



Estimating the burden of disease attributable to lead exposure in South Africa in 2000

Rosana Norman, Angela Mathee, Brendon Barnes, Lize van der Merwe, Debbie Bradshaw and the South African Comparative Risk Assessment Collaborating Group

Objectives. To estimate the burden of disease attributable to lead exposure in South Africa in 2000.

Design. World Health Organization comparative risk assessment (CRA) methodology was followed. Recent community studies were used to derive mean blood lead concentrations in adults and children in urban and rural areas. Population-attributable fractions were calculated and applied to revised burden of disease estimates for the relevant disease categories for South Africa in the year 2000. Monte Carlo simulation-modelling techniques were used for the uncertainty analysis.

Setting. South Africa.

Subjects. Children under 5 and adults 30 years and older.

Outcome measures. Cardiovascular mortality and disability-adjusted life years (DALYs) in adults 30 years and older and mild mental disability DALYs in children under 5 years.

Results. Lead exposure was estimated to cause 1 428 deaths (95% uncertainty interval 1 086-1 772) or 0.27% (95% uncertainty interval: 0.21 - 0.34%) of all deaths in South Africa in 2000. Burden of disease attributed to lead exposure was dominated by mild mental disability in young children, accounting for 75% of the total 58 939 (95% uncertainty interval 55 413 - 62 500) attributable DALYs. Cardiovascular disease in adults accounted for the remainder of the burden.

Conclusions. Even with the phasing out of leaded petrol, exposure to lead from its ongoing addition to paint, para-occupational exposure and its use in backyard 'cottage industries' will continue to be an important public health hazard in South Africa for decades. Young children, especially those from disadvantaged communities, remain particularly vulnerable to lead exposure and poisoning.

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Lead is a non-biodegradable, non-corrosive heavy metal, the toxic nature of which has been recognised for millennia.¹ However, the range of health effects at low exposures has been recognised only recently. Lead use escalated from the late 1800s, and through its multiplicity of uses has become a worldwide environmental pollutant present in air, dust, soil and water. The many uses include lead in petrol, paint, batteries, candles, crystal glass, cellular telephones, computers, television sets, pottery, ammunition, protective clothing, fishing and wheel-balancing weights, tobacco,² and in South Africa, traditional medicines.³ Human exposure occurs mainly through ingestion and inhalation, and to a small degree through dermal absorption.

Burden of Disease Research Unit, Medical Research Council of South Africa, Tygerberg, Cape Town

Rosana Norman, PhD

Debbie Bradshaw, DPhil (Oxon)

Environment and Health Research Unit, Medical Research Council of South Africa, Johannesburg

Brendon Barnes, MSocSc

Angela Mathee, PhD

Biostatistics Unit, Medical Research Council of South Africa, Tygerberg, Cape Town

Lize van der Merwe, PhD

Corresponding author: R Norman (rosana.norman@mrc.ac.za)

Flegal and Smith⁴ estimated that pre-industrial humans had blood lead concentrations as low as 0.016 µg/dl. In 2000, an estimated 120 million people around the world had blood lead concentrations of between 5 and 10 µg/dl, and about the same number had concentrations greater than 10 µg/dl, a level associated with considerable health risk. Children are particularly vulnerable and 40% of all children had blood lead concentrations above 5 µg/dl and 20% had concentrations ≥ 10 µg/dl, with the great majority (97%) of these living in the developing world.¹

Multiple health effects have been associated with lead exposure including systemic effects such as gastrointestinal effects, raised blood pressure, anaemia, nephropathy, effects on vitamin D metabolism, decreased growth, immune system, nervous system and behavioural/cognitive/IQ effects (and, as a result, multiple social effects including increased risk of violence and substance abuse), nerve conductive effects, hearing loss, developmental and reproductive effects, genotoxicity and death from encephalopathy.¹ However, the strength of the evidence supporting the association of these health effects with exposure to lead varies.

