REVIEWS ON ENVIRONMENTAL HEALTH VOLUME 25, NO. 2, 2010

Estimating the Burden of Disease Attributable to Four Selected Environmental Risk Factors in South Africa

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Summary: *Introduction*: The first South African National Burden of Disease study quantified the underlying causes of premature mortality and morbidity experienced in South Africa in the year 2000. This was followed by a Comparative Risk Assessment to estimate the contributions of 17 selected risk factors to burden of disease in South Africa. This paper describes the health impact of exposure to four selected environmental risk factors: unsafe water, sanitation and hygiene; indoor air pollution from household use of solid fuels; urban outdoor air pollution and lead exposure. *Methods*: The study followed World Health Organization comparative risk assessment methodology. Population-attributable fractions were calculated and applied to revised burden of disease estimates (deaths and disability adjusted life years, [DALYs]) from the South African Burden of Disease study to obtain the attributable burden for each selected risk factor. The burden attributable to the joint effect of the four environmental risk factors was also estimated taking into account competing risks and common pathways. Monte Carlo simulation-modeling techniques were used to quantify samplin$_{\mathrm{b}}$ uncertainty. *Results*: Almost 24 000 deaths were attributable to the joint effect of these four environmental risk factors, accounting for 4.6% (95% uncertainty interval 3.8-5.3%) of all deaths in South Africa in 2000. Overall the burden due to these environmental risks was equivalent to 3.7% (95% uncertainty interval 3.4-4.0%) of the total disease burden for South Africa, with unsafe water sanitation and hygiene the main contributor to joint burden. The joint attributable burden was especially high in children under 5 years of age, accounting for 10.8% of total deaths in this age group and 9.7% of burden of disease. *Conclusion*: This study highlights the public health impact of exposure to environmental risks and the significant burden of preventable disease attributable to exposure to these four major environmental risk factors in South Africa. Evidence-based policies and programs must be developed and implemented to address these risk factors at individual, household, and community levels.

Key words: unsafe water sanitation and hygiene, indoor air pollution from household use of solid fuels, lead exposure, urban outdoor air pollution, DALYs, comparative risk assessment

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INTRODUCTION

The first South African National Burden of Disease (SA NBD) study identified the underlying causes of premature mortality and morbidity experienced in South Africa in the year 2000 /1/. The initial estimates were revised based on additional data to estimate the disability-adjusted

life-years (DALYs) for single causes for the first time /2/. The DALYs are a comprehensive measure of disease burden combining the years of life lost (YLL) from premature mortality and years lived with disability (YLD) related to illness or injury /3/.

The SA NBD study was followed by a Comparative Risk Assessment (CRA) to estimate the contributions of 17 selected risk factors to the burden of disease in South Africa in 2000 /4/. The risk factors ranged from unsafe sex to vitamin A deficiency and were selected based on their contribution to the burden of disease and the input of a range of stakeholders /4/. Because the environment plays a significant role in the disease profile of a country, the CRA study also included four selected environmental risk factors: unsafe water, sanitation and hygiene; indoor air pollution from household use of solid fuels; urban outdoor air pollution, and lead exposure. Unsafe water, sanitation, and hygiene accounted for 2.6% of total DALYs in South Africa in 2000, whereas indoor smoke from solid fuels and lead exposure accounted for 0.4% each and urban air-pollution accounted for 0.3% of all DALYs in 2000 /4/.

Living in a middle-income country, South Africans are simultaneously at risk of ill health related to industrialization and to under-development. Populations in industrially developed urban settings are exposed to urban air pollution, lead, and other heavy metals, whereas those in poor peri-urban and rural settlements face additional environmental risks from inadequate access to water and sanitation and from indoor smoke from solid fuels.

That poor communities continue to suffer disproportionately high exposures to environmental risks is an issue of environmental justice. The location of industry and working-class communities in close proximity served the rapid industrial growth in the 1960s and 1970s. Yet, population growth in such communities on the one hand and increasing production by industries on the other hand has led to a major environmental challenge for the country as a whole /5/. In a number of 'hot-spot' areas, large industrial sources located close to poor communities result in high exposures and pose a danger to public health. Such exposures include sulfur dioxide (SO_2) emissions from oil refineries in the South Durban industrial basin and Cape Town, dust emissions from mine tailings in Gauteng (one of the more developed urban provinces), and SO_2 emissions from steel and chemical plants in the Vaal Triangle (an area characterized by a high concentration of industries).

This paper reviews the work carried out on the quantification of the health impact of exposure to unsafe water, sanitation, and hygiene /6/; indoor air pollution from household use of solid fuels /7/; urban outdoor air pollution /8/; and lead exposure /9/ in South Africa in 2000. This report constitutes the first national application of World Health Organization (WHO) Comparative Risk Assessment (CRA) methodology /10,11/ in Africa. In addition, the paper aims to estimate the joint effect of the four selected environmental risk factors. Dealing with the risk factors jointly is of particular relevance from a policy perspective. For both indoor and outdoor air pollution, the measures to combat these factors are closely linked. When trying to deal with indoor air pollution, policy makers turn to electricity as the solution without considering the impact of electricity generating plants on outdoor air pollution.

MAJOR ENVIRONMENTAL RISK FACTORS IN SOUTH AFRICA

Urban Outdoor Air Pollution

Outdoor air pollution (even at low ambient concentrations) is associated with outcomes such as respiratory symptoms, reduced lung function, and chronic bronchitis. Some health effects may be related to short-term exposure, while others are related to long-term exposure /12/.

Anthropogenic ('man-made') air pollution is a complex mixture with many toxic components. The commonly found air pollutants are SO_2, oxides of nitrogen (NO_x), ozone (O_3), volatile organic compounds, and suspended particulate matter (PM). A recent review of air pollutants in South Africa /13/ observed that the main anthropogenic sources of PM emissions are motor vehicles, industries burning dirty fossil fuels (coal, fuel oil and diesel) in appliances that generally do not have emission control devices, and domestic use of highly polluting coal, wood, and paraffin in un-electrified areas, mainly underdeveloped rural areas and peri-urban settlements.

Local studies have demonstrated measurable health effects of petrochemical emissions and other industrial air pollution. In a study among the community living in the north-west quarter of the City of Cape Town in the vicinity of a petro-chemical refinery producing approximately 18 tons of SO_2 daily, White et al. /14/ showed a measurable health effect, with more frequent asthmatic symptoms in schoolchildren associated with meteorologically estimated petrochemical emissions dose. In this study area, petrochemical refinery emissions were the most important risk factor for allergic disease symptoms in the ambient environment /14/.

Another study of respiratory conditions in children living in areas exposed to high levels of community air pollution found increased odds of 1.3 compared with areas with less pollution /15/. Opperman et al. /16/ found a high prevalence (65.9%) of upper respiratory illness in children 8-12 years of age in the Vaal Triangle, an area with high total suspended particulates (annual average 184 $\mu g/m^3$ in 1992). The South Durban Health Study /5/ found that relatively moderate ambient concentrations of NO_2, NO, PM_{10}, and SO_2 were strongly and significantly associated with reduced lung function among children with persistent asthma.

Indoor Air Pollution from Household Use of Solid Fuels

Although attention to air pollutant emissions is dominated by outdoor sources, human exposure is a function of the level of pollution in places where people spend most of their time /11,17-19/. Human exposure to air pollution is therefore dominated by the indoor environment. The use of solid fuels for cooking and heating is probably the largest traditional source of indoor air pollution globally—nearly half the world continues to cook with solid fuels, such as dung, wood, coal and agricultural residues. This risk group includes more than 75% of the populations in India and China and 50% to 75% of those in certain regions of South America and Africa.

In South Africa, nationally representative data on household energy are available from two sources—the Demographic and Health Survey of 1998 (SADHS 1998) /20/ and the national Census of 2001 /21,22/. Both data sources indicate that the distribution of households by main energy source used for cooking or heating differs markedly by population group and province. The population group classification is used in this review to demonstrate differences in the risk factor profile and the subsequent burden. The data are based on self-reported categories according to the population group categories used by Statistics South Africa. Such mentioning of differences allows for a more accurate estimate of the overall burden and may assist in higher effectiveness of future interventions. The authors do not subscribe to this classification for any other purpose.

About one-third of households in the country used solid fuels (wood, coal and dung) for cooking and heating, and of these households, 95% were black African /21,22/. A further one in five used paraffin (kerosene), and a very small proportion (less than 3%) used gas for cooking and heating. In 2001 almost 60% of households in Limpopo, a

predominantly rural province, used wood as the main source of energy for cooking (almost three times the national average), whereas in the more developed province of Gauteng, less than 1% of households used wood for cooking /21,22/.

