samples taken from the walls of grade one classrooms at the study schools is given in Table 24. As can be seen, paint lead concentrations ranged from 1.4 to 35 434 µg/g, and in 28% of the paint samples collected, lead concentrations exceeded the reference value.

**Table 24. LEAD CONCENTRATIONS IN SCHOOL PAINT SAMPLES**

<b>AREA</b>	<b>N</b>	<b>RANGE (µg/g)</b>	<b>% ≥ 5 000 µg/g</b>
<b>Rural Northern Cape</b>	4	88.0 – 23 222	25%
<b>Kimberley</b>	4	119.3 – 18 833	25%
<b>Cape Town</b>	11	236.0 – 35 434	36%
<b>Johannesburg</b>	10	1.4 – 8 653	20%
<b>ALL AREAS</b>	29	1.4 – 23 222	28%

The two samples with the highest paint lead concentrations were taken from Cape Town schools. All of the four schools in the Cape Town sample with excessive paint lead concentrations were located in the suburb of Woodstock.

In the Northern Cape the highest school paint lead concentrations were measured in Onseepkans (23 221  $\mu\text{g/g}$ ), whilst the sample with the second highest concentration was taken from a school in Kimberley (18 883  $\mu\text{g/g}$ ).

In Johannesburg the highest paint lead concentrations were from paint samples taken from schools in Westbury (7 626  $\mu\text{g/g}$ ) and in Alexandra (8 653  $\mu\text{g/g}$ ).

### **3.6.2 Case Study of Subject with Elevated Blood Lead Concentration**

The subject with the highest blood lead concentration (44.4  $\mu\text{g/dl}$ ) was a 7-year old girl from the Johannesburg suburb of Westbury. Her blood lead level was considerably higher than the mean Johannesburg sample concentration of 9.1  $\mu\text{g/dl}$ . Analysis of a repeat blood sample taken one month later, showed an increase in her blood lead concentration to 51.5  $\mu\text{g/dl}$ ; more than five times higher than the internationally accepted action level of 10  $\mu\text{g/dl}$ .

During follow-up home interviews conducted with the parents, it emerged that the girl was often observed to ingest putty, paint from indoor and outdoor walls, and soil from around the apartment building in which they lived. Concern over their daughter's pica tendency and health, had previously prompted the parents to take

her to a local hospital for investigation. However, over-exposure to lead had not been considered.

Subsequent analyses of lead concentrations in samples of paint, putty, soil and water taken from the girl's home and surrounding areas, showed that lead levels in water were below detectable levels, while soil lead concentrations ranged from 11.4 to 87.5  $\mu\text{g/g}$ . The levels of lead in paint samples taken from the walls of the girl's home were high, ranging up to 46 000  $\mu\text{g/g}$  (compared to the reference level of 5 000  $\mu\text{g/g}$ ), as was the lead concentration in a paint sample taken from a classroom of the school she attended (7 626  $\mu\text{g/g}$ ). A subsequent medical examination revealed evidence of microcytic anaemia.

### **3.6.3 Home Investigations - Johannesburg Children With Elevated Blood Lead Levels**

The results of the analyses of the lead content of environmental samples (paint, soil and dust) collected from the homes of the twenty children with the highest blood lead levels in the Johannesburg component of the study are given in Table 25. As can be seen, at least one sample of lead based paint was found in 6 (32%) of the 19 homes from which paint samples were collected. The mean lead concentration in all of the paint samples was 3 900  $\mu\text{g/g}$ , with individual results ranging from 100 to 46 000  $\mu\text{g/g}$ .

The lead concentrations in dust samples collected from the windowsills of nineteen of the twenty homes (one home had no windowsills) ranged from 150 to 2 400

$\mu\text{g}/\text{ft}^2$ , while the mean concentration was 801  $\mu\text{g}/\text{ft}^2$ . The majority (79%) of windowsill dust lead concentrations exceeded the reference level of 250  $\mu\text{g}/\text{ft}^2$ .

Five (25%) of the 20 homes had floor dust lead concentrations greater than or equal to the reference value of 40  $\mu\text{g}/\text{ft}^2$ , and the mean floor dust lead concentration was 80  $\mu\text{g}/\text{ft}^2$ .

The mean lead concentration in bare soil samples was 152.3 ppm, and only one of the eighteen homes with areas of bare soil was found to contain elevated lead concentrations.

**Table 25. LEAD CONCENTRATIONS IN HOME ENVIRONMENTAL SAMPLES**

Sample Type	N	% cases $\geq$ reference level	Median (all samples)	Mean (all samples)	Std Dev (all samples)	Range (all samples)	Reference Level*
Paint ( $\mu\text{g}/\text{g}$ )	19	32	3 000	3 900	0.916	100 – 46 000	5 000
Windowsill dust ( $\mu\text{g}/\text{ft}^2$ )	19	79	470	800.53	690.905	15 – 2 400	250
Floor dust ( $\mu\text{g}/\text{ft}^2$ )	20	25		80.44	158.363	3.4 – 640	40
Soil from principle play area (ppm)	18	6	49	152.28	233.611	40 – 1 000	400
Bare soil area (ppm)	19	0		139.16	187.179	40 - 860	1200

\* Reference levels of the United States Environmental Protection Agency and the Housing and Urban Development Department.

In Table 26 the findings that emerged from the home visits are presented in relation to the individual children. Data collected through questionnaire administration showed that in four homes a household member was involved in a lead-related activity, which included lead soldering, painting and motor vehicle repairs. Eleven children (55%) in the study sample lived in homes that were located within one block from a busy road. Two of the children were reported to currently have pica for paint chips, while 5 (25%) had been observed to ingest paint chips in the past. The majority of children (90%) were reported to often have

dirty or dusty hands and fingernails. As shown in Table 26, the highest paint lead concentrations were measured in the homes of the two study children with the highest blood lead concentrations.

**Table 26. PREVALENCE OF RISK FACTORS FOR ELEVATED BLOOD LEAD LEVELS\***

Blood lead level (µg/dl)	Maximum paint lead level (µg/g)	Windowsill dust lead level (µg/ft <sup>2</sup> )	Play area soil lead level (ppm)	Lives within 1 block of busy road	Household member involved in lead-related activity	Pica for paint
44.4	46 000	820	120	No	No	Yes
18.1	43 000	320	320	Yes	Painting	-
17.6	2 000	1 100	130	No	No	No
17.6	11 000	1 300	110	Yes	No	No
16.4	7 000	790	40	Yes	No	No
16.3	2 000	320	45	Yes	NO	No
16.3	100	2 200	40	Yes	Lead soldering	No
16.0	3 000	210	42	Yes	No	No
15.9	300	330	48	No	No	No
15.9	100	150	40	Yes	No	-
15.7	18 000	1 700	1 000	Yes	No	No
15.7	3 000	330	370	No	Car repairs	No
15.6	300	2 400	-	No	No	No
15.1	-	200	40	No	No	No
14.9	3 000	160	40	Yes	No	Yes
14.9	3 000	1 000	200	Yes	No	No
14.9	3 000	-	-	-	No	No
14.7	500	470	50	No	Painting	No
14.6	4 000	1 100	42	No	No	No
14.4	6 000	310	64	Yes	No	No

\*Risk factors exceeding reference levels are highlighted

### 3.6.4 Study of Residential Paint Lead Concentrations - Johannesburg

In light of evidence emerging from this study that points to lead based paint as an important source of lead exposure amongst South African children, it was decided to conduct a further survey of the lead concentrations in paint samples collected from Johannesburg dwellings. Following verbal consent from a household member, 316 paint samples were collected from 239 dwellings in sixty randomly selected

suburbs (stratified by age) across the City of Johannesburg. Suburbs were stratified into those established between 1901 and 1947 (during which white lead was used in paint); 1948 and 1977 (when the use of white lead was discontinued, but other forms of lead continued to be used in paint) and 1978 to the present day (a voluntary agreement to limit the addition of lead to paint was meant to be in force amongst members of the South African Paint Manufacturers' Association [SAPMA]). The expectation was that while elevated lead concentrations might be found in paint samples collected from suburbs proclaimed during the earlier two periods, few or none of the samples from the period 1978 to the present day would contain high lead levels.

The results of laboratory analyses of the lead content of the paint samples collected, are given in Table 27. As can be seen, of the 316 paint samples collected, 17% contained levels of lead equal to or greater than 5 000 µg/g (the USA reference level). The mean lead concentration found in all of the samples was 474 µg/g, with individual sample lead concentrations ranging from 10 to 29 000 µg/g. Paint samples were collected from a total of 239 homes, of which 48 (20%) contained at least one lead based paint sample. In 34 (57%) of the 60 suburbs included in the study, a lead based paint sample was collected from at least one home. There was little difference in the proportion of homes with lead-based paint across the three suburb age groups, with suburbs proclaimed in 1901-1947, 1948-1978 and 1979-present respectively having 50%, 60% and 60% of dwellings with lead-based paint.

**Table 27. PAINT LEAD CONCENTRATIONS IN GREATER JOHANNESBURG ( $\mu\text{g/g}$ )**

	% LEAD BASED PAINT	MEAN	STD DEVIATION	RANGE
All Samples (n = 316)	17	474	1 886	10 – 29 000
All Homes (n = 239)	20 <sup>1</sup>	419	1 262	10 – 15 250 <sup>3</sup>
All Suburbs (n = 60)	55 <sup>2</sup>	459	951	10 – 6 600 <sup>4</sup>
Suburbs Proclaimed 1901–1947 (n=20)	50 <sup>2</sup>	305	83	10 – 5 700
Suburbs Proclaimed 1948 –1978 (n=20)	60 <sup>2</sup>	545	124	10 – 7 400
Suburbs Proclaimed 1979–present (n=20)	60 <sup>2</sup>	594	301	10 – 29 000

<sup>1</sup> % of homes containing at least one lead based paint sample

<sup>2</sup> % of suburbs containing at least one home with lead based paint

<sup>3</sup> Range of the mean lead concentration for each individual home

<sup>4</sup> Range of the mean lead concentration for each of the suburbs

### 3.5.5 Analysis Of Lead Content of Off-The-Shelf Paint Samples

Following strong indications (from the case studies, as well as from the results of residential paint lead analysis) of an important and current role for lead-based paint in childhood lead exposure in South Africa, samples of enamel paint for residential use were purchased from the shelves of paint stores in Johannesburg (the selected stores were located in wealthy as well as low-income suburbs) and Cape Town, and analysed for lead content. The samples purchased included well known as well as lesser known brands.

The results of the lead paint content analysis showed that paint lead concentrations ranged from not detectable to 189 000  $\mu\text{g/g}$ . Of the 42 paint samples collected, 60% was lead based. The lead content of all samples of white enamel paint were within the HUD standard of 5 mg/g (5 000  $\mu\text{g/g}$ ) In respect of other colours of paint however, high lead levels were widespread, in some cases up to thirty-eight times as high as the reference level. In total, 83% on the non-white colours of paint were lead based.