Leaded petrol is one of the worst public health offenders. In countries still using leaded petrol, 90% of the lead in the environment comes from emissions in the form of fine particles that are inhaled and absorbed through the lungs.⁵ In the 1980s in South Africa, petrol lead levels were among the highest in the world. At that time well over 90% of the blood lead levels of Cape Town inner-city first-grade school children exceeded



10 µg/dl⁶ and mean blood lead concentrations ranged from 16 to 18 µg/dl.^{7,8}

In an informal settlement in the Durban metropolitan region, 50% of children had blood lead concentrations above 10 µg/dl, while in a rural area located 90 - 120 km from Durban only 2% of children had blood lead concentrations above this level.³ Blood lead levels among Johannesburg children ranged from 6 to 26 µg/dl, with a mean level of 11.9 µg/dl.⁹ In the lead mining town of Aggeneys in the Northern Cape, and in the comparison rural community of Pella about 40 km away, mean blood lead levels of young children were higher than would be expected in a rural setting (16 µg/dl in Aggeneys and 13 µg/dl in Pella).¹⁰

With the progressive lowering of the maximum permissible petrol lead level since 1986, and the introduction of unleaded petrol in the country in 1996, a decline in blood lead levels has been observed. By 2002, when the maximum permissible petrol lead concentration equalled 0.4 g/l, and unleaded petrol made up around 30% of the petrol market share, blood lead levels in Cape Town first-grade inner-city children had fallen, with 10% having lead levels of 10 µg/dl or higher.¹¹ While efforts are underway to phase out the use of leaded petrol, little attention has been devoted to childhood exposure to lead used in paint. In a recent study,¹² lead-based residential paint was found in 20% of sampled homes in various suburbs of Johannesburg. The present study aimed to estimate the health impact of this environmental pollutant by quantifying the burden of disease attributed to lead exposure in children under 5 and adults 30 years and older in South Africa in 2000.

Methods

Comparative risk assessment (CRA) methodology developed by the World Health Organization (WHO)^{13,14} and applied specifically to lead exposure¹ was used. Exposure was characterised by the population distribution of blood lead concentrations. The concentration of lead in blood was chosen as the exposure variable because it is an objective physiological

measure which is directly related to health outcome and reflects human exposure closely. As differences in blood lead concentrations between males and females are very small, exposure data for men and women were combined.

There were no nationally representative data on blood lead levels, and exposure was assessed separately for urban and rural areas. Exposure data for primary school children (aged 5 - 12 years) were pooled from studies carried out at three urban sites in Cape Town, Johannesburg and Kimberley.^{11,15} For the rural sample,¹⁵ occupational exposures or 'hotspots' (in Aggeneys, a lead mining town where lead levels were unusually high) were excluded. Due to data limitations, these same blood lead concentrations were used for children under 5 years and children 5 - 14 years of age (Table I), although health effects in children 5 - 14 years of age were not quantified in this analysis. For adults, urban exposure data were obtained from maternal blood lead levels in a study of pregnant women in Durban.¹⁶ Blood lead levels in rural adults were assumed to be the same as in rural children (Table I). As recent data were used, data were not corrected downward to adjust for programmes phasing out lead in petrol as was done in the global CRA study.¹

Blood lead levels follow a skewed distribution to the right that can be modelled with a lognormal distribution.¹ In the global study, Prüss-Üstün *et al.*¹ compiled geometric means with standard deviations (SDs) in order to represent population exposures. However, these parameters, based on the natural logarithms of the values, do not estimate the parameters (μ and σ^2) of the lognormal distribution. We calculated estimates of μ and σ^2 for the lognormal distribution using the method-of-moments:

Let m and s^2 be the mean and variance of a sample of lead levels, then μ is estimated as $\ln(m) - \frac{1}{2}\sigma^2$ and the estimate of σ^2 is $\ln(s^2 + m^2) - 2\ln(m)$ where the function $\ln(\cdot)$ is the natural logarithm.