Poorly designed and manufactured stoves and fireplaces burning solid fuels, as well as agricultural fires, emit significant quantities of health-damaging pollutants and carcinogenic compounds /23,24/. Limited ventilation is common and increases exposure, particularly for women and young children who spend much of their time indoors.

In South Africa, a few studies have raised concern about the association between indoor air pollution and acute lower respiratory infections (ALRIs) such as pneumonia, among children younger than 5 years of age. ALRIs are among the top 4 killers of South African children in this age group /25,26/. As early as 1982, Kossove found that of 132 infants with severe lower respiratory tract disease treated in an outpatient clinic, 70% were exposed to daily levels of smoke from cooking and heating, whereas only 33% of 18 infants free of respiratory illness were exposed to smoke (odds ratio (OR) > 4) /27/. Similarly, not using electricity for cooking and heating, as well as living in areas that are exposed to high levels of both indoor and outdoor air pollution, were found to be associated with acute respiratory infections in children /15,28,29/.

Another study among poor communities living in the Eastern Cape province showed a possible association between high levels of recurring respiratory symptoms among children and high levels of indoor air pollution (with levels of carbon monoxide (CO), SO_2, and NO_2 up to 12 times those of international guidelines) /30/. One of the most comprehensive South African studies, the Vaal Triangle Air Pollution Study (VAPS), highlighted, among others, high levels of air pollution in coal-burning urban areas, as well as the risk to upper and lower respiratory health associated with exposure /31,32/. Among rural children, the VAPS study also highlighted a significantly elevated risk of developing acute respiratory infection (OR > 5) among those living in wood- and coal-burning homes /33/. In a recent re-analysis of SADHS 1998 data, exposure to cooking and heating smoke from polluting fuels (paraffin included) was significantly associated with under-5 mortality after controlling for mother's age at birth, water source, asset index, and household density /34/. A study of indoor air quality among paraffin-burning urban households revealed that 42% exceeded the 1-hour guidelines for SO_2, 30% for CO, and 9% for NO_2 /35/. Baseline monitoring of PM_{10} in the more rural North West province showed that 68% of wood- and cow dung-burning households exceeded the United States Environmental Protection Agency (24-hour) guidelines, in some instances by a factor of 20 /36/. With the exception of the study by Wesley and Loening /37/, all of those published showed positive associations between indoor air pollution and child ALRIs. The majority of studies reported ORs between 1.88 and 3.5, comparable with other studies in low and middle income countries (ORs 2-3) /38/.

Unsafe Water, Sanitation, and Hygiene

Unsafe water, sanitation, and hygiene (WSH) remains a concern in South Africa. Census 2001 data indicate that 13.6% of households have no toilet facility /21/. A further 4.1% and 22.8% respectively use bucket and pit latrines with no ventilation—both inadequate forms of sanitation that increase risk of diarrheal and parasitic diseases /39-42/. The proportion of households with inadequate facilities is much higher in the Eastern Cape, KwaZulu-Natal, and Limpopo, the more rural provinces of South Africa.

Although most households have access to piped water (84.5%), the location is more than 200 m away for 12.4% of households, whereas 7.5% still use rivers, streams, or dams for drinking water,

placing residents at risk for diseases such as schistosomiasis /43/. Again the variation is wide, with 23.0% and 12.9% of households in the Eastern Cape and KwaZulu-Natal, respectively, using rivers or streams as their main water source /21/. The risk of exposure to infections as a result of inadequate water and sanitation is not limited to the home environment. In 2002, the Department of Water Affairs and Forestry /44/ estimated that 15% of clinics and nearly 12% of schools in South Africa were without sanitation.

Unsafe WSH as a risk factor is particularly important from a policy perspective because knowledge of how to reduce exposure through improving water and sanitation facilities and hygiene, and the effects on diarrheal and other illnesses of doing so, are fairly well developed /45-47/. Significant synergistic effects of improving WSH can be achieved, in terms of improving nutritional status /48/, reducing poverty, and promoting development /49,50/. Access to adequate basic facilities is also a rights issue and clearly interpreted as such within the South African Constitution. Internationally the Millennium Development Goals (MDGs) aim to halve the proportion of people without sustainable access to basic sanitation and safe drinking water by 2015, and South Africa has committed itself to contributing to this effort /51/. It is therefore of concern that in 2015, one in five and one in four South African households, respectively, are projected to have inadequate access to water and sanitation facilities /52/. Most of these homes are rural households, further contributing to rural urban inequalities.

In South Africa diarrheal diseases account for 3.1% of total deaths—the eighth largest cause of death nationally /53/. Among children under 5 years of age, diarrheal diseases are the third largest cause of death (11.0% of all deaths), and the third greatest contributor to the burden of disease, constituting 8.8% of all DALYs in this age group /53/. Survey data show that 13% of under-5s in South Africa were reported to have had a bout of diarrhea in the preceding 2 weeks /54/. Although the proportion of deaths attributable to diarrheal diseases in under-5s in 2000 was substantially lower than the 27.7% reported for 1984 /55/, the figures are still a cause for concern.

The WHO estimates that 0.75 cases of diarrhea per person occur worldwide every year. This rate varies between regions, with sub-Saharan Africa having the highest rate of 1.29 cases per person annually. In contrast, the respective rates in Europe and the United States are 0.18 and 0.07 cases per person per year /56/. In South Africa, the estimated incidence of diarrheal disease in under-5s in 2004, based on cases presenting to primary health facilities (and therefore likely to be an underestimate of true incidence), was 128.7/1000, with wide variations between provinces, from 8.1/1000 in Gauteng to 244.2/1000 in KwaZulu-Natal /57/. These differentials indicate a potential for reducing the disease burden through improvements in the provision of water and sanitation services and changes in hygiene behavior.

Lead Exposure

Through its multiplicity of uses, lead has become a worldwide environmental pollutant 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 /58/, and in South Africa, traditional medicines /59/. Human exposure occurs mainly through ingestion and inhalation, and to a small degree through dermal absorption.

Flegal and Smith /60/ 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 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; in 2004, WHO reported that worldwide 40% of all children had blood lead concentrations above 5 μg/dL and 20% had concentrations ≥ 10 μg/dL, with the vast majority (97%) living in low and middle-income countries /61/.

In countries still using leaded petrol, 90% of environmental lead comes from emissions in the form of fine particles that are inhaled and absorbed through the lungs /62/. In South Africa, petrol lead levels in the 1980s 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/dL /63/ and mean blood lead concentrations ranged from 16 to 18 μg/dL /64,65/.

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 reported. By 2002, when the maximum permissible petrol lead concentration equaled 0.4 g/dL 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 /66/. Although the use of leaded petrol was finally phased out in 2006, little attention has been devoted to childhood exposure to lead used in paint. In a 2005 study /67/, lead-based residential paint was found in 20% of sampled homes in various suburbs of Johannesburg.

METHODS

The disease burden attributable to the four important environmental risk factors was estimated by comparing the current local health status with a theoretical minimum counterfactual with the lowest possible risk. The approach for estimating the burden of disease attributable to exposure to these environmental risk factors is described in more detail elsewhere /6-9/. Briefly, the attributable fraction of disease burden in the population was determined by the prevalence of exposure to the risk factor in the population and the relative risk (RR) of disease occurrence given exposure. Population attributable fractions (PAFs) were then applied to the revised South African burden of disease estimates for 2000 (deaths, years of life lost (YLL), years lived with disability (YLD), and DALYs) /2/ for related health outcomes. For urban air pollution, only attributable mortality estimates were included in this analysis because the mortality effects of air pollution are the most important, and the local incidence data required for morbidity estimates are lacking. For indoor air pollution, PAFs were applied to burden of disease estimates for the year 2000 for each population group /2/. The total attributable burden for South Africa in 2000 was then obtained by adding the burden attributed to indoor smoke for the four population groups. The methodology used for individual risk factors is summarized below.

Urban Outdoor Air Pollution

Particulate matter refers to the total mass of airborne particles, irrespective of their chemical properties. The size of the particulate is important in terms of its ability to penetrate the lungs and cause adverse health effects /68/. Suspended PM is divided into three fractions—$PM_{2.5}$ is PM with aerodynamic diameters < 2.5 μm, PM_{10} is PM with aerodynamic diameters < 10 μm, and suspended PM > PM_{10} with aerodynamic diameters > PM_{10}. $PM_{2.5}$ and PM_{10}, which are most often selected as exposure metrics for the quantitative assessment of health effects in epidemiologic studies /12/, were used in the South African CRA study.