There was no apparent variation by site of purchase in relation to lead based paint. Paint with a high lead content was purchased in Cape Town as well as Johannesburg, and within Johannesburg from wealthy, middle-income as well as low-income suburbs. Lead in high concentrations was found in popular as well as lesser known brands of enamel paint.

Only 2 of the 25 samples of lead based paint displayed stickers warning potential purchasers about the high lead content of the paint.

### 3.5.6 Lead Content of Paint on Children's Toys and Coloured Pencils

Relative to the internationally accepted standard for lead in children's toys of 90 µg/g, paint lead concentrations up to 135 903 µg/g were measured in the sample of toys purchased for this preliminary scanning study (see Figure 28). Toys with a high lead content were available from major toy stores as well as from craft shops and flea markets. The highest lead concentrations were measured in the paint coating toys manufactured in South Africa, but elevated lead concentrations were also found in the paint on toys imported from Thailand and China.

**Table 28. RESULTS OF LEAD CONTENT ANALYSIS OF PAINTED, WOODEN CHILDREN'S TOYS**

CODE	COLOUR	DESCRIPTION	POINT OF PURCHASE	COUNTRY OF ORIGIN	PAINT LEAD CONCENTRATION (µg/g)
2A	Yellow	Building blocks	Toy Cave, Eastgate	Unknown	97 326 <sup>1</sup>
2B	Red	Building blocks	Toy Cave, Eastgate	Unknown	71 573
2C	Green	Building blocks	Toy Cave, Eastgate	Unknown	31 608
2D	Blue	Building blocks	Toy Cave, Eastgate	Unknown	692
4A	Yellow	Building blocks	Checkers	Unknown	<1.0
4B	Red	Building blocks	Checkers	Unknown	<1.0
5A	Yellow	Building blocks	Checkers	Germany	<1.0
5B	Red	Building blocks	Checkers	Germany	<1.0
14A	Yellow	Building blocks	Reggie's	China	66
14B	Red	Building blocks	Reggie's	China	<1.0
17A	Yellow	Airplane	Amberley Craft Market, Hurlingham	South Africa	113 823

17B	Red	Airplane	Amberley Craft Market, Hurlingham	South Africa	513
18A	Yellow	Building blocks	Play & School Room	China	347
18B	Red	Building blocks	Play & School Room	China	115
19A	Yellow	Shapes	Play & School Room	Thailand	27 621
19B	Red	Shapes	Play & School Room	Thailand	<1.0
20A	Yellow	Painted wooden blocks	ToysRUs	South Africa	<1.0
20B	Red	Painted wooden blocks	ToysRUs	South Africa	134 264
21A	Yellow	Painted wooden blocks	ToysRUs	South Africa	135 903
21B	Red	Building blocks	ToysRUs	South Africa	93 518
22A	Yellow	Building blocks	Squirrel Nutkin	South Africa	93 252
22B	Red	Building blocks	Squirrel Nutkin	South Africa	94 259
23A	Yellow	Building blocks	Private source	Unknown	46 775
23B	Red	Building blocks	Private source	Unknown	25

<sup>†</sup> Shaded areas indicate concentrations exceeding the internationally accepted standard of 90 µg/g for lead in toys.

\*Laboratory Analyses undertaken by the National Institute for Occupational Health

Somewhat elevated lead concentrations were also measured in the paint coating coloured pencils available from major South African stores (see Table 28).

**Table 29. LEAD CONCENTRATIONS IN PAINT COATING COLOURED PENCILS**

CODE	COLOUR	POINT OF PURCHASE	COUNTRY OF ORIGIN	PAINT LEAD CONCENTRATION (µg/g)
3A	Yellow	Toy Cave, Eastgate	Costa Rica	425
3B	Red	Toy Cave, Eastgate	Costa Rica	67
6A	Yellow	Checkers	South Africa	125
6B	Red	Checkers	South Africa	<1.0
7A	Yellow	Checkers	South Africa	<1.0
7B	Red	Checkers	South Africa	<1.0
8A	Yellow	Checkers	Brazil	<1.0
8B	Red	Checkers	Brazil	<1.0
9A	Yellow	Checkers	Unknown	53
9B	Red	Checkers	Unknown	62
15A	Yellow	CAN	Germany	57
15B	Red	CAN	Germany	<1.0
16A	Yellow	CAN	Brazil	<1.0
16B	Red	CAN	Brazil	<1.0

## Chapter 4: DISCUSSION

### 4.1 GEOGRAPHICAL VARIATIONS IN BLOOD LEAD DISTRIBUTIONS

This study has shown how, across and within the selected study sites and samples, blood lead levels varied significantly.

#### 4.1.1 Rural Areas

As was expected, the lowest blood lead concentrations in this survey were measured in the rural Northern Cape towns of Onseepkans and Pella, where the mean blood lead concentration was 6.1  $\mu\text{g}/\text{dl}$  (median blood lead concentration was 5.5  $\mu\text{g}/\text{dl}$ ) and 7.6% of the children studied had blood lead levels equaling or exceeding 10  $\mu\text{g}/\text{dl}$ . Nevertheless, relative to certain rural children elsewhere, the blood lead distribution amongst Onseepkans & Pella children may be considered to be high. For example, a study conducted amongst children in the rural Malaysian town of Setiu found that the mean blood lead level was 2.5  $\mu\text{g}/\text{dl}$  (Hashim et al 2000). A study conducted among rural native American communities in the state of Montana in the United States of America showed that the mean blood lead concentration in children aged one to five years was 2.4  $\mu\text{g}/\text{dl}$  (Howell & Russette 2004). In Sweden, following the removal of lead from petrol, blood lead levels of children now average around 2.1  $\mu\text{g}/\text{dl}$  (Stromberg et al 2003).

The blood lead concentrations in Onseepkans and Pella children are more comparable with those determined in rural settings of some developing countries.

For example a Senegalese study undertaken in rural children showed a mean blood lead concentration of 6.1 µg/dl (Diouf et al 2003). It is possible that the higher than expected blood lead levels of children in Onseepkans and Pella emanate from naturally high soil lead concentrations in the area, lead in water or from sources such as lead based paint.

#### **4.1.2 Lead Mining Town**

In the lead mining town of Aggeneys, the mean blood lead concentration was 7.9 µg/dl, and 14% of the children studied had blood lead levels equalling or exceeding 10 µg/dl. The mean blood lead concentration in Aggeneys children was significantly higher than in children from the non-mining towns of Pella and Onseepkans located around 40 to 100 kilometres away ( $p = 0.003$ ). Blood lead levels in Aggeneys were elevated despite a significantly higher socio-economic profile in this community relative to the impoverished communities of Onseepkans & Pella.

It is notable that the mean blood lead concentration amongst Aggeneys children was similar to that measured in children sampled from the City of Johannesburg, and significantly higher than the mean level measured in their urban counterparts from the cities of Cape Town and Kimberley. Even children from the heavily trafficked inner city Cape Town suburb of Woodstock (mean level = 6.9 µg/dl), expected to be amongst the highest risk groups for elevated blood lead levels, had lower blood lead concentrations than children from Aggeneys.

The blood lead levels amongst Aggeneys children were also high when compared with children from certain lead mining areas elsewhere in the world. For example, in the Swedish mining and milling town of Falun, where lead contamination levels are considered to be high, the mean blood lead level amongst 33 children was 3.1 µg/dl (Bjerre et al 1993). A study undertaken in the USA showed a mean blood lead level of 6.5 µg/dl in children from a lead mining area, compared with 3.4 µg/dl amongst children in a non-lead mining area. Mining waste, as well as lead-based paint, was considered to be the cause of elevated blood lead levels in children living in the lead mining area (Murgueytio et al 1998a).

The blood lead levels of Aggeneys children were comparable with what was measured in 431 children aged 7 years in the Upper Silesian Industrial Zone of Katowice in Poland, which is well recognized for the magnitude of environmental problems, and where a study showed that the mean blood lead concentration was 7.9 µg/dl (Zejda et al 1995).

A high precision lead isotope study undertaken in Australia to evaluate the pathway of lead dust from a lead mine to the homes of employees showed that blood lead levels in the children of mine employees (5.7 µg/dl) were higher than in other children (4.1 µg/dl). The authors demonstrated that the houses of the lead mine employees could be contaminated by mine lead transported home on the clothes, shoes, hair, skin, and in some cases motor vehicles, of the workers. In one case, dust shaken from clothes of a mine employee contained 3 000 ppm of lead, 100% of which was determined through isotopic analyses to emanate from the lead mine. Lead dust was transported from the mine into the homes of workers despite

precautions taken by mine employees to minimize transportation of lead into their houses (Chiaradia et al 1997).

A further study, conducted to evaluate the relationship between lead mining waste and childhood lead poisoning, looked at 125 children between the ages of 6 and 71 months living in a lead mining area of south western Missouri (United States of America), showed that homes in the lead mining area had significantly increased soil and dust lead levels and that children there had significantly higher blood lead levels. The primary source of dust lead was soil lead. There was a strong relationship between blood lead levels and dust, soil, and paint lead. The authors concluded that soil lead from mining operations played a significant role in children's blood lead levels (Murgueytio et al 1998b).

In a study undertaken in 1991, in which potential pathways of exposure to lead amongst Aggeneys children were examined, it was shown that children whose fathers bathed on arriving home, had lower blood lead concentrations than those who showered at work. The authors postulated that workers in more dusty occupations within the mine were more likely to have been required to shower at work in accordance with occupational hygiene measures, while workers holding "cleaner" jobs were likely to be leaving work in their own clothing and showering or bathing at home (von Schirnding et al 2003). Showering at work may also have been insufficient to reduce the lead load on workers' skin and clothing, with subsequent transfer of lead particles into the home environment and lead exposure amongst children.

The findings of the current study lead to a conclusion that despite their economically advantaged status (relative to children from Onseepkans and Pella), children from Aggeneys are at risk of lead exposure from local mining activity, and that escalated attention is warranted in Aggeneys to investigate in detail, the sources and pathways of childhood exposure to lead, and develop a plan of action to reduce blood lead levels locally and in lead mining towns elsewhere in South Africa. Such a plan of action should include, amongst other aspects, a public campaign to increase the proportion of people who are aware of the health hazards of lead (and the mechanisms to reduce lead exposure), increased attention to the stringency with which occupational hygiene measures are applied amongst employees of the lead mine and blood lead screening amongst children.

Para-occupational lead exposure is also a concern in relation to non-mining industries. For example, a study undertaken of children with a parent who worked in a radiator repair workshop compared with those not occupationally exposed to lead, showed that dust lead levels in the homes of exposed children were significantly elevated. Higher blood lead levels were measured in children whose fathers worked in a home-based radiator repair workshop (22.4 µg/dl) compared with an external workshop (14.2 µg/dl). The lowest blood lead concentrations were recorded in children whose parents were not occupationally exposed to lead (5.6 µg/dl) (Aguilar-Garduno et al 2003).