For adults and children separately, the proportion of the population at specified blood lead concentrations in the urban

Table I. Estimates of age-specific mean blood lead levels and standard deviations (µg/dl), South Africa, 2000

Population	Parameter	Age groups (yrs)			Data sources Study site	Year	N		
		0 - 4	5 - 14*	15+					
Urban	Mean	7.5	7.5	7.4	Children (5 - 12 years) ^{11,15} in Cape Town Johannesburg Kimberley Pregnant women in Durban ¹⁶ for adults 15+	2002	429		
	SD	3.31	3.31	3.85					
								2002	382
								2003	355
Rural	Mean	5.5	5.5	5.5	Children (5 - 12 years) ¹⁵	2003	98		
	SD	2.33	2.33	2.33					

*Children 5 - 14 years not included in the analysis.
SD = standard deviation.



and rural populations was then estimated on the basis of the lognormal distribution. These were weighted according to the urban/rural breakdown based on Census 2001¹⁷ data to provide a national estimate of exposure.

The outcomes assessed in this study were similar to those in the global CRA study¹ and included cardiovascular diseases (CVDs) from elevated systolic blood pressure (in adults 30 years and older), and IQ reduction in children under 5 years. A loss of IQ points was considered to be a disease burden when resulting in mild intellectual impairment or mild mental disability (MMD), which was defined as having an IQ score of 50 - 69 points.¹

Anaemia and gastrointestinal effects are associated with extremely high exposures to lead¹ and due to individual variation, only 20% of people with blood lead concentrations above the thresholds,^{1,18} were assumed to develop symptoms in the global CRA study. These outcomes were not included in the local assessment, however, since firstly these conditions do not appear in the South African burden of disease list,¹⁹ and secondly, in the local setting the number of people affected at very high concentrations (with the 20% adjustment) is negligible.

Nephropathy and encephalopathy are associated with even higher exposures to lead, and these exposures were not quantified in the local and global CRA since they occur in extreme cases. Other outcomes that are likely to be causal but were not quantified because of a lack of sufficient evidence on causality included developmental and reproductive system effects, and social consequences of IQ loss.¹ The health risks associated with blood lead concentrations considered in this analysis are summarised in Table II. In the absence of scientific consensus of the lowest level of population risk, a blood lead concentration threshold of 5 µg/dl was used in this analysis for the related health risks.¹

Following the framework of Prüss-Üstün *et al.*¹ in the global assessment, the incidence of MMD resulting from IQ

reduction attributable to lead exposure was estimated and used to calculate lead-induced MMD DALYs in children under 5 years of age. Based on a meta-analysis of several studies, Schwartz²⁰ estimated that a mean loss of 2.57 (standard error (SE) 0.41) IQ points was associated with an increase in blood lead concentration from 10 to 20 µg/dl. The global CRA study consequently assumed a linear relationship of 1.3 IQ points lost per 5 µg/dl increase in blood lead, for blood lead concentrations between 5 and 20 µg/dl. This linear relationship was divided into 3 intervals of 5 µg/dl, and the mean loss of IQ points in each interval was assigned to its mean blood lead concentration.¹ A loss of 3.5 IQ points was assumed for blood lead concentrations ≥ 20 µg/dl for a total of 4 IQ loss categories (Table III).

In order to estimate the incidence of MMD resulting from IQ reduction attributable to lead exposure, it was first necessary to estimate the proportion of the child population in IQ categories corresponding to lead exposure intervals based on the assumption that intelligence in human populations approximates a normal distribution²¹ (Table III). We then estimated the number of children affected (also referred to as the rate of illness) in each IQ-loss category by applying the distribution of blood lead concentrations in the child population. The proportion of children under 5 at indicated blood lead levels was multiplied by the fraction of children within the interval 70+x IQ points, for whom a loss of x points results in a final IQ score of < 70 points.¹ It was assumed that loss of IQ and consequent MMD occurs only once during the first year of life and the rate of illness for the age group < 1 year was divided by 5 in order to estimate incidence rates of MMD in children aged < 5 years.