Quantifying the impact of air pollution in cities around South Africa is challenging due to the limited availability of information on exposure to air pollution and its adverse effects on health in our local setting. Air pollution monitoring efforts tend

to focus on 'hot-spot' areas, with only a few stations positioned to monitor population exposure, making difficult the assessment of overall exposure to urban air pollution. At this stage, the national monitoring network is limited, uneven in distribution across the urban population, and not standardized. In general, the monitoring network does not conform to recommended international practice /69/. Nevertheless, we made use of the available data to estimate population exposure to urban outdoor air pollution (in terms of PM_{10} and $PM_{2.5}$) and the mortality burden attributed to this exposure by gender and age group in South Africa for the year 2000 /8/.

Urban areas in this study comprised the six metropolitan areas defined by Statistics South Africa /22/ and the Sasolburg district that falls in the Vaal Triangle. Metropolitan areas are conurbations featuring a high population density; intense movement of people, goods, and services; extensive development; and multiple business districts and industrial areas /22/. The annual mean PM_{10} concentrations were calculated from continuous measurements (mostly hourly) taken in the years 2000 to 2003 by air pollution monitoring networks in the City of Cape Town, City of Johannesburg, Ethekwini (Durban), as well as the Nelson Mandela (Port Elizabeth) metropolitan areas, averaging out monthly and seasonal variations /70/. The monitoring data extracted from a few air-quality studies conducted in Ekurhuleni (East Rand metro) and other urban areas not covered by the network were also used /13/. The Tshwane metropolitan area (Pretoria), accounting for 13.4% of the total metropolitan population, has no air pollution monitoring data and was assumed to have the average exposure of the other areas. The map in Figure 1 shows that the monitoring networks and stations across the country are largely situated in metropolitan areas.

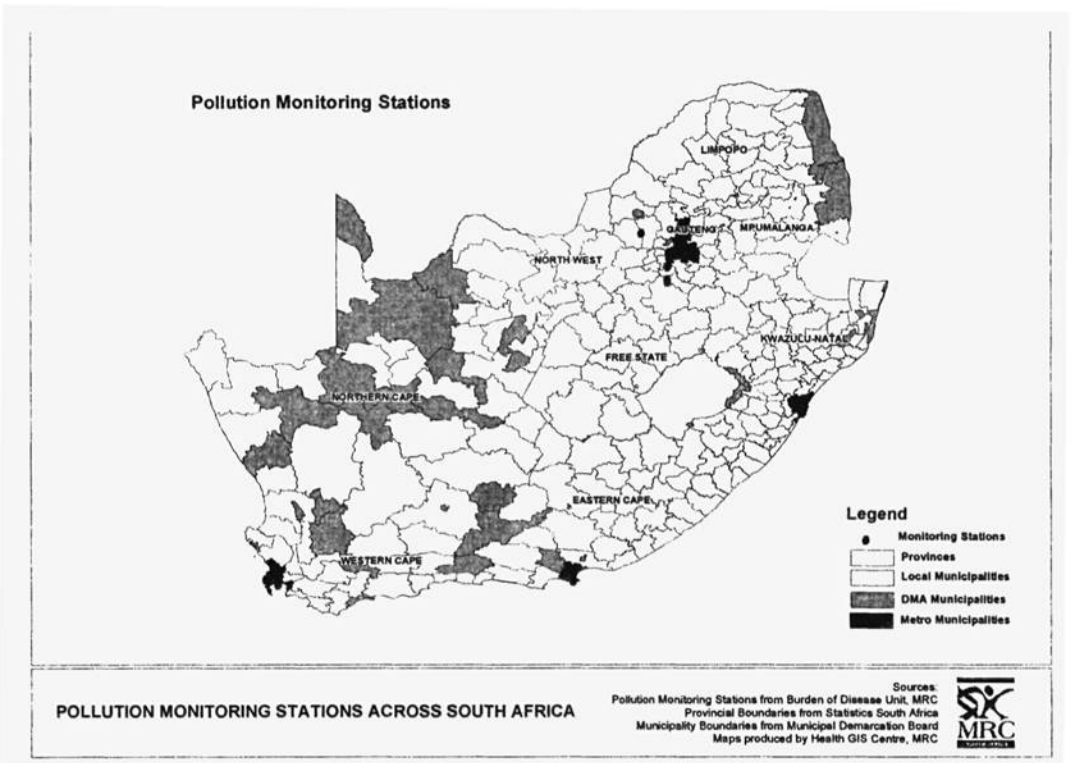

Fig. 1: Map showing monitoring stations (networks and studies) across the country. (Reproduced with permission from South African Medical Journal /8/.)

The PM_{10} estimates were converted to estimates of $PM_{2.5}$ using available information on the geographic variation of pollution sources, factors influencing the ratio of $PM_{2.5}$ to PM_{10}, as well as the observed ratio from local studies for which monitoring data were available for both PM_{10} and $PM_{2.5}$. For these areas, the ratio was between 0.5 and 0.65 /13,71/. In areas without local data on $PM_{2.5}$ /PM_{10} ratios, we assumed a ratio of 0.5. A ratio of 0.35 was assigned to peri-urban areas with high fugitive emissions (for example, dust from unpaved roads or from soil or sand particles) and areas with high mining activity. This approach is consistent with that used in the global air pollution risk assessment study /12/.

The population-weighted annual average concentrations of PM_{10} and $PM_{2.5}$ exposures for each setting were calculated based on the population within a 5-km radius of the monitoring sites. Urban air pollution sources include stack (10-90 m long) emissions and emissions from ambient and domestic sources, and may be considered to have impacts as far as 10-20 km from the source, depending on the nature of the emission.

Selecting a relatively small radius of 5 km was regarded as the optimal compromise between representing localized sources and more distant air pollution sources. The 'small area level' dataset from Census 2001 /22/ was used to determine the population residing in the assumed 5-km impact zone around each monitoring point by the Geographical Information Systems (GIS) Unit of the South African Medical Research Council.

Variations across the impact zone were assumed to average across the area, as well as with time over an annual period. The population-weighted mean PM_{10} and $PM_{2.5}$ concentrations for all urban areas in South Africa in 2000 were also estimated (Table 1). The health effects associated with PM exposure include lung cancer and respiratory disease, as well as certain specific cardiovascular outcomes /72/. The three health outcomes assessed by Cohen et al. /12/ in the global study were included in this study, classified using ICD-9 codes /73/:

1. mortality due to cardiopulmonary disease in adults aged 30 years and older;
2. mortality due to lung cancer in adults aged 30 years and older; and
3. mortality due to acute lower respiratory infections (ALRIs) in infants and children (aged 0-4 years).

Morbidity outcomes that are likely to be causal but were not quantified because of lack of sufficient evidence on prevalence or hazard size, or both, included cardiovascular and respiratory morbidity, including hospitalization for cardio-vascular or respiratory disease; emergency room and urgent care visits; asthma exacerbation; acute and chronic bronchitis; respiratory symptoms; and decreased lung function /12/.

A recent review of South African-based studies of the health effects of air pollution concluded that no study was able to provide local estimates of the risk /74/. In these analyses, we used the RR estimates from Cohen et al.'s /12/ base-case analyses in which annual average concentrations measured from 1979 to 1983 American Cancer Society (ACS) data were used as estimates of exposure /75/. After deriving the concentration-response functions for the three endpoints, Cohen et al. assumed a log-linear risk model, which led to the following formulae for the RR for outcome *i* related to $PM_{2.5}$ and PM_{10} that were used in this study:

$$RR_{2.5i} = \exp[\beta_{2.5i} \times (C_{2.5} - 7.5\ \mu g/m^3)]$$
$$RR_{10i} = \exp[\beta_{10i} \times (C_{10} - 15\ \mu g/m^3)],$$

where $C_{2.5}$ and C_{10} are the South African-specific population-weighted mean concentrations of $PM_{2.5}$ and PM_{10}, respectively, and $\beta_{2.5i}$ and β_{10i} are the slopes of the concentration-response functions for $PM_{2.5}$ and PM_{10}, respectively. In the absence of background air pollution data, we used the counter-

Table 1: *Population-weighted mean PM_{10} and $PM_{2.5}$ concentrations for urban areas, South Africa, 2000*