#### **4.1.3 Urban Areas**

Mean blood lead levels among the samples of children included in the urban study sites of Cape Town, Kimberley and Johannesburg respectively were 7.1 µg/dl

(9.6%  $\geq$  10  $\mu\text{g}/\text{dl}$ ), 6.4  $\mu\text{g}/\text{dl}$  (7%  $\geq$  10  $\mu\text{g}/\text{dl}$ ) and 9.1  $\mu\text{g}/\text{dl}$  (35%  $\geq$  10  $\mu\text{g}/\text{dl}$ ). The blood lead levels among urban South African children are high in comparison with levels amongst children elsewhere. For example, a study undertaken in West and East Germany showed that blood lead levels there were generally low, with geometric means equalling between 3.9  $\mu\text{g}/\text{dl}$  and 5.1  $\mu\text{g}/\text{dl}$  in the western German and between 4.2  $\mu\text{g}/\text{dl}$  6.8  $\mu\text{g}/\text{dl}$  in the eastern German study areas (Begerow et al 1994). A Finnish study conducted amongst children from Helsinki showed that the mean blood lead level amongst children attending a day care centre was 2.6  $\mu\text{g}/\text{dl}$  (Ponka 1998). Blood lead levels amongst children aged 4 to 6 years from an urban Delhi (India) slum was 7.8  $\mu\text{g}/\text{dl}$ , which was considerably lower than in the current Johannesburg sample (9.1  $\mu\text{g}/\text{dl}$ ). In the Delhi sample 18% of children had blood lead levels equalling or exceeding 10  $\mu\text{g}/\text{dl}$  (Kaira et al 2003), compared with 35% in the Johannesburg sample.

The mean blood lead levels among the children sampled in the current study are relatively closely aligned with that determined in certain developing country settings. For example, a study of urban Senegalese children showed a mean blood lead concentration of 10.6  $\mu\text{g}/\text{dl}$  (Diouf et al 2003), which is higher than the mean blood lead concentration determined for the Johannesburg sample. A study conducted in Jakarta, Indonesia (Albalak et al 2002) showed that the geometric mean blood lead level amongst children there was 8.6  $\mu\text{g}/\text{dl}$  and 35% of children had blood lead levels equal to or greater than 10  $\mu\text{g}/\text{dl}$ .

There was considerable variation in this study in the distribution of blood lead concentrations within urban areas. For example, significant area differences in

blood lead concentrations were observed within Cape Town, with children attending schools in the relatively heavily trafficked suburbs of Woodstock and Mitchell's Plain having significantly higher blood lead concentrations than their counterparts in Hout Bay, where traffic density levels are lower. The mean blood lead level in Hout Bay was 4.8 µg/dl, which was considerably lower than the mean levels in Woodstock or Mitchell's Plain, where mean blood lead levels was 6.9 µg/dl. The mean blood lead level at one Hout Bay school was 3.3 µg/dl, which is approaching the concentrations being measured amongst children in the Scandinavian countries and the United States of America (Thomas et al. 1999). In contrast, mean blood lead concentrations up to 8.1 µg/dl were measured in a Woodstock school, and 24% of Woodstock children had blood lead concentrations that were equal to or exceeded 10 µg/dl.

Similarly within the Johannesburg sample, the mean blood lead concentration amongst children from Alexandra was 10.4 µg/dl, while in Westbury/Riverlea the mean level was 9.6 µg/dl. In contrast the mean blood lead level in the inner city sample was 8.0 µg/dl.

#### **4.2 TEMPORAL CHANGES IN BLOOD LEAD DISTRIBUTIONS**

This study has shown that blood lead levels among South African children have declined substantially since the mid-1980s and early 1990s, when only leaded petrol was available in the country, and research showed that 90 to 100% of urban children had elevated blood lead concentrations (von Schirnding et al 1991a). Reductions in average blood lead concentrations have been observed in rural as

well as urban settings, in Black African and Coloured as well as White children, and in heavily trafficked suburbs as well as suburbs with relatively low traffic volumes.

It is highly probable that a substantial proportion of the observed reductions in South African children's blood lead concentrations is attributable to reductions in the maximum permissible concentration of lead in petrol between 1986 and 1989, and the introduction in 1996 of unleaded petrol in South Africa. Such a reduction in children's blood lead concentrations, following a partial or complete phase-out of the use of leaded petrol, mirrors what has been seen in countries throughout the world (Thomas et al 1999). For example, phasing out the use of leaded petrol in Shantou, China in 1998 was followed by a decline in the proportion of children with blood lead levels equal to or exceeding 10 µg/dl from 44% in 1999 to 23% by 2001. Mean blood lead concentrations were reduced from 10.4 to 7.9 µg/dl over the same period (Luo et al 2003). Similar reductions were observed in Shanghai, China between 1997 and 1999 (Yan et al 2002). Limitation of the use of lead in petrol was followed by reductions in blood lead concentrations from 12 µg/dl in 1990 to 6.3 µg/dl in 1995 in Spain's Tarragona Province (Schuhmacher et al 1996). Mean blood levels amongst Helsinki children declined from 4.6 µg/dl in 1983 to 3.0 µg/dl in 1988, and to 2.6 µg/dl in 1989 (Ponka 1998), in association with reductions in vehicular emissions of lead.

Following discontinuation of the use of leaded petrol in the United States of America, the mean blood lead concentrations of children aged 1 to 5 years declined from 13.7 to 3.2 µg/dl for non-Hispanic white children and from 20.2 to 5.6

$\mu\text{g}/\text{dl}$  for non-Hispanic black children. The prevalence of blood lead levels equalling  $10 \mu\text{g}/\text{dl}$  or greater for children aged 1 to 5 years declined from 85% to 6% for non-Hispanic white children and from 98% to 21% for non-Hispanic black children. The authors concluded that the major cause of the observed decline in blood lead levels was most likely the removal of 99.8% of lead from petrol and the removal of lead from soldered cans. The authors also declared that further efforts were needed to remove other sources of lead, for example lead in paint, dust and soil, but cautioned that these would be more difficult than removing lead from gasoline and soldered cans had been (Pirkle et al 1994).

While it is expected that further reductions in the blood lead distribution amongst South African children will be seen towards and beyond 2006 (which is the targeted deadline for phasing out the use of leaded petrol in the country), additional effort will also be needed to address the contribution of non-petrol sources of lead to ongoing lead exposure and poisoning in South African children.

Despite the overall, considerable improvement in childhood blood lead distributions observed, the findings of this study indicate that an intolerably high proportion of young South African children continue to have elevated blood lead levels, especially those who live in conditions of poverty. Overall 17% of the children studied had blood lead levels equaling or exceeding the international action level of  $10 \mu\text{g}/\text{dl}$ . Taking account of a large body of evidence associating health effects such as mild intellectual impairment and hearing loss with blood lead concentrations around  $3 \mu\text{g}/\text{dl}$ , it is of concern that 78% of subjects in this study had blood lead levels of  $5 \mu\text{g}/\text{dl}$  or higher, possibly marking lead exposure and

poisoning as the number one environmental health problem in South Africa, at least in urban areas. These findings hold major implications for South Africa as a country striving to overcome the educational, development, economic and political consequences of *apartheid*, especially in relation to education, social development, health, the reduction of crime and poverty elimination. In light of increasing concerns around the absence of a threshold of safety for lead in blood and health effects such as reductions in IQ, it is appropriate that every effort be made in South Africa to bring children's blood lead levels down to the lowest possible level, preferably < 5 µg/dl.

#### **4.3 RISK FACTORS FOR ELEVATED BLOOD LEAD CONCENTRATIONS**

As expected, the study pointed to multiple and wide-ranging risk factors for elevated blood lead concentrations in the children studied. Risk factors varied across the various study sites. In respect of the total sample, age, population group, the age of the house in which the child lived, the number of people living in the house, the type of plumbing in the home, the proximity of the house to a busy road, the presence of peeling interior paint, the number of vehicles owned by household members, parental education, paternal occupation, whether or not the child attended a crèche or pre-school institution, the mode of transport used to travel to school, a pica tendency for paint or sand/soil and the presence of a household member who worked from home were significantly associated with elevated blood lead levels.

### **4.3.1 Age**

Blood lead levels are well known to vary with age (Haley & Talbot 2004, Tong et al 1998). Lead concentrations are usually highest among younger children, especially those aged two to four years, whose exploratory and mouthing behaviour is at a peak. The surveys reported here were conducted amongst first grade school children with an average age of 7 years, and therefore represents a considerable under-estimate of the likely blood lead distributions in the highest risk age group.

### **4.3.2 Socio-economic Status/Population Group**

In South Africa population group continues to be an accepted proxy for socio-economic status, with Black African and Coloured children in general characterized by low levels of socio-economic status relative to White children. In this study the mean blood lead concentrations in Black African (7.5 µg/dl) and Coloured (7.4 µg/dl) children were significantly higher than that in White children (4.2 µg/dl). Within Aggeneys, for example blood lead levels in Coloured children (8.4 µg/dl, n = 16) were twice as high as in White children (4.2 µg/dl, n = 3). Similarly, in Cape Town, the mean blood lead concentration in Black African children was 6.0 µg/dl and in Coloured children 7.0 µg/dl, while in White children the mean blood lead concentration was 3.7 µg/dl.

Variations by socio-economic status were also evident in relation to geographic location. For example, the highest blood lead levels were measured in the highly impoverished Johannesburg township of Alexandra, where more than half of the children studied had high blood lead levels. At one Alexandra school, more than three-quarters of the children studied had blood lead concentrations equaling or

exceeding the current action level of 10 µg/dl, and the mean blood lead concentration was 11.1 µg/dl. In contrast, at a relatively wealthy school in Hout Bay (Cape Town), attended for the most part by White children, the mean blood lead concentration was 2.7 µg/dl and none of the children had blood lead concentrations of 10 µg/dl or higher.

With 57 to 75% of South African children (mainly Black African and Coloured children) considered to be living in poverty (UCT 2003), lead poisoning may be considered a major public health concern relevant to the majority of South African urban children, as well as those living in selected rural settings, for example in lead mining towns or areas of natural lead mineralization.

#### **4.3.3 Housing Conditions**

In this study blood lead concentrations tended to increase with the age of the house in which the child lived. For example, the mean blood lead concentration of children whose dwellings were less than 25 years old was 7.2 µg/dl, while the mean in children whose houses were between 25 & 50 and > 50 years old respectively was 7.5 µg/dl and 7.7 µg/dl ( $p = 0.041$ ).

In Kimberley and Cape Town, though not in the remaining study sites or in respect of the total sample, blood lead levels were lower amongst children from informal settlements. This may have been due to the location of informal settlements away from busy roads and/or a lower proportion of painted dwellings in informal settlements.

Crowded housing is often associated with low socio-economic status and elevated blood lead concentrations. In this study increasing household size was associated with increasing blood lead concentrations, with each additional person associated with an increase in blood lead concentration of 0.11 µg/dl ( $p = 0.002$ ). An association between blood lead concentrations in children and levels of crowding was also observed in a lead exposure study undertaken in Durban, South Africa (Nriagu et al 1997).