The standard IQ distribution is based on a population less exposed to additional risk factors for IQ loss which may be more common than exposure to lead in developing countries. Prüss-Üstün *et al.*¹ estimated an adjustment ratio to account for the increased risk of mental disability in developing countries

Table II. Summary of health risks associated with blood lead concentrations considered in this analysis

Mediating outcome	Blood lead concentration threshold (µg/dl)		Description of relationship
	Children	Adults	
IQ reduction (children aged 0 - 4 years)	5	NA	Linear relationship assumed BPb between 5 and 20 µg/dl: loss of 1.3 IQ points per 5 µg/dl increase in BPb BPb above 20 µg/dl: loss of 3.5 IQ points
Increased systolic blood pressure (adults aged 30 years and older)	NA	5	Linear relationship assumed BPb between 5 and 20 µg/dl: increase of 1.25 mmHg for males, and 0.8 mmHg for females per 5 µg/dl increase in BPb. BPb above 20 µg/dl: increase of 3.75 mmHg for males, and 2.4 mmHg for females.

Source: Adapted from Prüss-Üstün *et al.*, 2004.¹
NA = not applicable; BPb = blood lead concentration.



Table III. Proportion of the population with shift in IQ score assuming a normal distribution*

Blood lead levels ($\mu\text{g}/\text{dl}$)	Mean BPb ($\mu\text{g}/\text{dl}$)	Loss of IQ points (loss of 1.3 IQ points per 5 $\mu\text{g}/\text{dl}$ increase in BPb)	IQ loss categories	% of the population with shift in IQ score
5 - < 10	7.5	0.65 = (1.3/2)	70 - 70.65	0.24
10 - < 15	12.5	0.65 + 1.3	70 - 71.95	0.80
15 - < 20	17.5	1.95 + 1.3	70 - 73.25	1.45
≥ 20		3.50	70 - 73.50	1.59

Adapted from Prüss-Üstün *et al.*, 2004.¹
 *Mean IQ score of 100, standard deviation of 15.²¹
 BPb = blood lead concentration.

that would result from anaemia, meningitis, pertussis, hookworm and iodine deficiency.^{22,23} However, MMD caused by protein-energy malnutrition could not be taken into account. In this analysis, the estimated incidence rate per 1 000 children affected by lead-induced MMD was multiplied by the adjustment ratio estimated for the WHO African region which includes South Africa (AFR-E region adjustment ratio 2.01)¹ to yield a national incidence of MMD of 0.96 per 1 000 for children under 5 years of age.

Following standardised global burden of disease (GBD) methodology,^{24,25} years lived with disability (YLDs) due to MMD were calculated using the national incidence, duration derived from the disease modelling tool DisMod II,²⁶ and the MMD Dutch disability weight of 0.290.²⁷ As loss of IQ potentially increases the risk of other diseases and violence-related injuries,^{28,29} and mental disability has been associated with lower life expectancy,^{1,30} a relative risk of total mortality for mental disability of 2 (double that for the total population) was used. The duration of disability was estimated at 42.6 years for males and 48.4 years for females. MMD YLDs in children under 5 years were directly attributed to lead exposure.

The contribution of exposure to lead to CVD burden in adults is mediated through increased blood pressure. Hazards for increased blood pressure associated with increased blood lead concentrations were obtained from a meta-analysis by Schwartz³¹ and a published analysis of data from the second National Health and Nutrition Examination Survey (NHANES II).¹ As in the global CRA study, the relationship was assumed to be linear between 5 and 20 $\mu\text{g}/\text{dl}$, with a 1.25 mmHg^{1,31} increase in systolic blood pressure for each increase of 5 $\mu\text{g}/\text{dl}$ in blood lead concentration in men and an increase of 0.8 mmHg³² for each 5 $\mu\text{g}/\text{dl}$ increase in women (Table II).¹ Using the same method used for loss of IQ points, the linear increase was converted into 3 equal intervals using the midpoints of the intervals with the corresponding increase in blood pressure. An increase in blood pressure of 3.75 mmHg for males and 2.4 mmHg for females was used for blood lead concentrations ≥ 20 $\mu\text{g}/\text{dl}$ (Table II) for a total of 4 increased systolic blood pressure categories. The number of people affected in each category was determined from the distribution

of blood lead concentrations in the population.