Metro/Urban area	Population*	Mean PM_{10} concentration	$PM_{2.5}/PM_{10}$ ratio	Estimated $PM_{2.5}$ Concentration	Data source
City of Cape Town	615 022	29.3	.0.58	17.0	SS
Khayelitsha	225 183	56.8	0.55	31.2	SS
Ethekwini (Durban)	3 090 121	40.2	0.58	23.3	EM
Nelson Mandela Metro	93 703	49.2	0.58	28.5	NMM
City of Johannesburg	505 315	46.1	0.57	26.4	CJ
Alexandra	166 971	44.0	0.57	25.2	CJ
Orange farm	192 268	64.6	0.57	37.0	CJ
Soweto	688 427	50.0	0.55	27.5	CJ
Randburg	129 646	46.0	0.57	26.4	F
Rustenburg	94 920	57.0	0.35	20.0	F
Vaal Triangle	90 571	68.9	0.57	39.5	F
Kempton Park	118 654	42.0	0.57	24.1	F
Pop.-weighted mean (urban areas with monitoring data)	5 537 718	46.9	0.57	26.6	-

*Population within 5 km radius of monitoring sites - Census 2001 [22]. CJ = City of Johannesburg; SS = Scientific Services Cape Town; EM – Ethekwini Municipality (Durban); F = Fridge study (9); NMM = Nelson Mandela Metro (Port Elizabeth). (Reproduced with permission from South African Medical Journal [8])

factual or theoretical minimum risk exposure annual average values of 7.5 $\mu g/m^3$ for $PM_{2.5}$ and 15 $\mu g/m^3$ for PM_{10}, as estimated by the WHO global urban outdoor air pollution risk assessment study /12/.

In MS Excel, we calculated PAFs using the classical epidemiologic formula for a two-category exposure (baseline no-risk):

$$PAF = \frac{P(RR-1)}{P(RR-1)+1}$$

where P is the prevalence of exposure (indexed as population weighted mean concentrations of PM_{10} or $PM_{2.5}$, depending on the health outcome, for urban areas in South Africa), and RR is the relative risk of disease in the exposed versus unexposed, as calculated above.

National PAFs for the three endpoints were calculated by weighting the PAF for urban areas in proportion to the total population (33%) residing in the six metropolitan areas (including Sasolburg)

Table 2: *Exposure to indoor air pollution from household use of solid fuels by population group*, South Africa, 2000.*

	Household solid fuel use				
Population group*	African	Coloured	White	Asian/ Indian	South Africa
Fuel type					
Solid fuel use	41%	15%	2%	1%	33%
Biomass	32%	14%	2%	1%	26%
Coal	9%	1%	0%	0%	7%
	Exposure# adjusted by ventilation factor				
Fuel type					
Solid fuel use	24%	9%	1%	1%	20%
Biomass	19%	8%	1%	0%	16%
Coal	5%	1%	0%	0%	4%

Source: Census 2001 [22]. Reproduced with permission from South African Medical Journal [7]

*Population group of household head

#Exposure to solid fuels = % households using solid fuels for cooking or heating after taking into account the ventilation in the households (ventilation coefficient 0.6).

using Census 2001 data /22/, and assuming that the non-metropolitan areas were not at risk.

Indoor Air Pollution - Household Use of Solid Fuels

Household solid fuel use was estimated at the population group level using binary classifications of exposure to household fuel use (exposed to solid fuels if using wood, coal, or dung; or not exposed if using electricity, gas, or paraffin for cooking or heating) based on Census 2001 data /22/. Owing to marked differences in fuel use in the four different population groups, the analysis was carried out separately for each. To account for differences in other factors such as type of housing which may affect levels of indoor air pollution, we adjusted the exposure variable by a ventilation factor:

Household-equivalent solid fuel exposed population = (population using solid fuel) × (ventilation factor)

The ventilation factor or coefficient reflects the share of people being exposed after taking into account the ventilation in the household. Solid fuel use outdoors results in complete ventilation and a ventilation coefficient of 0, whereas a poorly ventilated household would have a coefficient of 1. Based on expert opinion and taking into account that due to the mild climate, heating is necessary only for about 3 months of the year, we used an estimate of 0.6 (range 0.4-0.8 to allow for seasonal variation) as the ventilation factor. The theoretical minimum for this risk factor is no use of solid fuels for the production of household energy, and this has been achieved in many populations.

The estimated exposure to indoor air pollution from household use of solid fuels is presented in Table 2 by population group. Separate estimates of exposure resulting from use of coal are also presented. A third of South African households used solid fuels for cooking or heating, with marked population group differences ranging from 41% of black African households to only 1% to 2% of

Indian and white households. After taking into account ventilation, exposure to solid fuels was estimated at 24% in the black African, followed by 9% in the colored population and about 1% in both the Indian and white population groups (Table 2).

Smith and colleagues /76/ carried out a comprehensive review of the epidemiologic evidence available for each disease endpoint to select the health outcomes caused by exposure to indoor smoke from the use of solid fuels. Three health outcomes had strong evidence of a causal relationship: ALRIs in children under 5 years of age, and chronic obstructive pulmonary disease (COPD) and lung cancer (from the use of coal) in adults 30 years and older. Available data indicate that men are at lower risk than women because of lower exposures. The outcomes potentially associated with solid fuels use but not quantified because of a lack of sufficient evidence on causality included CVD, cataracts, tuberculosis, asthma, perinatal effects, including low birth weight, and lung cancer from biomass. Estimates of RRs and confidence intervals (CI) from meta-analyses of studies that controlled for the confounding effects of chronic respiratory disease and smoking /76/ are used in this study. PAFs were calculated using the classical epidemiologic formula outlined under urban air pollution.

Smoking is an important risk factor for the diseases associated with indoor smoke from solid fuels, specifically lung cancer and COPD. Yet, information on the joint effects of smoking and solid fuel use is scarce. In previous analyses, to avoid possible overestimation of the burden of disease attributable to indoor smoke, PAFs for lung cancer and COPD caused by exposure to indoor smoke were applied to the disease burden remaining after removing the burden attributable to tobacco (with an adjustment for occupational exposure) /77/. This approach was conservative as the attributable risks do not add up to 100%, and some of the effect attributable to tobacco may also be attributable to indoor smoke from household use of solid fuel, although the risks of tobacco in any individual are far greater than those from air pollution. For this paper, PAFs for lung cancer and COPD were applied to burden of disease estimates for lung cancer and COPD, and we quantified the joint effect of smoking, urban air pollution, and solid fuel use on lung cancer and COPD (see below).

Unsafe Water, Sanitation, and Hygiene

The composite risk factor, unsafe WSH, was defined as including 'multiple factors, namely the ingestion of unsafe water, lack of water linked to inadequate hygiene, poor personal and domestic hygiene and agricultural practices, contact with unsafe water, and inadequate development and management of water resources or water systems /78/.

We used two approaches for estimating the disease burden attributable to unsafe WSH, according to disease outcome /6/. For selected related diseases other than infectious diarrhea (combined in the category 'intestinal parasites') and for schistosomiasis, the burden was assumed to be 100% attributable to exposure to unsafe WSH /78/. Although largely attributable to unsafe WSH, trachoma is not common in South Africa and is therefore not listed separately in the South African burden of disease list and could not be included in this analysis. A number of other diseases related to unsafe WSH, such as hepatitis A and scabies, could not be included as their risk factor-disease relationships remain unclear /79/.

For the diarrheal disease burden from unsafe WSH, estimates were based on calculating a population-attributable fraction derived from exposure information. A scenario-based approach was applied /78/. Here the risk of diarrheal disease is conditioned by a typical exposure or representative combination of risk factors at commonly encountered levels. Scenarios were defined on the basis of, firstly, type of water and sanitation infrastructure, and secondly, load of

fecal-oral pathogens in the environment. The resulting six exposure scenarios capture combinations of the risk factors related to unsafe WSH as follows:

- *Scenario I: ideal situation or theoretical minimum, conferring lowest possible population risk for transmission, corresponding to the absence of transmission of diarrheal disease through WSH. Environmental fecal-oral pathogen load is very low in this scenario. The prevalence for this scenario is assumed to be zero in all WHO regions because even in the most developed regions cases of food poisoning, etc. occur.*
- *Scenario II: typically encountered in developed or high income countries. This scenario has a low-to-medium load of fecal-oral pathogens in the environment, characterized by more than 98% coverage in improved water supply and sanitation, and a regional incidence of diarrhea of less than 0.3/person/year /80,81/.*
- *Scenario III: various improved forms of provision that reduce the risk of exposure compared with scenario IV; a transitionary scenario between high (scenario IV) and low (scenario II) environmental pathogen loads /82/.*
- *Scenarios IV-VI: high fecal-oral pathogen environments that are typical in low and middle income countries and characterized by poor access to water and sanitation.*

The risk estimates assigned to each exposure scenario were based on those of Prüss-Üstün et al. /78/, who used large surveys and reviews of multi-country studies to derive risk averages—the average risk related to the described scenarios across the world and in an array of situations /78/. The ideal situation (scenario I) was assigned a relative risk (RR) of 1.0. Because of the uncertainties associated with these risk estimates, upper and lower uncertainty boundaries were constructed, drawing on those used for the global study /78/. The lower estimate was based on the diarrheal disease risk reduction achieved through personal hygiene interventions only; the best estimate on the reduction from improvements in both water quality and personal hygiene; and the upper estimate on the additional improvements from provision of a continuous piped water supply. No differences in RRs across age groups or between genders were assumed.