#### **4.3.3 Lead in Petrol**

The use of high concentrations of lead in petrol in South Africa has undoubtedly resulted in widespread lead exposure over the past seven to eight decades amongst children from all walks of life. In terms of primary prevention measures, the 2002 parliamentary decision to phase out the use of lead in petrol in South Africa has been critical. This study has demonstrated that lowering of the maximum permissible petrol lead concentration, and the introduction of unleaded petrol, has been followed by significant reductions in children's blood lead concentrations in rural and urban areas. Nevertheless, with around 70% of petrol purchased in South Africa still being leaded, lead from petrol remains a significant source of lead exposure amongst children in the country, especially amongst those who live in homes or attend schools located in close proximity to heavily trafficked roads.

#### **4.3.4 Lead in Paint**

Of particular concern is the evidence from this study of the contribution of past and ongoing use of lead based paint to elevated blood lead levels amongst children in the country. In Kimberley and Johannesburg, as well as in respect of the total study

sample, pica for paint was demonstrated to be a significant risk factor for elevated blood lead concentrations. The presence of peeling paint on the interior and exterior walls of dwellings was, similarly, significantly associated with elevated blood lead concentrations, as was the age of the dwelling, which could be associated with the potential for peeling paint.

The analysis of the lead content in paint samples collected from homes in randomly selected Johannesburg suburbs (stratified by age of proclamation) showed that in 60% of the suburbs studied, at least one home had been coated with lead-based paint. In total, the lead content in 17% of the paint samples collected was elevated, and lead based paint had been used in one-fifth of the homes investigated. Since this preliminary scanning exercise did not involve comprehensive testing of all rooms in the selected dwellings, it is likely that these figures considerably under-estimate the true proportion of dwellings with lead based paint in the City of Johannesburg.

Measurement of the lead content of paint samples collected from the school buildings studied showed that between 20% and 36% of schools were painted with lead-based paint. In the Alexandra school where the highest blood lead concentrations were measured (the mean blood lead concentration was 11.3 µg/dl and 76% of children attending the school had high blood lead concentrations), the lead content of the school paint sample was 8 600 µg/g. In Cape Town the lead concentration in school paint samples ranged up to 34 434 µg/g.

The young girl with the highest blood lead concentration in this study (between 44 and 52 µg/dl) had been ingesting paint chips with lead concentrations ranging up to 46 000 µg/g, which considerably exceeds the reference level for lead based paint in the United States of America - 5 000 µg/g. It was suspected that she was also ingesting paint chips from the walls of her school building, where sampling showed that the paint lead concentration also exceeded the USA standard. Analysis of paint samples collected during home visits to the twenty children with the highest blood lead concentrations in the Johannesburg phase of the study showed that one third had concentrations of lead that exceeded 5 000 µg/g. In this study it was reported that 4% of children had been observed to ingest paint.

Of particular concern in terms of primary prevention of lead poisoning is the finding from the preliminary screening study of the lead content of paint samples purchased from the shelves of paint stores in Cape Town and Johannesburg, that lead currently continues to be added to paint in South Africa intended for residential purposes. While elevated lead concentrations were not determined in PVA (“water-based”) paints and in white shades of enamel paints, in red, yellow and orange colours of enamel paint, 83% of samples were determined to be lead based. The lead concentrations of lead based paint samples ranged up to 189 000 µg/g, which is 38 times the USA definition of lead based paint. High lead concentrations were found in lesser known as well as popular brands of paint. It was also observed that only one of the eleven brands of lead based paint tested made use of warning stickers to alert prospective purchasers to the hazardous contents.

Particularly alarming has been the evidence from this study that highly elevated lead concentrations are to be found in the paint coatings on widely available children's toys and coloured pencils in South Africa. Young children especially may be at risk of ingestion of lead based paint during periods when mouthing behaviour is pronounced. Chewing of pencils is also a common habit amongst older children.

Given the ongoing challenge of preventing lead poisoning from applied paint in even well resourced countries such as the USA, it is unacceptable that paint manufacturers in South Africa continue to add lead to paint at this time. It is evident that the voluntary agreement reached among members of the South African Paint Manufacturers Association (SAPMA) in the 1970s has been woefully inadequate to protect the country's children against preventable lead poisoning from paint used within and around their homes and schools. Over and above the evidence that SAPMA members are currently and unethically flouting their own agreement, not all paint manufacturers belong to the SAPMA, strengthening the need for regulatory control of the actions of paint manufacturers in South Africa in regard to the use of lead.

In the past, the role of lead-based paint in elevated childhood blood lead levels may have been masked by the vast contribution from lead particles associated with motor vehicle exhaust emissions. With the planned phase-out of the use of leaded petrol in the country by 2006, lead-based paint is likely to gain increasing prominence as a key contributor to the public health problem of lead exposure in South Africa. As shown by the experience in the United States of America (Needleman 1998), addressing the problem of lead-based paint, will prove to be

particularly challenging and costly, especially in the context of a country such as South Africa (as well as African countries in general), where levels of poverty and inequity are high, where a process of rapid urbanization continues, where resources are constrained and where housing backlogs are significant.

#### **4.3.5 Plumbing**

Although there is little evidence from the school-based water sampling component of this study that lead in water is a major concern, analysis of the questionnaire data showed that blood lead levels were higher among children who lived in homes with plumbing made from a metallic substance. It may be useful to undertake a detailed study of the lead concentrations in end-point use water samples in various locations across the study, using established sampling and analytical methodologies.

#### **4.3.6 Informal Sector Exposure to Lead**

The informal sector plays a key role and represents around 10% of the retail economy in South Africa (Ligthelm 2004). Home-based informal sector or “cottage industry” activities such as repairing of motor vehicles, dismantling of lead batteries, repairs to electrical appliances using lead solder and paint work, may put household members, especially young children, at high risk of environmental lead exposure. Also important is the transfer of lead particles from work into home environments on the hair, clothing, shoes and skin of adults working in lead-related occupations such as painting.

In this study blood lead concentrations were higher among children who lived where someone worked from home. Also, in the homes of three of the twenty children in Johannesburg with the highest blood lead concentrations, lead-related activities were being undertaken within the child's home environment. In one case lead solder was regularly used (to repair electrical appliances as a means to household income) in the single-roomed informal backyard dwelling which housed the family of five, and where all household activities, including dining, were undertaken. The child's parents were unaware of the health risks associated with lead exposure. In a second case motor vehicle repair work was being undertaken at home, while in the third case a painting business was being run from the child's house.

After taking account of potential confounding factors such as socio-economic status, blood lead levels in the total sample remained significantly associated with gender, the mode of transport used to travel to school, attending a pre-school institution, pica for paint and living in a dwelling with interior peeling paint.

#### **4.4 STRENGTHS AND WEAKNESSES OF THE THESIS**

##### **4.4.1 Weaknesses**

This study was conducted to assess the risk of lead exposure amongst high risk children living in conditions of relative poverty. The sampling process was not designed to be nationally representative, and the results consequently cannot be extrapolated to all South African children. Middle class and wealthy children of the same age group are likely to have considerably lower blood lead concentrations

relative to the findings of this study. Nevertheless, since more than 50% of South African children are considered to be living in poverty, the findings are likely to be relevant to a large proportion of the country's children.

For reasons of convenience, grade one school children, whose mean age was seven years, were selected for this study. At this age, blood lead concentrations are estimated to be around 40% lower than at the age of highest risk which is two to four years of age. It is therefore of concern that the true prevalence of elevated blood lead concentration in children at the highest risk age group would be considerably higher than observed in this study.

For the purposes of the temporal analyses, conducted to compare blood lead levels before and after the introduction of unleaded petrol, the findings of the current study (2002/3) were compared with those from studies undertaken in 1991 (Cape Town, Aggeneys and Pella) and 1995 (Johannesburg). Between 1991 and 2002, improvements in laboratory methods and techniques, such as improved mechanisms for cleaning laboratory glassware, may have resulted in lower levels of contamination associated with the latter studies. Similarly, improved analytical techniques over this period may have lowered the levels of detection for lead in blood. These improvements are however, unlikely to have affected in a significant manner, the overall outcome of the study. The findings of this study mirror what has been seen around the world following the introduction of unleaded petrol.

The blood lead samples collected during the studies in Cape Town, Pella and Aggeneys of 1991 were analyzed in the analytical laboratories of the Red Cross

War Memorial Children's Hospital, while the lead content of the blood samples collected during the surveys of 1995 and 2002/3 were analyzed at the National Institute for Occupational Health. While both of these laboratories participate in national and international quality control programmes, the possibility nevertheless exists that differences in approaches and systems of the two laboratories may have had an effect on the blood lead levels determined.

For the purposes of the temporal analyses, conducted to compare blood lead levels before and after the introduction of unleaded petrol, the findings of the current study (2002/3) were compared with those from studies undertaken in 1991 (Cape Town, Aggeneys and Pella) and 1995 (Johannesburg). Between 1991 and 2002, improvements in laboratory methods and techniques, such as improved mechanisms for cleaning laboratory glassware, may have resulted in lower levels of contamination associated with the latter studies. Similarly, improved analytical techniques over this period may have lowered the levels of detection for lead in blood. These improvements are however, unlikely to have affected in a significant manner, the overall outcome of the study. The findings of this study mirror what has been seen around the world following the introduction of unleaded petrol.

The blood lead samples collected during the studies in Cape Town, Pella and Aggeneys of 1991 were analyzed in the analytical laboratories of the Red Cross War Memorial Children's Hospital, while the lead content of the blood samples collected during the surveys of 1995 and 2002/3 were analyzed at the National Institute for Occupational Health. While both of these laboratories participate in national and international quality control programmes, the possibility nevertheless

exists that differences in approaches and systems of the two laboratories may have had an effect on the blood lead levels determined. affected the results It is have

***Time Lag between Main and Comparison Studies (prior to the introduction of unleaded petrol)***

The blood lead analyses for this study was conducted by the National Institute for Occupational Health in Johannesburg. However, for the purpose of temporal comparisons, baseline blood lead distributions were provided by studies conducted in 1991 (Cape Town, Pella and Aggeneys). The lead content of these blood samples (collected in 1991) were determined by the laboratories of the Red Cross Children's Hospital in Cape Town. A degree of error may have resulted from improvements in laboratory and analytical methods that may have occurred over the 11-year period between the two studies. In addition, there may have been differences in equipment, techniques and practices between the two laboratories that could have affected the blood lead determinations in 2002/3 relative to 1991.

***Questionnaire Administration***

The mode of questionnaire administration, for example self-administered versus administration by an interviewer, has been shown to contribute to bias, which may have important implications for the validity of the results of research (Bowling 2005). In this study, the self-administration mode may have contributed to greater honesty in responses, but may also have been associated with, for example, increased levels of missing information and bias, where respondents did not understand or misinterpreted certain questions. This study may have benefited

from the fact that a single mode of questionnaire administration (self-administration) was adopted throughout the survey. Bias has been shown to be greater between, rather than within, different modes of questionnaire administration (Bowling 2005).