The burden of cardiovascular disease attributable to lead exposure in adults 30 years and older was calculated using the population-attributable fraction (PAF) formula below:

$$PAF = \frac{\sum_{i=1}^k p_i (RR_i - 1)}{\sum_{i=0}^k p_i (RR_i - 1) + 1}$$

where p_i is the rate of illness estimated for the i th blood pressure category corresponding to each blood lead interval, RR_i is the relative risk of disease for exposure level i , and k is the total number of exposure levels. The risk values for CVD, for the defined increases in systolic blood pressure associated with increased blood lead concentrations, were based on risk ratios obtained from a collaborative meta-analysis of individual participant data from 61 separate prospective studies³³ used in the high blood pressure assessment.³⁴ PAFs were then applied to revised South African burden of disease estimates for deaths, years of life lost (YLLs) due to premature mortality, YLDs and DALYs¹⁹ in 2000 for the CVD categories to calculate attributable burden.

Customised MS Excel spreadsheets to calculate the burden attributable to lead exposure were based on templates used in the WHO global CRA study (A Prüss-Üstün, WHO – personal communication, 2005). Monte Carlo simulation-modelling techniques were used to present uncertainty ranges around point estimates reflecting the main sources of uncertainty in the calculations. The @RISK software version 4.5 for Excel³⁵ was used, which allows multiple recalculations of a spreadsheet, each time choosing a value from distributions defined for input variables. Lognormal distributions of the mean blood lead concentration were specified by age and sex. For the association of increased blood lead concentrations with loss of IQ points, a normal distribution with a mean of 100 and SE of 1 (i.e. 95% of the simulated means will lie between 98 and 102 points) was specified for the mean IQ in human populations. A normal distribution was also specified around the estimated decrease in IQ points associated with a 5 $\mu\text{g}/\text{dl}$ increase in blood lead concentration, with SE derived from that published in the meta-analysis.²⁰ For the association between systolic



blood pressure and blood lead, a normal distribution was specified around the risk estimates^{31,32} and the SEs of these risk estimates based on the 95% confidence interval (CI) of 0.87 - 1.63.³¹ For the CVD relative risk input variables a normal distribution was specified around the logged point estimate and its SE derived from the published 95% CI.³³ For each of the output variables (namely attributable burden as a percentage of total burden in South Africa in 2000), 95% uncertainty intervals were calculated bounded by the 2.5th and 97.5th percentiles of the 2000 iteration values generated.

Results

It was estimated that 23 million South Africans had blood lead concentrations between 5 and < 10 µg/dl in the year 2000, and about 4 million people had blood-lead concentrations ≥ 10 µg/dl (Table IV). Blood lead concentrations in rural populations were lower than those in urban populations. Seventy-seven per cent of urban children had blood lead concentrations ≥ 5 µg/dl, 18.6% had concentrations ≥ 10 µg/dl, and 0.6% had concentrations ≥ 20 µg/dl. About half of rural children had blood lead concentrations ≥ 5 µg/dl, only 5% had concentration ≥ 10 µg/dl, and effectively no rural children had levels ≥ 20 µg/dl.

The number of people whose health is affected by exposure to lead, estimated from the fraction of the population having blood lead concentrations at which health effects occur, is presented in Table V. These rates of illness were used to calculate the incidence of MMD in children under 5 and PAFs for CVD in adults. The incidence rate of MMD due to lead exposure was estimated at 4.82 per 1 000 in children under 1 and at 0.96 (95% uncertainty interval: 0.56 - 1.41) per 1 000 in the 0 - 4-year age group (data not presented). Using this incidence, an estimated 43 986 DALYs due to MMD were causally attributed to lead exposure. Assuming that each cohort of children aged 0 - < 1 year is exposed to the same amount of lead year after year, the prevalence of MMD attributable to lead was estimated at 0.5% in children aged 0 - 4.