Data on prevalence and population distribution of exposure were obtained from the South African Census 2001 /21/, which reported the main source of water supply and toilet facilities available to households. Based on the data, households were allocated to 1 of 3 categories: poor, intermediate, or good access to water supply and sanitation facilities (Table 3), which were matched as closely as possible to the exposure scenarios. The situation for certain population subgroups in South Africa was sufficiently different from that of other African countries such that it could not be captured entirely by scenario IV. Urban households with full sanitation coverage and good access to improved water supplies were considered at low risk of diarrheal disease. Using Census 2001 data we assigned households in urban areas and with access to improved water supply and full sanitation coverage (27.8% of all) to scenario II. The 22.9% of households with piped water and full sanitation coverage but in rural or urban informal settlements were assigned to scenario IV, together with households with intermediate water and sanitation facilities. The remaining 20.7% were placed in scenarios Va, Vb and VI (Table 3).

Population-attributable fractions were calculated in Excel using the formula for risk factors with multiple categories /83/.

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

Table 3: *Households with different standards of water and sanitation access, South Africa 2000*

		Standard of sanitation facilities			
		Poor	Intermediate	Good	Total
Standard of water supply	No. of households	1 926 641	4 448 707	5 136 206	11 511 555
Poor	923 360	4.0%	4.0%	0.02%	8.0%
Intermediate	7 329 232	12.4%	34.4%	16.8%	63.7%
Good	3 258 963	0.32%	0.17%	27.8%	28.3%
Total	11 511 555	16.7%	38.7%	44.6%	100.0%

Exposure scenario							
	I	II	III	IV	Va	Vb	VI
% of households assigned to each scenario in South Africa	0	27.8	0	51.4	4.0	12.7	4.0

Poor = water from a dam, pool, or stagnant water source from a river, stream or rainwater tank, no sanitation or a bucket system;
Intermediate = water from a spring or borehole or piped water collected from up to 200 m away (outside dwelling or yard) or from a water vendor and basic sanitation (pit latrine with or without ventilation);
Good = piped water into the residence and flush toilet and living in urban areas.

Note: Households with piped water into the residence or yard and a flush toilet in rural areas or informal urban settlements were assigned to the intermediate category.
Source: Census 2001 [22]. Reproduced with permission from South African Medical Journal /6/.]

where p_i is the prevalence of exposure in level $_i$, RR_i is the RR of disease in exposure level $_i$ and $_k$ is the total number of exposure levels.

Lead Exposure

Lead exposure was characterized by the population distribution of blood lead concentrations. No nationally representative data on blood lead levels were available and exposure was assessed separately for urban and rural areas. Exposure data for primary school children (aged 5 to 12 years) were pooled from studies carried out at three urban sites in Cape Town, Johannesburg, and Kimberley /66,84/. For the rural sample /84/, occupational exposures or 'hotspots' (in Aggeneys, a lead mining town where lead levels were unusually high) were excluded. Due to data limitations, the same blood lead concentrations were used for children under 5 years and for children 5-14 years of age (Table 4), 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 /85/. Blood lead levels in rural adults were assumed to be the same as in rural children (Table 4).

Table 4: *Estimates of age-specific mean blood lead levels and standard deviations (µg/dl), South Africa 2000*

Population	Parameter	Age groups (yrs)			Data sources		
		0 to 4	5 to 14*	15+	Study site	Year	N
Urban	Mean	7.5	7.5	7.4	Children (5-12 years) in Cape Town [66, 84]	2002	429
					Johannesburg	2002	382
	SD	3.31	3.31	3.85	Kimberley	2003	355
					Pregnant women in Durban [85] for adults 15+	1996	296
Rural	Mean	5.5	5.5	5.5	Children (5-12 years) [66, 84]	2003	98
	SD	2.33	2.33	2.33			

SD=standard deviation * Children 5-14 years not included in the analysis (Reproduced with permission from South African Medical Journal [9])

The proportion of the population at specified blood lead concentrations in the urban and rural populations was estimated for adults and children separately based on the lognormal distribution /9/, weighted according to the urban/rural breakdown based on Census 2001 /22/ data to provide a national estimate of exposure.

The outcomes assessed in this study were similar to those in the global CRA study /61/ and included cardiovascular diseases (CVDs) from elevated systolic blood pressure (in adults 30 years and older), and IQ reduction in children under 5 years of age. A loss of IQ points was considered to lead to disease burden when resulting in mild intellectual impairment or mild mental disability defined as an IQ score of 50-69 points /61/.

Anemia and gastrointestinal effects, nephropathy, and encephalopathy were not quantified in this analysis because they occur only in extreme cases. Other outcomes that are likely to be causal, but were not quantified because of lack of sufficient evidence on causality included developmental and reproductive system effects and social consequences of IQ loss /61/. In the absence of a scientific consensus of the lowest level of population risk, we used a blood lead concentration threshold of 5 µg/dL in this analysis for the related health risks.

Following the framework of Prüss-Üstün et al. /61/ in the global assessment, the incidence of mild mental disability resulting from IQ reduction attributable to lead exposure was estimated and used to calculate lead-induced mild mental disability DALYs in children under 5 years of age, as described in detail elsewhere /9/.

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 Schwartz /86/ and a published analysis of data from the second National Health and Nutrition Examination Survey (NHANES II) /61/. As detailed in Norman et al. /9/, the CVD PAFs were calculated using the formula for risk factors with multiple categories /83/ outlined under unsafe WSH.

The risk values for CVD for the defined increases in systolic blood pressure associated with increased blood lead concentrations were based on the risk ratios obtained from a collaborative meta-analysis of individual participant data from 61 separate prospective studies /87/ used in the high blood pressure assessment /88/.

Joint Effect Estimation

For each health outcome, the PAFs for each risk factor were combined in a multiplicative calculation of the total PAF of the disease in question attributed to all risk factors using the formula:

$$\text{Joint PAF} = 1 - \prod_{i=1}^{n}(1 - PAF_i)$$

where PAF_i is the PAF of individual risk factors, i is the individual risk factor, and n is the total number of risk factors /89/. We used this formula to estimate the burden attributable to the joint effect of the four environmental risk factors. We also quantified the joint effect of urban air pollution, indoor air pollution, and tobacco use on COPD and lung cancer in adults.

Uncertainty Analysis

Monte Carlo simulation-modeling techniques were used to present uncertainty ranges around point estimates reflecting the main sources of sampling uncertainty in the calculations. Ersatz software version 1.0 /90/ was used as an add-in to Excel, allowing multiple recalculations of the Excel spreadsheet, each time choosing a randomly drawn value from the distributions defined for input variables.

In the unsafe WSH analysis, we specified a triangular probability distribution around the RR estimates with 3 points: the published best RR estimate as the most likely point and the published upper and lower uncertainty boundaries /82/ as the maximum and minimum entered values of the distribution.

For urban air pollution, indoor air pollution, and lead exposure for the RR input variables, we made the standard assumption that the natural logarithm of the RR has a normal distribution and used standard errors derived from the published 95% CIs. We used the Ersatz random function ErRelativeRisk with a correction that takes the RR and SE[ln(RR)] as parameters, and recalculates them to produce a mean effect size equal to the point estimate of the RR in the uncertainty analysis /91/.

In the indoor air pollution analysis, we specified for the ventilation coefficient a uniform probability distribution across the range 0.4 to 0.8. For urban air pollution, we assumed that the observed mean PM_{10} concentrations in each area could vary by 20%, and we specified a triangular distribution with three points (minimum, most likely (the observed concentration) and maximum).

For estimating $PM_{2.5}$ from PM_{10} using the ratio method, we again specified a triangular probability distribution with the upper and lower estimates published by air pollution studies /12,13/, depending on whether the area was metropolitan (0.5-0.65) or a dusty urban mining area (0.2-0.5), as the maximum and minimum entered values of the distribution.