#### **4.4.2 Strengths**

This study has made a major contribution to understanding the extent of children's exposure to lead in key South African settings (urban, rural, lead mining town). The study findings have contributed significantly to the development of policies and programmes in South African aimed at protecting children against environmental lead hazards.

### **4.5 OBSTACLES TO THE PREVENTION OF LEAD EXPOSURE & POISONING**

#### **4.5.1 Public Awareness of Lead Hazards**

A particular concern in efforts to protect South African children from the preventable hazard of environmental lead exposure is the low levels of awareness in the general public of the sources, mechanisms of exposure, ill health and social outcomes and prevention measures in relation to lead exposure. This study has shown that despite an extensive and growing global body of knowledge of the health and social hazards of lead, there is limited awareness in this regard in the general public in South Africa. For example, an assessment of awareness of the health risks of lead among respondents to the questionnaire administered in the Northern Cape province – Kimberley, Pella/Onseepkans and Aggeneys showed

that in Kimberley only 44% of people thought that lead could be harmful to health, while 23% thought lead was not harmful to health and 33% did not know. In the lead mining town of Aggeneys, 67% of respondents thought that lead could be harmful to health, while 19% thought there was no health risk associated with lead and 14% did not know. In the rural towns of Pella and Onseepkans 25% of respondents thought lead was harmful to health, 23% thought lead was not harmful and 52% did not know whether or not lead could harm health.

There are also concerns regarding awareness of the sources, and mechanisms of exposure to lead among children, in the health sector, as well as other sectors of relevance such as social development and education. The case study of the girl with a severe pica habit and a blood lead level of 52 µg/dl in respect of whom the possibility of lead poisoning was not considered by doctors and nurses at the paediatric department of the hospital to which she was taken by her parents, also illustrates the low levels of awareness within the health sector of this widely prevalent environmental health risk to children. In 1999, 2000, 2001 and 2002 only 4, 0, 10 and 13 cases of lead poisoning were reported to the Department of Health nationally. These figures were not age-specific.

The low levels of public and sectoral awareness of lead hazards is a major obstacle to efforts to protect South African children from the preventable hazard of environmental lead exposure. A nation-wide campaign to improve public awareness of the hazards of lead exposure is now long overdue, and needs to be implemented as a matter of urgency, with parents, pregnant women and children themselves being key target groups.

#### **4.5.2 Need for Further Research, Screening & Surveillance**

This study counts among a relatively small number of research projects undertaken over the past twenty years to describe the problem of lead exposure in South African children. It is important that additional research be conducted to further characterize the extent of lead exposure, and identify the highest risk settings and groups, as well as risk factors. The existing evidence of widespread childhood lead exposure in South Africa also points to the need for a nation-wide surveillance programme and screening in high risk areas.

#### **4.5.3 Policy & Regulation**

The regulatory framework in South Africa for the protection of children against environmental lead is weak and lacking in integration. The most important piece of legislation in this regard relates to the decision to phase out of the use of leaded petrol in South Africa by January 2006 (Government Gazette, General Notice 3324 of 2003, Joint Implementation Strategy for the Control of Exhaust Emissions from Road-Going Vehicles in the Republic of South Africa). In terms of the Occupational Health and Safety Act (Act No. 85 of 1993 – Section 43), Lead Regulations were drafted to protect workers, and by extension, their families against lead exposure emanating from occupation use. However, as is shown in the phase of this study undertaken in the lead mining town of Aggeneys, it is likely that inadequate enforcement of the prescribed occupational hygiene measures is resulting in a sub-optimal level of protection, and the consequent transfer of lead particles from the work setting into the home environment on the skin and clothing of workers. There is also no legislation requiring that prospective purchasers of new homes be

alerted to the presence of lead in paint. While a voluntary agreement exists amongst members of the South African Paint Manufacturers' Association to limit the addition of lead to paint, the inadequacy of this measure has been demonstrated in this study. Not only are SAPMA members flouting their own agreement, it is estimated that only around 65% of paint manufacturers in the country are SAPMA members.

The absence of blood lead standards for children, protocols for medical response to cases of elevated blood lead and regulation of the lead content of paint are additional examples of critical gaps in the policy and regulatory framework to address lead exposure in South African children. There are also concerns around the availability of resources and skills to adequately monitor environmental lead exposure and enforce standards and regulations should these be promulgated. It is likely that any measures taken to prevent childhood lead exposure and poisoning in South Africa will rely heavily on the actions of parents and consumers.

#### **4.6 ETHICAL OBLIGATIONS**

As part of the ethical obligations of the researchers, a suitably qualified company was appointed to remove all lead based paint from the apartment in which the subject with a blood lead level of 44.4 µg/dl lived, and dispose of it in a hazardous waste dump site. The apartment walls were re-plastered and decorated with paint pre-tested to ensure that the lead concentration was within acceptable limits.

#### **4.7 IMPLEMENTATION OF POLICIES AND PROGRAMMES**

The results of this epidemiological study, together with the supplementary investigations, were conveyed to the Minister of Health and presented to the Executive Management Team of the National Department of Health, the National Department of Minerals and Energy as well as to the Gauteng Provincial Education Department. The information presented catalyzed, or contributed to, important policy and programmatic decisions, including:

- Confirmation of a parliamentary decision to phase out the use of leaded petrol in South Africa by 2006;
- A decision to regulate the use of lead in paint in South Africa. This decision was spurred, in particular, by the finding from this study, that painted, wooden children's toys in South Africa may contain alarmingly high concentrations of lead;
- The implementation of a nation-wide awareness campaign to alert parents, pregnant women, educators, health workers and children of the sources, mechanisms of exposure, health and social risks and means of protections against environmental lead.
- Discussions within the Department of Health on further measures to protect children against exposure to environmental lead.

## **CHAPTER 5 - CONCLUSIONS**

### **5.1 SUMMARY OF THE OBJECTIVES AND FINDINGS**

The results of this study indicate that there has been a significant reduction in children's blood lead concentrations in the cities of Cape Town and Johannesburg. This reduction is most likely a cumulative impact of steps taken to reduce the lead content of petrol, and to introduce unleaded petrol in the country in 1996. It is likely that in the run-up to and beyond 2006, the target date for the cessation of the use of leaded petrol in South Africa, children's blood lead levels will decline further.

Despite the significant reductions observed, lead exposure and poisoning constitutes a widespread and serious environmental and public health problem in South Africa. Large numbers of South African children continue to have elevated blood lead levels, in a range associated with effects such as reductions in IQ scores, hyperactivity, shortened concentrations spans, hearing loss and poor performance at school. Children living, or attending schools, in impoverished areas have the highest blood lead concentrations. For example in Alexandra schools, up to 76% of first grade children had blood lead concentrations equaling or exceeding the international action level of 10 µg/dl.

Lead in petrol has in the past been the overwhelming source of exposure to environmental lead in children. Petrol lead continues to be a significant contributor to elevated blood lead concentrations. For example, children attending schools

close to heavily trafficked roads, and those residing on busy roads, have higher blood lead concentrations than those who do not. In this study however, the increasingly important role of lead-based paint, possibly masked in the past by the overwhelming contribution from petrol lead, has emerged to be an important risk factor for high blood lead levels, particularly among children with the highest blood lead concentrations. The policies and interventions required to address lead-based house and school paint as a source of exposure in children, are likely to be considerably more challenging, costly and protracted than for lead in petrol.

Control, reduction, prevention and intervention strategies to address environmental lead exposure sources are more complex than those for confined occupational sources, because the former is varied and heterogeneous in nature. Effective interventions will similarly need to be trans-sectoral in nature.

## **5.2 ORIGINALITY/NEW INSIGHTS**

This study has highlighted the ongoing and challenging role of lead-based paint in elevated blood lead concentrations amongst South African children. In particular, this study has highlighted how South African children have, for around three decades, been unnecessarily put at risk of lead poisoning from paint as a consequence of members of the South African Paint Manufacturers Association breaching their own voluntary agreement, and the lack of regulation of the paint industry in South Africa.

### **5.3 POLICY IMPLICATIONS**

There is an urgent need for the development of a holistic & centralized approach to the prevention of lead exposure and poisoning among South African children. Important within a holistic programme of action to reduce childhood lead exposure in South Africa and other African countries, are increased public, health and education sector awareness of the sources and mechanisms of exposure to lead, the implementation of research and screening programmes to identify high risk areas and groups, the development of blood lead standards for children, the provision of secondary prevention measures (both medical and environmental) and strengthening the role of civil action to prevent childhood lead poisoning. In terms of lead-based paint in particular, banning or the development of standards for the use of lead in paint is critical. Testing needs to be undertaken to identify high risk dwellings and school buildings, the availability of rapid test kits for lead-based paint needs to be increased, provisions should be made for the safe removal of lead-based paint from the worst affected dwellings and school buildings and prospective home owners need to be alerted of the presence of lead-based paint in properties of interest and the implications for household health.

### **5.4 FUTURE RESEARCH NEEDS**

This study can only be regarded as a preliminary investigation into the problem of lead exposure amongst South African children. The investigations reported here need to be expanded to address a number of information gaps, including:

- Characterization of the distribution of blood lead levels in the highest risk age groups;
- The risk of lead exposure amongst children living in settings with naturally high lead levels;
- Further examination of the risk of lead exposure from paint
- Investigation of risk of para-occupational exposure to lead in children;
- Investigation of the risk of lead exposure in children who live in homes where lead-related cottage industries are being conducted.
- The impact on children's blood lead concentrations of extended use of unleaded petrol.

## **5.5 THE WAY FORWARD**

Given the range of sources of lead exposure in South African children, the establishment of a trans-sectoral commission (including representation from the Departments of Health, Education, Environment and Tourism, Labour, Provincial and Local Government, Trade and Industry and Housing, as well as the academic, business and non-governmental sectors) to reduce the risk of lead exposure and poisoning is essential.

Primary prevention, through elimination of the sources of lead should be an overarching goal of the proposed trans-sectoral lead exposure reduction commission. Key actions should include prohibiting the use of lead in petrol, paint, water pipes and industrial emissions, removing or containing lead based paint in homes,

schools and other public institutions, as well as children's toys and furniture and preventing exposure from the use of lead in industry and the informal sector.

In the context of South Africa, where the lead exposure situation has not yet been extensively characterized, epidemiological investigations, surveillance programmes, screening programmes and source identification, the enforcement of regulations and a long-term government commitment to eliminating lead as a threat to the next generation of children is critical. The implementation of high level awareness campaigns in the general public, as well as within the health, education, social development and other sectors, to improve knowledge of the hazards, sources and pathways of exposure to lead, is crucial.