CVD PAFs were lower in females due to the weaker relationship between systolic blood pressure and blood lead concentration in females compared with males (Table VI). Overall, an estimated 1 428 deaths (95% uncertainty interval 1 086 - 1 772) or 0.27% of all deaths in South Africa in 2000 could be attributed to lead exposure (Table VI). Attributable DALYs were slightly higher, 58 939 (95% uncertainty interval 55 413 - 62 500), or between 0.34% and 0.39% of all DALYs in South Africa in 2000 attributable to lead exposure. The MMD burden in children under 5 years accounted for 74.6% of the total DALYs attributable to lead exposure, while ischaemic heart disease (7.0%), stroke (10.4%), hypertensive disease (7.6%) and other CVDs (0.4%) accounted for much smaller proportions of the total attributable burden (Fig. 1).

Discussion

These estimates of attributable burden indicate that although lead reduction programmes have been initiated in South Africa, these have not yet been fully implemented and as such this environmental risk still had a significant impact on health in 2000. Large fractions of the local population had significantly elevated blood lead concentrations and more than half (53%) of the national population (about 23 million) had blood lead concentrations between 5 and 10 µg/dl. About 10% of the population (almost 4 million South Africans) were estimated to have blood lead concentrations above 10 µg/dl. Rural populations in South Africa were also exposed to lead, although to a somewhat lower degree.

It should be noted that significant assumptions were necessary to overcome the lack of nationally representative exposure data. In urban adults, exposure data were obtained from a single community study in females and extrapolated to the whole country. Rural adults were assumed to have the same mean blood lead concentration as rural children, and this may lead to an overestimation of exposure in rural adults since adult exposures are usually lower than those in children. Nationally representative blood lead surveys are necessary in our country in order to improve the accuracy of these estimates.

It was estimated that lead exposure resulted in a burden of disease of about 44 000 DALYs caused by MMD, and about 13 700 YLLs and 15 000 DALYs caused by CVD. These 2 disease outcomes together accounted for between 0.34% and 0.39% of all DALYs in South Africa in 2000. The burden of ischaemic heart disease, stroke, hypertensive disease and other CVDs attributable to lead exposure constituted about 1.5% of the total CVD burden in South Africa in 2000.

This estimate of burden attributable to lead exposure is probably an underestimate of the true attributable burden since only MMD caused by IQ reduction and CVD endpoints were considered in this analysis. The burden of gastrointestinal symptoms and anaemia caused by lead was not assessed since the rate of illness for these related health outcomes was negligible. In the global assessment the contribution of lead to burden from gastrointestinal effects and anaemia was considered small compared with that caused by risk factors such as unsafe water, poor sanitation and hygiene, and iron deficiency. Several additional related health outcomes such as increased delinquent behaviour and its impact on injuries could not be quantified in this assessment due to insufficient evidence. In addition, lack of information on the health impact of low lead levels, the exclusion of data from studies carried out at 'hot spots' with high exposure as well as other conservative assumptions listed in this study have all contributed to a possible underestimation of the burden of disease attributable to lead exposure.



Mean blood lead concentrations in the South African population were slightly lower than the AFR-E estimates in both children and adults for 2 main reasons. Firstly, more recent and locally representative data were used in this analysis. Secondly, the method used in the estimation of parameters for the distribution of blood lead concentrations in the global assessment¹ yielded inflated estimates of the SDs. The proportion of children affected and the rate of illness at the lower blood lead concentrations in this study was several-fold higher than the rate estimated for AFR-E, while that at high blood lead concentrations ($\geq 20 \mu\text{g}/\text{dl}$) was much

lower. The global assessment method of parameter estimation underestimates the percentage of people at lower blood lead concentrations but overestimates the proportion at risk at higher blood lead concentrations.

Nevertheless, the estimated prevalence of MMD attributable to lead exposure (0.5%) was comparable to that estimated for the AFR-E region.¹ It has been estimated that 40% of MMD is of genetic origin, 20% is caused by environmental factors and 40% is of unknown aetiology³⁶ and in developing countries the contribution of lead to the total incidence of MMD is thought to be as high as 15 - 20%.¹ The high prevalence of MMD in

Table IV. Proportion of the urban and rural population at indicated blood lead levels, South Africa, 2000