For lead exposure, normal distributions of the mean blood lead concentration were specified by age and gender. 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 (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 µg/dL increase in blood lead concentration as detailed in Norman et al. 2007 /9/. For each output variable (namely attributable and joint 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 2000 iteration values generated.

RESULTS

For each environmental risk, the PAFs, the attributable deaths, and the DALYs by gender are presented in Table 5.

Table 5: *Burden of disease attributable to environmental risk factors in South Africa in 2000*

URBAN OUTDOOR AIR POLLUTION									
	MALE			FEMALE			PERSONS		
Outcome	**PAFs**	**Deaths**	**DALYs***	**PAFs**	**Deaths**	**DALYs***	**PAFs**	**Deaths**	**DALYs***
Lung cancer (adults 30+)	**5.1%**	**237**	**2 449**	**5.1%**	**113**	**1 155**	**5.1%**	**350**	**3 604**
Cardiopulmonary disease (adults 30+)	**3.7%**	**1 936**	**18 031**	**3.7%**	**2 286**	**18 391**	**3.7%**	**4 222**	**36 423**
Hypertensive disease		189	1 754		419	3 239		608	4 993
Ischemic heart disease		651	5 694		613	4 088		1 264	9 782
Stroke		483	4 479		742	6 144		1 225	10 623
Inflammatory heart disease		85	944		92	899		177	1 843
Other CVD		59	665		80	846		139	1 510
COPD		287	2 385		179	1 415		466	3 799
Asthma		119	1 344		118	1 300		237	2 644
Other respiratory diseases		64	765		42	462		106	1 227
A RI (0-4 yrs)	**1.1%**	**34**	**1 130**	**1.1%**	**32**	**1 062**	**1.1%**	**65**	**2 193**
Lower respiratory		33	1 103		31	1 041		64	2 144
Upper respiratory		1	25		1	20		1	45
Otitis media		0	2		0	1		0	4
Total		**2 207**	**21 610**		**2 430**	**20 609**		**4 637**	**42 219**
95% UI		757-3 687	8 191-35 718		761-4 138	7 164-34 742		1 480-7 838	15 395-70 591
% of total burden		**0.8%**	**0.4%**		**1.0%**	**0.4%**		**0.9%**	**0.4%**
95% UI		0.3-1.3%	0.1-0.6%		0.3-1.7%	0.1-0.7%		0.3-1.5%	0.1-0.7%
UNSAFE WATER AND LACK OF SANITATION AND HYGIENE									
	MALE			FEMALE			PERSONS		
Outcome	**PAF**	**Deaths**	**DALYs**	**PAF**	**Deaths**	**DALYs**	**PAF**	**Deaths**	**DALYs**
Diarrheal diseases	84%	6 692	199 565	84%	6 676	186 595	84%	13 368	386 160
Schistosomiasis	100%	11	15 329	100%	9	6 733	100%	20	22 062
Intestinal parasites**	100%	26	6 413	100%	20	4 155	100%	46	10 568
Total		6 729	221 307		6 705	197 483		13 434	418 790
95% UI		6 373-6 966	210 691-228 391		6 350-6 942	187 557-204 107		12 723-13 909	398 248-432 498
% of total burden		2.5%	2.6%		2.7%	2.6%		2.6%	2.6%
95% UI		2.3-2.5%	2.5-2.7%		2.6-2.8%	2.4-2.6%		2.4-2.7%	2.5-2.7%

Table 5: *Burden of disease attributable to environmental risk factors in South Africa in 2000 (continued)*

LEAD EXPOSURE									
	MALE			FEMALE			PERSONS		
Outcome	**PAFs**	**Deaths**	**DALYs**	**PAFs**	**Deaths**	**DALYs**	**PAFs**	**Deaths**	**DALYs**
Ischemic heart disease	1.7%	273	2 853	1.1%	149	1 257	1.5%	422	4 110
Stroke	2.5%	273	3 326	1.5%	248	2 819	1.9%	521	6 145
Hypertensive disease	4.3%	203	2 086	2.7%	277	2 378	3.2%	479	4 464
Other CVD	2.1%	3	129	1.3%	2	105	1.7%	6	234
Mild mental disability	100%	0	21 608	100%	0	22 378	100%	0	43 986
Total		752	30 002		676	28 937		1 428	58 939
95% UI		478-1 034	26 948-33 150		411-942	26 373-31 539		1 014-1 835	54 545-63 197
% total burden		0.27%	0.35%		0.27%	0.37%		0.27%	0.36%
95% UI		0.17-0.38%	0.32-0.39%		0.17-0.38%	0.34-0.41%		0.19-0.35%	0.34-0.39%
INDOOR AIR POLLUTION FROM HOUSE HOLD USE OF SOLID FUELS									
	MALE			FEMALE			PERSONS		
Outcome	**PAF**	**Deaths**	**DALYs**	**PAF**	**Deaths**	**DALYs**	**PAF**	**Deaths**	**DALYs**
ALRI	23.6%	732	25 052	23.8%	696	23 527	23.7%	1 428	48 579
COPD	12.3%	865	10 418	23.4%	1 024	10 391	16.1%	1 889	20 809
Lung Cancer (coal)	1.6%	68	811	1.9%	35	435	1.7%	103	1 245
Total		1 665	36 280		1 755	34 352		3 420	70 633
95% UI		613-2 817	20 063-53 805		1 147-2 314	22 940-45 056		2 012-4 912	45 187-96 271
% total burden		0.6%	0.4%		0.7%	0.4%		0.7%	0.4%
95% UI		0.2-1.0%	0.2-0.6%		0.5-0.9%	0.3-0.6%		0.4-0.9%	0.3-0.6%

*includes only YLLs; COPD= Chronic obstructive pulmonary disease; **including ascariasis, trichuriasis, hookworm
ALRI - Acute lower respiratory infections; CVD – cardiovascular diseases; UI - uncertainty interval;

Unsafe water, sanitation, and hygiene accounted for 2.6% of total DALYs in South Africa in 2000, whereas indoor smoke from solid fuels and lead exposure accounted for 0.4% each and urban air-pollution accounted for 0.3% of all DALYs in 2000.

The joint effect of unsafe WSH, indoor air pollution, urban air pollution, and lead exposure was responsible for 4.7% (95% uncertainty interval 3.8% to 5.3%) of all deaths in South Africa 2000 (Figure 2). The total burden of disease attributable to the joint effect of these four environmental risk factors in 2000 was 600,660 DALYs equivalent to 3.7% (95% uncertainty interval 3.4-4.0%) of the total disease burden for South Africa (**Figure 3**). The largest contributor to joint attributable burden is unsafe WSH, with 64.3% of the joint burden caused by diarrheal diseases. Respiratory infections and

CVDs each account for similar proportions (8%) of the joint attributable burden, and lead-induced mild mental disability in children under 5 years of age accounts for about 7% of the joint burden (Figure 4). The largest proportion of the disease burden attributable to these risks is experienced by children under 5 years of age, with 9.7% (95% uncertainty interval 9.1% to 10.2%) of the disease burden from all causes in this age group attributable to the joint effect of all four environmental risks. The joint effect of indoor and outdoor air pollution is responsible for a quarter of all ALRI burden in children under 5 years of age.

Lead caused 44,000 DALYs due to mild mental disability in this age group, and 84% of the diarrheal disease burden could be attributable to unsafe WSH in children under 5 years of age. This finding ranks the joint effects of the environmental risk factors as the third largest risk factor for disease among under-5 year olds, behind vertical transmission of HIV due to unsafe sex and undernutrition. These four environmental risk factors are therefore considerably more important in this age group than in the South African population as a whole.