Steps that need to be considered as part of a lead poisoning prevention strategy for South Africa include:

- The implementation of a national public education campaign to increase awareness of the sources, pathways of exposure, dangers, signs and symptoms and protective measures in respect of exposure to lead amongst children. The target groups of such lead awareness campaigns should include pregnant women, parents, children, health workers, educators and social development officials;
- The setting of blood lead standards for South African children;
- The development of response protocols for children with elevated blood lead levels, including aspects related to follow-up blood lead testing, home investigations, chelation therapy and primary prevention measures such as decontamination of homes and schools;

- Institutional arrangements for blood lead testing at health facilities;
- The implementation of universal blood lead screening in high risk areas and groups;
- Research and surveillance to identify high risk groups and settings;
- Centralized analysis and interpretation of the results of blood lead testing;
- Banning of the addition of lead to paint for residential and school use and application on children's toys, furniture, play ground equipment and other items.
- Nutritional supplementation, especially of iron, zinc and calcium, in high risk groups and settings.
- Safe Housing/School Maintenance & Decontamination Programmes;
- The institution/encouragement of environmental hygiene practices that will facilitate the removal of lead from the school environment, and associated training programmes for relevant staff.

## LIST OF REFERENCES

Ahmed M, Ahmad P, Kutbi II (1989). Lead pollution in urban and rural Saudi Arabian children. *Bull Environ Contam Toxicol* **43(5)**: 660-6.

Aguilar-Garduno C, Lacasana M, Tellez-Rojo MM, Aguilar-Madrid G, Sanin-Aguirre LH, Romieu I, Hernandez-Avila M (2003). Indirect lead exposure among children of radiator repair workers. *American Journal of Industrial Medicine* **43(6)**: 662-7.

Albalak R, Noonan G, Buchanan S, Flanders WD, Gotway-Crawford C, Kim D, Jones RL, Sulaiman R, Blumenthal W, Tan R, Curtis G, McGeehin MA (2003). Blood lead levels and risk factors for lead poisoning among children in Jakarta. *Science of the Total Environment* **301(1-3)**: 75-85.

Baily P, Norval E, Killroe-Smith TA, Skikne MI, Röllin H (1979). The Application of Metal-Coated Graphite Tubes to the Determination of Trace Metals in Biological Materials. *Microchemical Journal* **24**: 107-116.

Begerow J, Freier I, Turfeld M, Kramer U, Dunemann L (1994). Internal lead and cadmium exposure in 6-year-old children from western and eastern Germany. *International Archives of Occupational and Environmental Health* **66(4)**: 243-8.

Bellinger D, 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)**: 1037-43.

Bjerre B, Berglund M, Harsbo K, Hellman B (1993). Blood lead concentrations of Swedish preschool children in a community with high lead levels from mine waste in soil and dust. *Scandinavian Journal of Work, Environment and Health* **19(3)**: 154-61.

Bowling A (2005). Mode of questionnaire administration can have serious effects on data quality. *J Public Health (Oxf)* **27(3)**: 281-91.

Budd P, Montgomery J, Evans J, Trickett M (2004). Human lead exposure in England from approximately 5500 BP to the 16th century AD. *Science of the Total Environment* **318(1-3)**:45-58.

Canfield RL, Henderson MA, Cory-Slechta DA, Cox C, Jusko T, Lanphear B (2003). Intellectual impairment in children with blood lead concentrations below 10 µg/dl. *The New England Journal of Medicine* **348(16)**: 1517–524.

Cao X, Ma LQ, Chen M, Hardison DW Jr, Harris WG (2003). Weathering of lead bullets and their environmental effects at outdoor shooting ranges. *J Environ Qual* **32(2)**: 526-34.

Centers for Disease Control (2001). Fatal pediatric lead poisoning – New Hampshire, *Journal of the American Medical Association* **286(1)**: 38-39.

Centers for Disease Control (1991). Prevention of childhood lead poisoning. *Curr Opin Pediatr* **12**: 428-437.

Chetty N, Jinabhai CC, Green-Thompson RW (1993). Lead levels in maternal and umbilical cord blood at King Edward Hospital, Durban. *South African Medical Journal* **83(3)**: 227.

Chiaradia M, Chenhall BE, Depers AM, Gulson BL, Jones BG (1997). Identification of historical lead sources in roof dusts and recent lake sediments from an industrialized area: indications from lead isotopes. *Science of the Total Environment* **205(2-3)**: 107-28

Chiaradia M, Gulson BL, MacDonald K (1997). Contamination of houses by workers occupationally exposed in a lead-zinc-copper mine and impact on blood lead concentrations in the families. *Occupational and Environmental Medicine* **54(2)**:117-24.

Chisolm JJ Jr & Harrison HE (1957). The treatment of acute lead encephalopathy in children. *Pediatrics* **19(1)**:2-20.

Chisolm JJ Jr & Harrison HE (1956). The exposure of children to lead. *Pediatrics* **18(6)**: 943-58.

Chisolm JJ Jr, Thomas DJ, Hamill TG (1985). Erythrocyte porphobilinogen synthase activity as an indicator of lead exposure in children. *Clin Chem* **31**: 601-5.

Counter SA, Buchanan LH, Ortega F, Amarasiriwardena C, Hu H (2000). Environmental lead contamination and pediatric lead intoxication in an Andean Ecuadorian village. *International Journal of Occupational and Environmental Health* **6(3)**: 169-76.

de la Burde B and Choate ML (1975). Early asymptomatic lead exposure and development at school age. *J Pediatr* **87(4)**: 638-42.

de la Burde B and Choate MS Jr (1972). Does asymptomatic lead exposure in children have latent sequelae? *J Pediatr* **81(6)**: 1088-91.

Department of Health (1979), Lead Poisoning in Children. Department of Health, Pretoria, South Africa.

Department of Housing and Urban Development (1995). Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing. Washington D.C., United States of America, June 1995.

Deveaux P, Kibel MA, Dempster WS, Pocock F, Formenti K (1986). Blood Lead Levels in Pre-school Children in Cape Town. *South African Medical Journal* **69**: 421-424.

Dillman RO, Crumb CK, Lidsky MJ (1979). Lead poisoning from a gunshot wound. Report of a case and review of the literature. *American Journal of Medicine* **66(3)**: 509-14.

Diouf A, Garcon G, Thiaw C, Diop Y, Fall M, Ndiaye B, Siby T, Hannotiaux MH, Zerimech F, Ba D, Haguenoer JM, Shirali P (2003). Environmental lead exposure and its relationship to traffic density among Senegalese children: a pilot study. *Hum Exp Toxicol* **22(10)**: 559-64.

Dwivedi SK and Dey S (2002). Medicinal herbs: a potential source of toxic metal exposure for man and animals in India. *Archives of Environmental Health* **57(3)**: 229-31.

Dzioubinski O & Chipman R (1999). Trends in Consumption and Production: Selected Minerals. Discussion paper of the United Nations Department of Economic and Social Affairs (ST/ESA/1999/DP.5).

EPA (United States Environmental Protection Agency). 40 CFR Part 745. Lead; Identification of Dangerous Levels of Lead; Final Rule. Washington, DC. 2001. Available: <http://www.epa.gov/lead/leadhaz.htm> [20 January 2003].

Ernhart CB, Landa B, Schell NB (1981). Subclinical levels of lead and developmental deficit--a multivariate follow-up reassessment. *Pediatrics* **67(6)**: 911-9.

Gibson L (1904). A plea for painted railings and painted walls of rooms as the source of lead poisoning amongst Queensland children. *Australasian Medical Gazette* **23**: 149-153.

Gomaa A, Hu H, Bellinger D, Schwartz J, Tsaih SW, Gonzalez-Cossio T, Schnaas L, Peterson K, Aro A, Hernandez-Avila M (2002). Maternal bone lead as an independent risk factor for fetal neurotoxicity: a prospective study. *Pediatrics* **110**: 110-8.

Goyer RA (1995). Nutrition and metal toxicity. *American Journal of Clinical Nutrition* **61(3 Suppl)**: 646S-650S.

Greensher J, Mofenson HC, Balakrishnan C, Aleem (1974). Lead poisoning from ingestion of lead shot. *Pediatrics* **54(5)**: 641-3.

Grobler SR, Theunissen FS, Maresky LS (1996). Evidence of undue lead exposure in Cape Town before the advent of leaded petrol. *South African Medical Journal* **86(2)**: 169–171.

Gulson BL, Mizon K, Law AJ, Korsch MJ, Davis JJ (1994). Sources and pathways of lead in humans from the Broken Hill mining community - an alternative use of exploration methods. *Econ. Geol* **89**: 889-908.

Haley VB, Talbot TO (2004). Seasonality and trend in blood lead levels of New York State children. *BMC Pediatrics* **4(1)**: 8.

Harris I (1976). Lead encephalopathy. *South African Medical Journal* **50(35)**: 1371–3.

Hashim JH, Hashim Z, Omar A, Shamsudin SB (2000). Blood lead levels of urban and rural Malaysian primary school children. *Asia Pac J Public Health*, **12(2)**: 65–70.

Hernberg S and Nikkanen J (1970). Enzyme inhibition by lead under normal urban conditions. *Lancet* **1(7637)**: 63–4.

Howell EM and Russette L (2004). An innovative blood lead screening program for Indian children. *Public Health Rep* **119(2)**: 141–3.

International Labour Office (1921). Convention 13 concerning the use of white lead in painting. ILO publications. (No.13).

Jacobs DE, Mielke H, Pavur N (2003). The high cost of improper removal of lead-based paint from housing: a case report. *Environmental Health Perspectives* **111(2)**: 185–6.

Joseph K and Verwey W (2001). An overview of lead recycling in South Africa. Paper presented at an UNCTAD workshop. Building National Capacity in Rapidly

Industrializing Countries on Sustainable Management of Recoverable Material/Resources. Bangkok, 20-22 September 2001.

Kalra V, Chitralkha KT, Dua T, Pandey RM, Gupta Y (2003). Blood lead levels and risk factors for lead toxicity in children from schools and an urban slum in Delhi. *J Trop Pediatr* **49(2)**: 121-3.

Kotok D, Kotok R, Heriot JT (1977). Cognitive evaluation of children with elevated blood lead levels. *Am J Dis Child* **131(7)**: 791-3.

Landrigan PJ (2002). The worldwide problem of lead in petrol. *Bulletin of the World Health Organization* **80(10)**: 768.

Landrigan PJ, Tamblyn PB, Nelson M, Kerndt P, Kronoveter KJ, Zack MM (1980). Lead exposure in stained glass workers. *American Journal of Industrial Medicine* **1(2)**: 177-80.

Lanphear BP and Roghmann KJ (1997). Pathways of lead exposure in urban children. *Environmental Research* **74(1)**: 67-73.

Ligthelm A (2004). Size, structure and profile of the informal retail sector in South Africa. A report by the South African Bureau of Market Research.

Luo W, Zhang Y, Li H (2003). Children's blood lead levels after the phasing out of leaded gasoline in Shantou, China. *Archives of Environmental Health* **58(3)**: 184-7.

Mathee A, Röllin HB, Ditlopo NN, Theodorou P (2003). Childhood lead exposure in South Africa. *South African Medical Journal* **93(5)**: 313.

Mathee A, von Schirnding YER, Levin J, Ismail A, Huntley R, Cantrell A (2002). A survey of blood lead levels amongst young Johannesburg school children. *Environmental Research* **90(3)**: 181-4.