Blood lead concentrations	Population	Age group (yrs)		
		0 - 4	5 - 14	15+
BPb 5 - < 10 $\mu\text{g}/\text{dl}$	Urban (%)	58.7	58.7	58.0
	Rural (%)	46.6	46.6	46.6
	National (%)	53.1	53.1	52.7
	Population affected (N)	2 805 238	4 895 283	15 526 112
BPb 10 - < 15 $\mu\text{g}/\text{dl}$	Urban (%)	15.4	15.4	13.1
	Rural (%)	4.3	4.3	4.3
	National (%)	10.3	10.3	9.0
	Population affected (N)	542 897	947 383	2 656 284
BPb 15 - < 20 $\mu\text{g}/\text{dl}$	Urban (%)	2.6	2.6	2.0
	Rural (%)	0.3	0.3	0.3
	National (%)	1.6	1.6	1.2
	Population affected (N)	82 816	144 518	363 523
BPb $\geq 20 \mu\text{g}/\text{dl}$	Urban (%)	0.60	0.60	0.40
	Rural (%)	0.00	0.00	0.00
	National (%)	0.32	0.32	0.23
	Population affected (N)	16 789	29 297	66 697

BPb = blood lead concentration.

Table V. Number of people affected (per 1 000 population) by loss of IQ points and increased systolic blood pressure with increased blood lead concentrations caused by exposure to lead, South Africa, 2000

Mediating outcome	Rate per 1 000 population
IQ loss categories	
<i>Children < 1 years</i>	
IQ (1) - loss of 0.65 points (5 - < 10 $\mu\text{g}/\text{dl}$ BPb)	531.0*
IQ (2) - loss of 1.95 points (10 - < 15 $\mu\text{g}/\text{dl}$ BPb)	102.8*
IQ (3) - loss of 3.25 points (15 - < 20 $\mu\text{g}/\text{dl}$ BPb)	15.7*
IQ (4) - loss of 3.5 points ($\geq 20 \mu\text{g}/\text{dl}$ BPb)	3.2*
Increased systolic blood pressure categories	
<i>Males (≥ 15 yrs)[†]</i>	
BP (1) + 0.625 mmHg (5 - < 10 $\mu\text{g}/\text{dl}$ BPb)	526.9
BP (2) + 1.875 mmHg (10 - < 15 $\mu\text{g}/\text{dl}$ BPb)	90.1
BP (3) + 3.125 mmHg (15 - < 20 $\mu\text{g}/\text{dl}$ BPb)	12.3
BP (4) + 3.75 mmHg ($\geq 20 \mu\text{g}/\text{dl}$ BPb)	2.3
<i>Females (≥ 15 yrs)[†]</i>	
BP (1) + 0.4 mmHg (5 - < 10 $\mu\text{g}/\text{dl}$ BPb)	526.9
BP (2) + 1.2 mmHg (10 - < 15 $\mu\text{g}/\text{dl}$ BPb)	90.1
BP (3) + 2.0 mmHg (15 - < 20 $\mu\text{g}/\text{dl}$ BPb)	12.3
BP (4) + 2.4 mmHg ($\geq 20 \mu\text{g}/\text{dl}$ BPb)	2.3

*Values presented were those estimated for the age group less than 1 year. These were divided by a factor of 5 (as the age group 0 - 4 years includes five 1-year cohorts of children) in order to estimate incidence rates of MMD in children aged < 5 years.

[†]Same rates of illness assumed for adults 30+.

BP = blood pressure (systolic); BPb = blood lead concentration.



to identify emerging risk factors for elevated blood lead concentrations in children as well as adults (especially with regard to 'cottage industries' and the use of lead in cultural/traditional practices), significant scaling up of awareness programmes to address lack of knowledge of the sources and hazards of lead, and initiatives, in the context of a resource-poor country such as South Africa, to address the problem of the estimated 25% of homes and schools that are currently coated with lead-based paint. Testing needs to be carried out to identify high-risk buildings, and provision should be made for the safe management of lead-based paint in the worst-affected dwellings and schools using a combination of in-place containment and more permanent removal methods.¹²

Given the associations between lead exposure and poor school performance as well as delinquent behaviour, it is extremely important for South Africa to set standards for children's blood lead levels and to develop protocols to respond to children with elevated blood lead levels.

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