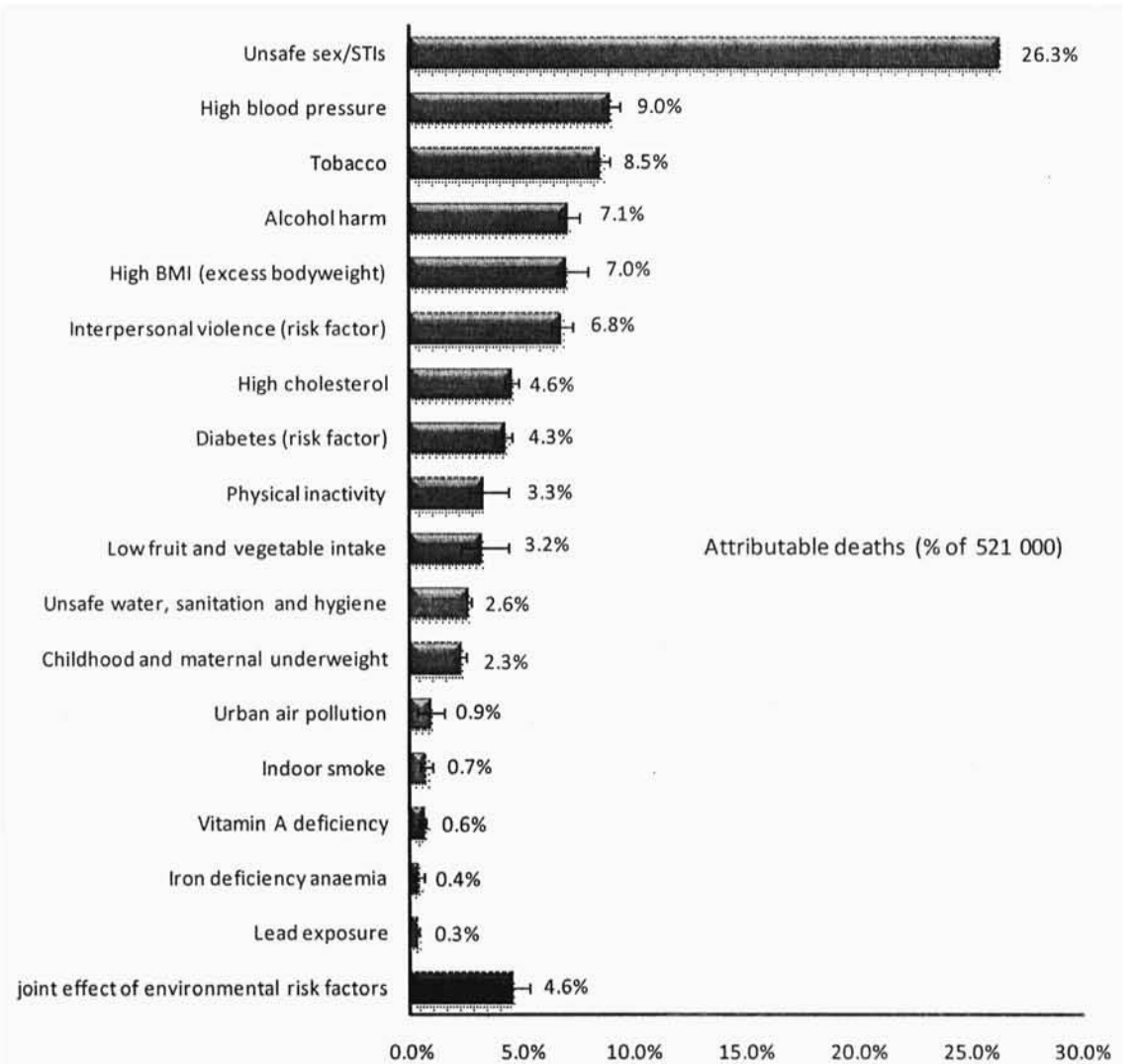

Fig. 2: Proportion of total deaths attributable to the joint effect of 4 environmental risk factors, South Africa, 2000

Almost 20% of all CVD deaths could be attributed to the joint effect of lead exposure, urban air pollution and tobacco. In this analysis, 78.1% of all lung cancer DALYs in males and 65.9% in females could be attributed to the joint effect of tobacco, indoor air pollution and urban outdoor air pollution. Similarly for COPD 73.5% of the burden in males and 61.3% of the burden in females could be attributed to the joint effect of tobacco, indoor air pollution and urban outdoor air pollution.

DISCUSSION

An analysis conducted on the joint effects of four major environmental risk factors has revealed that the total burden of disease attributable to the joint effect of these four risk factors in 2000 accounted for 3.7% (95% uncertainty interval 3.4%-4.0%) of the total disease burden for South Africa, similar to the contribution from tobacco smoking which ranked fourth of 17 risk factors analyzed (4% of all DALYs). Environmental risk factors are a major preventable cause of death among children under 5 years of age, accounting for 10.8% of deaths from preventable disease in this age group as well as considerable loss of healthy life (9.7% of all DALYs). The substantial burden of disease caused by these exposures highlights the importance of International Comparisons.

Urban air pollution. In urban areas of South Africa, the average annual exposures to ambient PM_{10} (46.9 $\mu g/m^3$) and $PM_{2.5}$ (26.6 $\mu g/m^3$) are at levels well above those considered to be without increased risk of mortality (15 $\mu g/m^3$ and 7.5 $\mu g/m^3$, respectively). The highest annual concentrations of PM_{10} and $PM_{2.5}$ were estimated for the Vaal Triangle, an area characterized by a concentration of industries, followed by Orange Farm, an impoverished informal settlement. Concentrations were also high in the peri-urban

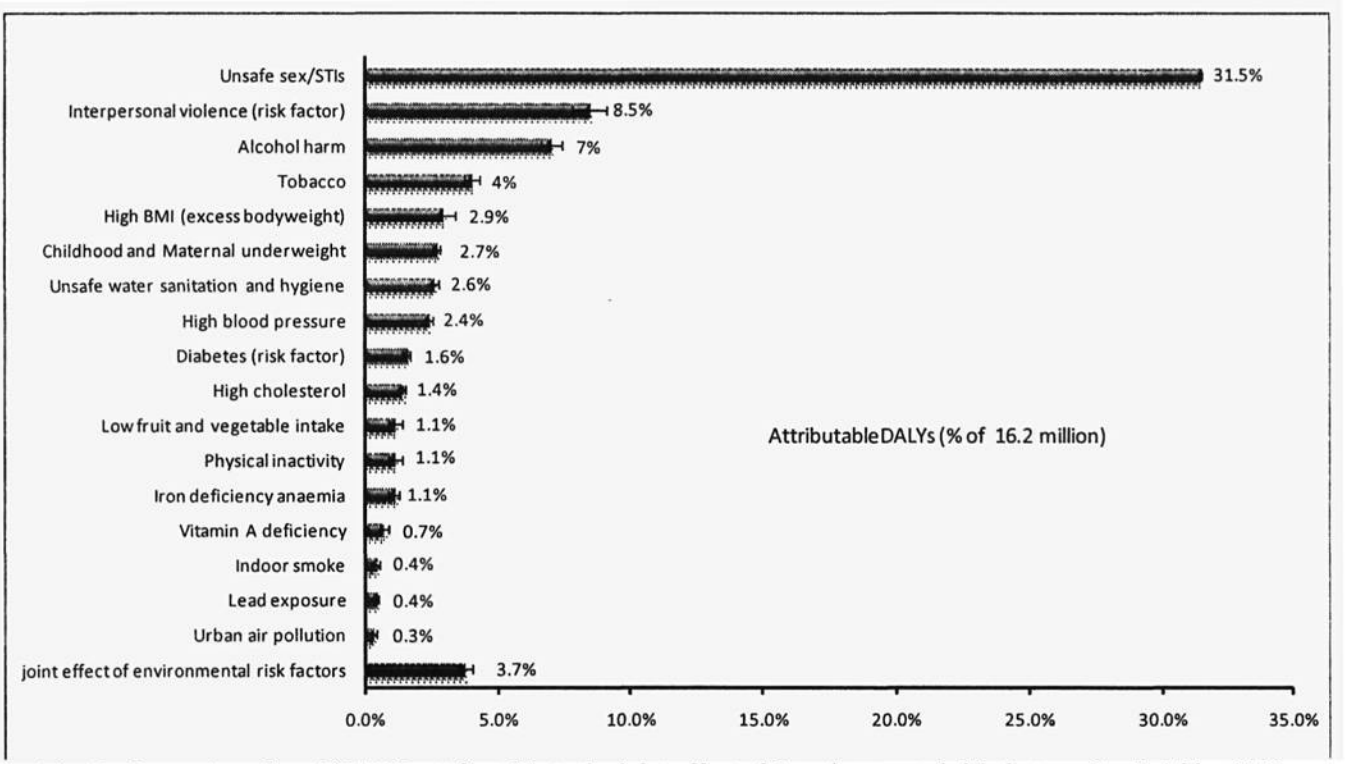

Fig. 3: Proportion of total DALYs attributable to the joint effect of 4 environmental risk factors, South Africa 2000

areas of Khayelitsha and Soweto. Exposure to urban air pollution caused 0.9% of all deaths in 2000 with a fairly wide uncertainty range (95% uncertainty interval 0.3%-1.5%).

The results of the global risk assessment study /12/ revealed considerable variation in the estimates across the 14 subregions of the world, with the greatest burden occurring in the more polluted and rapidly growing cities of low and middle income countries. The global study estimated PAFs that were comparable to those in the South African study, with air pollution in urban areas worldwide estimated to cause about 3% of mortality attributable to cardiopulmonary disease in adults, about 5% of mortality attributable to cancers of the trachea, bronchus and lung, and about 1% of mortality attributable to ARIs in children under the age of 5 years.