Mathee A, von Schirnding YER, Ismail A, Huntley R (1996). Surveys of blood lead burdens among school children and newborns in Greater Johannesburg. *Urbanization and Health Newsletter* **29**: 43-49.

Mathee A (1995). Cord blood lead levels and associated risk factors in Greater Johannesburg, South Africa. MSc Thesis, London School of Hygiene and Tropical Medicine.

McCabe EB (1979). Age and sensitivity to lead toxicity: a review. *Environmental Health Perspectives* **29**: 29-33.

McQuirter JL, Rothenberg SJ, Dinkins GA, Kondrashov V, Manalo M, Todd AC (2004). Change in blood lead concentration up to 1 year after a gunshot wound with a retained bullet. *Am J Epidemiol.* **159(7)**: 683-92.

Murgueytio AM, Evans RG, Roberts D (1998a). Relationship between soil and dust lead in a lead mining area and blood lead levels. *J Expo Anal Environ Epidemiol* **8(2)**: 173-86.

Murgueytio AM, Evans RG, Sterling DA, Clardy SA, Shadel BN, Clements BW (1998b). Relationship between lead mining and blood lead levels in children. *Archives of Environmental Health* **53(6)**: 414-23.

Needleman H.L (1998). Childhood lead poisoning: the promise and abandonment of primary prevention. *American Journal of Public Health* **88 (12)**: 1871-7.

Needleman HL & Bellinger D (1991). The health effects of low level exposure to lead. *Annual Reviews in Public Health* **12**: 111-40.

Needleman HL and Gatsonis C (1990). Low level lead exposure and the IQ of children. *Journal of the American Medical Association* **263**: 673-678.

Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C, Barrett P (1979). Deficits in psychologic and classroom performance of children with elevated dentine levels. *N Eng J Med* **300(13)**: 689-95.

Needleman HL, Riess JA, Tobin MJ, Biesecker GE, Greenhouse JB (1996). Bone lead levels and delinquent behavior. *Journal of the American Medical Association* **275(5)**: 363-9.

Nriagu JO (1983). Lead and lead poisoning in antiquity: Environmental Science and Technology Series, John Wiley & Sons, New York, ISBN NO. 0471-08767-X, *Geochimica et Cosmochimica Acta* **51(1)**: pp 174.

Nriagu JO, Blankson ML, Ocran K (1996). Childhood lead poisoning in Africa: a growing public health problem. *Science of the Total Environment* **181(2)**: 93-100.

Nriagu J, Jinabhai CC, Naidoo R, Coutsoudis A (1997). Lead poisoning of children in Africa III. Kwazulu Natal, South Africa. *Science of the Total Environment* **197(1-3)**: 1-11.

Piomelli S, Corash L, Corash MB, Seaman C, Mushak P, Glover B, Padgett R (1980). Blood lead concentrations in a remote Himalayan population. *Science* **210**: 1135-1137.

Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Flegal KM, Matte TD (1994). The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES). *Journal of the American Medical Association* **272(4)**: 284-91.

Ponka A (1998). Lead in the ambient air and blood of children in Helsinki. *Science of the Total Environment* **219(1)**: 1-5.

Poole C, Smythe LE, Alpers M. (1980). Blood lead levels in Papua, New Guinea children living in a remote area. *Science of the Total Environment* **15**: 17-24.

Raghunath R, Tripathi RM, Sastry VN, Krishnamoorthy TM (2000). Heavy metals in maternal and cord blood. *Science of the Total Environment* **250(1-3)**: 135-41.

Rees D and Schneider H (1993). Para-occupational lead poisoning in Soweto. *South African Medical Journal* **83(6)**: 443.

Republic of South Africa Government Gazette (2003). Joint implementation strategy for the control of exhaust emissions from road-going vehicles in the Republic of South Africa. Notice 3324 of 2003, Vol. 462 No. 25741.

Retief-Steyn F (1976). Lood as Bedryfs en Openbare Gesondheidsgevaar in Suid Afrika, Unpublished M.D. Thesis, University of Pretoria, South Africa.

Rogan WJ and Ware JH (2003). Exposure to lead in children--how low is low enough? *New England Journal of Medicine* **348(16)**: 1515-6.

Röllin HB, Kilroe-Smith TA, Theodorou P (1988). Quality control for analysing lead in blood: evaluation and comparison of participating laboratories. *South African Journal of Science* **84**: 233-4.

Rummo JH, Routh DK, Rummo NJ, Brown JF (1979). Behavioral and neurological effects of symptomatic and asymptomatic lead exposure in children. *Archives of Environmental Health* **34(2)**: 120-4.

Schuhmacher M, Belles M, Rico A, Domingo JL, Corbella J (1996). Impact of reduction of lead in gasoline on the blood and hair lead levels in the population of Tarragona Province, Spain, 1990-1995. *Science of the Total Environment* **184(3)**: 203-9.

Schwarz J and Otto D (1991). Lead and minor hearing impairment. *Archives of Environmental Health* **46(5)**: 300–305.

Schwartz J and Otto D (1987). Blood lead, hearing thresholds, and neurobehavioral development in children and youth. *Archives of Environmental Health* **42(3)**: 153-60.

Selevan SG, Rice DC, Hogan KA, Euling SY, Pfahles-Hutchens A, Bethel J (2003). Blood lead concentration and delayed puberty in girls. *The New England Journal of Medicine* **348(16)**: 1527–1536.

Silva PA, Hughes P, Williams S, Faed JM (1988). Blood lead, intelligence, reading attainment, and behaviour in eleven year old children in Dunedin, *New Zealand Journal of Child Psychology and Psychiatry* **29(1)**: 43-52.

Snodgrass RA (1986). Lead in South Africa. *Journal of the South African Institute of Mining and Metallurgy* **86(4)**: 97-111.

Stromberg U, Lundh T, Schutz A, Skerfving S (2003). Yearly measurements of blood lead in Swedish children since 1978: an update focusing on the petrol lead free period 1995-2001. *Occupational and Environmental Medicine* **60(5)**: 370-2.

Thomas VM, Socolow RH, Fanelli JJ, Spiro TG (1999). Effects of reducing lead in gasoline: an analysis of the international experience. *Environmental Science and Technology* **33(22)**: 3942-3948.

Tong S (1998). Lead exposure and cognitive development: persistence and a dynamic pattern. *Journal of Paediatric and Child Health*. **34(2)**: 114-8.

Tong S, Baghurst PA, Sawyer MG, Burns J, McMichael AJ (1998). Declining blood lead levels and changes in cognitive function during childhood: the Port Pirie Cohort Study. *Journal of the American Medical Association* **280(22)**: 1915-9.

Tong S, von Schirnding YER, Prapamontol T (2000). Environmental lead exposure: a public health problem of global dimensions. *Bulletin of the World Health Organisation* **78(9)**: 1068-77.

United States of America Environmental Protection Agency. Identification of Dangerous Levels of Lead - Final Rule 40 CFR Part 745. Washington, D.C. Available: <http://www.epa.gov/lead/leadhaz.htm> [20 January 2003].

University of Cape Town, Children's Institute. Fact Sheet on Childhood Poverty, 2003.

Vittinghoff E, Shiboski SC, Glidden DV, McCulloch CE (Eds) (2005). *Regression Methods in Biostatistics – linear, logistic, survival, and repeated measures models*. Springer Science+Business Media Inc. ISBN 0-387-20275-7.

von Schirnding YER, Bradshaw D, Fuggle RF (1991a). Blood Lead Levels in South African Inner City Children, *Environmental Health Perspectives* **94**: 125-130.

von Schirnding YER, Fuggle RF, Bradshaw D (1991b). Factors Associated with Elevated Blood Lead Levels in Inner-city Cape Town Children. *South African Medical Journal* **79(8)**: 454-456.

von Schirnding YE, Fuggle RF (1986). Zinc Protoporphyrin Levels and Lead Absorption in Children Attending South African Schools in the Cape Peninsula, South Africa. *The Southern African Journal of Epidemiology and Infection* **1**: 11-15.

von Schirnding YE, Fuggle RF (1984). A Study of the Relationship Between Low Level Lead Exposure and Classroom Performance in South African Children. *The International Journal of Biosocial Science* **6(2)**: 97-106.

von Schirnding Y, Mathee A, Kibel M, Robertson P, Strauss N, Blignaut R (2003). A study of pediatric blood lead levels in a lead mining area in South Africa. *Environmental Research* **93(3)**: 259-63.

von Schirnding Y, Mathee A, Robertson P, Strauss N, Kibel M (2001). Distribution of blood lead levels in schoolchildren in selected Cape Peninsula suburbs subsequent to reductions in petrol lead. *South African Medical Journal* **91(10)**: 870-2.

Wang L, Xu SE, Zhang GD, Wang WY (1989). Study of lead absorption and its effect on children's development. *Biomed Environ Sci* **2(4)**: 325-30.

Woolley DE (1984). A perspective of lead poisoning in antiquity and the present. *Neurotoxicology* **5(3)**: 353-61.

World Health Organisation (1985) Measuring Change in Nutritional Status. Guidelines for Assessing the Nutritional Impact of Supplementary Feeding Programmes for Vulnerable Groups. Geneva.

Wu T, Buck GM, Mendola P (2003). Blood lead levels and sexual maturation in U.S. girls: the Third National Health and Nutrition Examination Survey, 1988-1994. *Environmental Health Perspectives* **111(5)**: 737-41.

Yan C, Wu S, Shen X, Zhang Y, Jiang F, Yin J, Zhou J, He J, Ao L, Zhang Y, Li R (2002). The trends of changes in children's blood lead levels since the introduction of lead free gasoline in Shanghai. *Zhonghua Liu Xing Bing Xue Za Zhi* **23(3)**: 172-4.

Zejda JE, Sokal A, Grabecki J, Panasiuk Z, Jarkowski M, Skiba M (1995). Blood lead concentrations in school children of Upper Silesian Industrial Zone, Poland. *Cent Eur J Public Health* **3(2)**: 92-6.

# ANNEXURE 1 - THE QUESTIONNAIRE

**Main/Eng** Study Code \_\_\_\_\_



## MEDICAL RESEARCH COUNCIL SCHOOLS ENVIRONMENT & HEALTH STUDY

This questionnaire is part of a Medical Research Council environment & health study being conducted at your child's school. We ask that you take the time (about 30 minutes) to complete the questionnaire, and then send it back to the school with your child tomorrow. We thank you in advance for your assistance in this important study.

If you have questions or need further information, please do not hesitate to call Rajeshree Naidoo on 011 643 7403 or 083 454 2611.

**School Name:** .....

**Class:** .....

**SECTION A: BACKGROUND DETAILS**

*In this section we would like to obtain a few background details about your child.*

1. What is the child's first name? .....

2. What is the child's surname? .....

3. What is the home address? .....  
.....

4. What is the telephone number? .....

5. In which year was the child born? .....

6. Is the child a **(please circle correct answer)**

1. Boy

2. Girl

7. What language does the child usually speak at home? **(circle one only)**

1. English

2. Afrikaans

3. Xhosa

4. Sotho

5. Zulu

Other (please specify) .....

8. What is the “race”/population group of the child?

*(This question is being asked because population group is still closely linked to economic status in South Africa, which in turn is closely linked to certain environmental factors.)*

1. Black African
2. Coloured
3. Asian
4. White
5. Other (please specify) .....

9. How does your child usually get to school **(please circle one answer only)**

1. Walk
2. Bus/Taxi
3. Train
4. Private car
5. Bicycle
6. Other (please specify) .....

10. Is this the first time the child is in Grade 1?

1. Yes
2. No
3. Don't know

11. How would you describe the child's schoolwork?

1. Good
2. Average

3. Poor
4. Don't know

12. After school, or during weekends, where does the child play most of the time? (circle one answer only)

1. Inside the house
2. Outside the house
3. Street
4. At some other place (please specify) .....
5. Don't know

13. How long has the child lived in this house?

.....years

14. Where did the child live before she/he came to live here?

1. An urban area
2. A rural area
3. has lived here all of his/her life

15. Did the child attend a crèche or play-school before he/she went to school?

1. Yes
2. No
3. Don't know

16. Would you say he/she is **more** active than other children of the same age?

1. Yes
2. No
3. Don't know

17. Would you say he/she is **over-active**?

1. Yes
2. No
3. Don't know

18. Have you noticed the child often sucking his/her fingers or chewing his/her nails?

1. Yes
2. No
3. Don't know

19. Have you ever noticed the child putting non-food objects into his/her mouth?

**(circle one answer only for each item)**

<b>Paint</b>	Yes	No	Don't know
<b>Cement/plaster</b>	Yes	No	Don't know
<b>Sand/Soil</b>	Yes	No	Don't know
<b>Sticks</b>	Yes	No	Don't know
<b>Match sticks</b>	Yes	No	Don't know
<b>Cigarette ends</b>	Yes	No	Don't know

<b>Other</b>	Yes	No	Don't know
--------------	-----	----	------------

20. Does he/she **still** eat non-food items?

1. Yes
2. No
3. Don't know

21. Do you use any pottery dishes for cooking or storing food in?

1. Yes
2. No
3. Don't know

22. What is the child's religion? .....

23. How frequently does the child wash his/her hands on the following occasions:

Before eating	Always	Sometimes	Never
<b>After playing in dusty areas</b>	Always	Sometimes	Never
<b>Before going to sleep</b>	Always	Sometimes	Never

## **SECTION B: HEALTH AND DIET**

*In this section some information about the child's health and diet is needed.*

1. Is the child well at present?

1. Yes
2. No
3. Don't know

2. If the child is **not well**, what are the problems?

.....  
.....

3. Does the child suffer from any of the following? (**circle the relevant health problems**)

1. Abdominal (stomach) pain
  2. Convulsions (fits)
  3. Anaemia
  4. Frequent tiredness
  5. Headaches
  6. Constipation
  7. Diarrhoea
  8. Vomiting
  9. Unable to concentrate properly
  10. Irritability
  11. Weakness and pain in joints/muscles
- Other (please specify).....

4. Has your child ever been given any home remedies for illness or to improve health?

1. Yes
2. No
3. Don't know

5. Does the child normally have a good appetite?

1. Yes
2. No
3. Don't know

6. How often does the child eat tinned/canned food?

1. Often
2. Seldom

7. Does the child often play in places where his/her fingernails get dirty?

1. Yes
2. No

**SECTION C: HOUSING**

*In this section we would like to have some information about the household in which the child is presently living*

1. Is this home:

- 1. Owned
- 2. Rented

2. How would you describe the child's home?

- 1. House
- 2. Flat
- 3. Backyard dwelling
- 4. Informal house (shack)
- 5. Other (please specify) .....

3. Approximately, how old is the child's home?

..... years

4. Is the child's home on or within one block of a **very busy road**?

- 1. Yes
- 2. No

5. How many rooms, not counting the kitchen, bathroom or toilet, does this home have? .....

6. What fuel is used **most** of the time for cooking?

1. Electricity
2. Paraffin
3. Gas
4. Wood
5. Coal
6. Car batteries
7. Other (please specify) .....

7. What fuel is used **most** of the time for heating the home?

1. Electricity
2. Paraffin
3. Gas
4. Wood
5. Coal
6. Car batteries
7. None
8. Other (please specify) .....

8. Does anyone regularly smoke at home?

1. Yes
2. No

9. How many people regularly smoke cigarettes in the home? (At least one cigarette per day at home)

.....

10. Is the home often:

1. Very dusty
2. Slightly dusty
3. Not dusty

11. Do you use an outside toilet?

1. Yes
2. No
3. Don't know

12. Where do you get your water from **most** of the time?

1. Indoor tap
2. Outdoor tap
3. Rainwater tank
4. Borehole
5. River/stream
6. Other

13. What type of plumbing (water pipes) does the home have?

1. Metal
2. Plastic
3. Other (please specify) .....

14. Is there paint peeling from the **inside** walls, doors or windowsills of the home?

1. Yes
2. No
3. Don't know

15. Is there paint peeling from the **outside** walls, doors or windowsills of the home?

1. Yes
2. No
3. Don't know

16. Does the home need major repairs?

1. yes
2. No
3. Don't know

17. Has there been any painting, decorating or renovation in the home during the past year?

1. Yes
2. No
3. Don't know

18. Has there been any major repair work done to the house in the past year?

1. Yes
2. No
3. Don't know

19. How would you describe the traffic in the road in which you live?

1. Busy
2. Quiet
3. Don't know

20. How many cars are owned by people who **permanently** live in the child's house?

.....

21. Does anyone regularly do car repair work at the child's home?

1. Yes
2. No
3. Don't know

22. Does anyone regularly do spray painting at the child's home?

1. Yes
2. No
3. Don't know

23. Are there any of the following pets at the child's home?

1. Cats
2. Dogs
3. Other (please specify) .....

23. Does anyone fix electrical appliances (for example toasters and television sets) here at home?

1. Yes
2. No
3. Don't know

24. Does anyone use lead solder here at home?

1. Yes
2. No
3. Don't know

25. When dusting the house, do you use a:
1. dry cloth
  2. damp cloth (soaked in water only)
  3. damp cloth soaked in water + a cleaning solution. (such as washing powder or Handy Andy)
26. When the house is swept, what is used:
1. a dry broom
  2. a wet mop (soaked in water only)
  3. a wet mop (soaked in water & a cleaning solution such as washing powder or Handy Andy).

**SECTION D: SOCIAL ASPECTS**

*In this section we will ask some questions relating to other people living in this home.*

1. How many people live in this house? .....
2. How many siblings (sisters and brothers) does the child have? .....
3. With whom does the child live (you may circle more than one answer)
  1. Mother and Father
  2. One parent only

- 3. A relative (grandparent, aunt etc.)
- 4. Guardian
- 5. Other (please specify) .....

4. What is the highest education qualification of the child's father/male guardian?

.....

5. What type of job does the child's father/male guardian have?

.....

6. Where does the child's father/male guardian work? (for example a battery factory or school)

.....

7. What is the highest education qualification of the child's mother/female guardian?

.....

8. What type of job does the child's mother/female guardian have?

.....

9. Where does the child's mother/female guardian work? (for example a battery factory or school)

.....

10. Does anyone else (besides the child's mother or father) living in this house have a job?

- 1. Yes
- 2. No
- 3. Don't know

If yes, how many people? .....

What are their jobs?.....

11. Does anyone living in the house work from home?

- 1. Yes
- 2. No
- 3. Don't know

If yes, what do they do? .....

12. Does the child regularly play with any dogs or cats?

- 1. Yes
- 2. No
- 3. Don't know

13. Please list the hobbies of people living in the house (for example fixing cars, welding, make pottery, etc.).

.....

14. What is the total monthly income for this household?

1. R0 to R1000
2. R1001 to R3000
3. R3001 to R5000
4. R5001 to R8000
5. R8001 to 10 000
6. more than R10 000

15. Do you believe that lead in the environment can cause health problems in children?

1. Yes
2. No
3. Don't know

If yes, what health problems can lead cause?

.....

**COMMENT:** Please list any comments about the questionnaire or study.

.....  
.....  
.....

**END OF QUESTIONNAIRE**

**THANK YOU** for answering the questions. Your assistance is highly appreciated.

## ANNEXURE 2 - SUBJECT INFORMATION SHEET



### SUBJECT INFORMATION SHEET

Dear Parent/Guardian

The Medical Research Council is conducting an environment and health study at selected schools in various parts of the country. Specifically, the study will be looking at levels of metals such as lead and manganese in the environment. A study we conducted ten years ago, showed that in certain areas of the country, children may be exposed to levels of lead which can affect their health. We would like now to determine the current situation.

We would appreciate very much the participation of your child in our study. If you agree, we will be requesting you to complete a questionnaire, which should take about 30 minutes. The questionnaire will focus on information about your child's playing habits, school performance, diet, general health status, your house type, and proximity to traffic. A trained research assistant will visit your home and help you with the questionnaire if necessary.

We also ask that you allow us to take a 7ml blood sample from your child (about 1 teaspoon). A professional nursing sister will take the blood sample at school. Sterile, disposable equipment will be used and disposed of immediately, so there is no chance of transfer of infection from one child to another. The technique is safe and there is only a slight prick as the needle passes through the skin. Over the years we have sampled blood from many hundreds of children in this way without any problems. We will measure the concentration of metals such as lead in the blood.

The results of this study will be published, but no names of children will be given to anyone. You may request the results in relation to your child. You are free to withdraw from the study at any time without having to give a reason. Remember that your participation in the study is completely voluntary and not taking part in it, or withdrawing from it, carries no penalty of any sort – schooling will not be influenced.

**If you would like to discuss the study further, or have any questions, please do not hesitate to contact Ms Rajeshree Naidoo in Johannesburg, telephone: 011 643 7403 or 083 454 2611.**

## ANNEXURE 3 - CONSENT FORM



### PARENT/GUARDIAN INFORMED CONSENT

<b>Name and surname of parent/guardian</b>	<b>Name of school</b>
<b>Name and surname of child</b>	<b>Name of class</b>
<b>Street address</b>	<b>Name of teacher</b>
<b>Age of child</b>	<b>Study code</b>

I.....  
 (Full name(s) and surname of parent/guardian)

hereby agree to the participation of my child in the environment and health research project of the Medical Research Council.

I have been informed about the research objectives and I understand that my child will be expected to donate a sample of blood (7 ml = slightly more than 1 teaspoon) that will be taken by qualified medical personnel under aseptic conditions.

I understand also that I will be expected to complete a questionnaire.

I understand that ethical approval for this investigation has been obtained.

I participate in this study on condition that all results will be treated with strict confidentiality.

I acknowledge that the results of this research project will be published in medical and scientific journals. However, my child's name will not be mentioned.

I understand my participation is voluntary, and that I am free to withdraw from the project at any time without prejudice.

Parent/Guardian's Signature.....Date.....