Indoor air pollution. Indoor air pollution from the household use of solid fuels appears to be of less serious public health importance in South Africa than in the rest of sub-Saharan Africa, partly due to the lower exposure and better ventilation assumed in this study. In the global assessment, estimates for the African region were based on extrapolations from fuel use surveys, and all African countries were assigned a ventilation coefficient of 1. In 2002, however, the WHO country-specific estimates for South Africa estimated the percentage of the population using solid fuels at 18%, much lower than for other

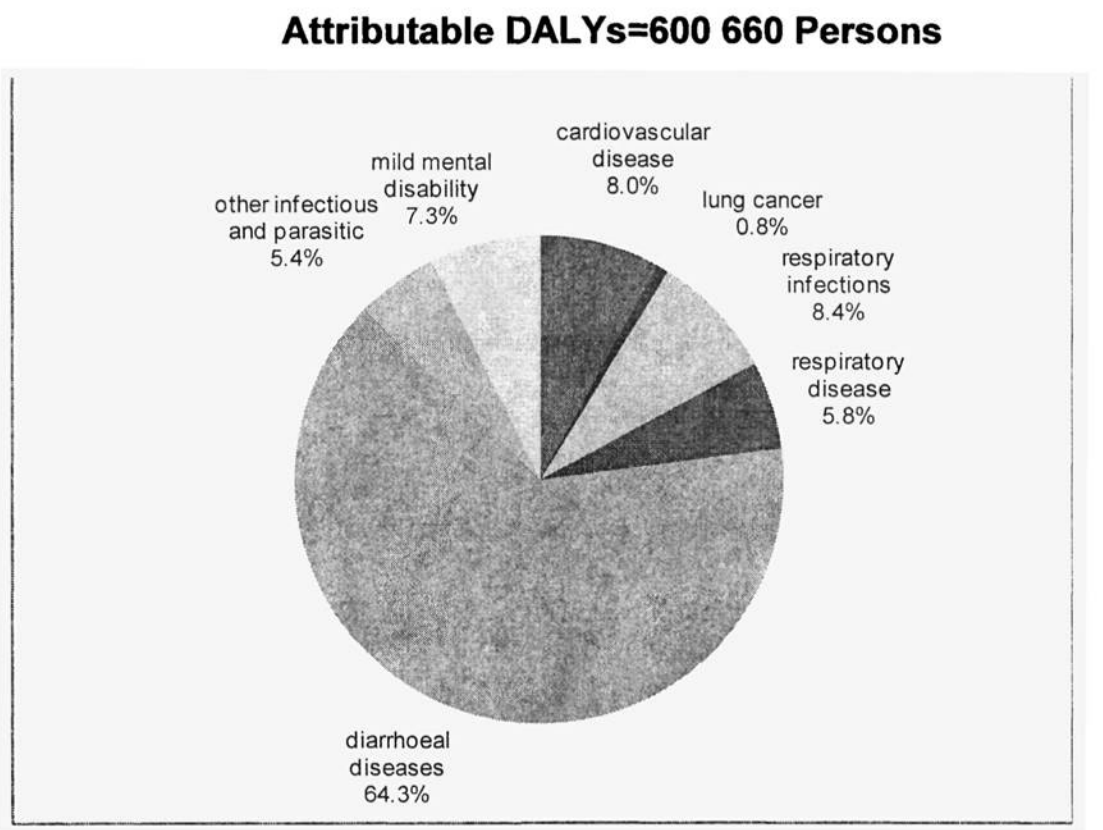

Fig. 4: Disease burden attributable to the joint effect of 4 environmental risks, South Africa 2000

African countries, and only 0.1% of DALYs were attributable to indoor air pollution from solid fuel use /92/. This amount is less than the local estimate where indoor air pollution from household use of solid fuels caused 0.4% of all DALYs (95% uncertainty interval 0.3% to 0.6%) in South Africa in 2000. Such differences are probably the result of the higher exposure estimates used in our local assessment and of the analysis being carried out by population group. Based on the Census 2001 data and taking ventilation into account, exposure to solid fuels was estimated to be as high as 24% in the African population and 20% overall (Table 2).

Unsafe WSH. Estimates are comparable with those reported in the global unsafe WSH risk factor assessment, although in South Africa, the proportion of all attributable deaths and DALYs in the age group 0 to 4 years is less than the global estimates for this age group /78/. This difference is probably the result of the approach used to allocate the South African population to different exposure scenarios for determining risk of diarrheal diseases from unsafe WSH. Based on data from the 2001 Census, of the South African population, 27.8% and 51.4% were respectively allocated to scenarios II and IV, which is very different from the distribution for the WHO African (AFR-E) region (countries in sub-Saharan Africa), where 0% was allocated to scenario II, 42% to scenario IV, and 9% and 38% to scenarios Vb and VI, respectively /78/. Nevertheless, we believe that when compared with other low and middle income countries in the African region, the allocation described here reasonably reflects access to improved water and sanitation facilities for certain urban populations in South Africa.

Lead exposure. Although lead-reduction programs have been initiated in South Africa, these measures 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 approximately half (53%) of the national population (about 23 million) had blood lead concentrations between 5 and 10 μg/dL. Blood lead concentrations above 10 μg/dL were estimated for about 10% of the population (almost 4 million South Africans). Rural populations in South Africa were also exposed to lead, although to a somewhat lower degree.

In both children and adults, mean blood lead concentrations in the South African population were slightly lower than the estimates for the WHO African region (AFR-E) for two main reasons. Firstly, more recent and locally representative data were used in this analysis. Secondly, 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 concentrations, as described in more detail elsewhere /9/.

Nevertheless, the estimated prevalence of mild mental disability attributable to lead (0.5%) was comparable to that estimated for the African region /61/. Indeed, 40% of mild mental disability is estimated to be of genetic origin, 20% is caused by environmental factors, and 40% is of unknown etiology /93/; in low- and middle-income countries, the contribution of lead to the total incidence of mild mental disability is thought to be as high as 15%-20% /61/. The high prevalence of mild mental disability in low and middle income countries highlights the role of avoidable and preventable exposure to harmful environmental factors like lead.

Study Limitations

In this study, attempts have been made to quantify sampling uncertainty. Clearly, however, some uncertainty around these estimates remains beyond the sampling uncertainty that could not be quantified. Extrapolating the overall risks from other countries to South Africa, in the absence of reliable local estimates of the risk of exposure to

such environmental factors, is an important source of uncertainty. Also some uncertainty around the cause of death and burden of disease estimates can be detected /1,2/. In general, however, the study could be improved through more representative data on exposures, disease burden, and the epidemiologic relationship between the risk factor and health. Specific study limitations have been identified for each risk factor and are outlined below.

Urban outdoor air pollution. Estimates of mortality attributable to urban outdoor air pollution based solely on the effect of annual average exposure to $PM_{2.5}$ and PM_{10} are probably an underestimate of the actual burden. If it were possible to identify, accurately measure, and include exposure to all known pollutants, taking into account interrelated effects, the burden attributable to this risk factor would be higher /94, 95/. Our estimate is conservative as we assume exposure occurred only in the metropolitan areas (and Sasolburg) and not in the more scattered urban areas or rural areas. Nevertheless, our initial estimates indicate that the health impact is significant.

Notably, most studies concerned with the health effects of air pollution have been conducted in cities in North America and Europe, with comparatively few elsewhere. Therefore, compared with mortality findings, the uncertainty rises to a greater degree when morbidity findings are extrapolated to low- and middle-income countries because the estimation requires both a concentration-response function and a baseline incidence rate /72/. Because of the lack of data on the risk of increased morbidity or levels of exposure in rural settings, our study is likely to understate the extent of the burden. Future estimates of burden of disease attributable to urban air pollution should include such morbidity outcomes as asthma exacerbation. This aspect, however, will depend on further research into the health effects of air pollution in low- and middle-income countries, including South Africa. Such research should also aim to provide better estimates not only of ambient concentrations but also of the characteristics of urban air pollution, including the size distribution and chemical composition of PM, and the contribution of various sources to PM and other air pollutant concentrations. A more comprehensive estimate of the burden attributable to air pollution should include estimates for annual (and seasonal) average concentrations of the other common pollutants: SO_2, NO_2, O_3, CO, and volatile organic compounds, such as benzene, a well-known hematopoietic carcinogen. In addition, limiting the analyses to urban areas may underestimate the burden attributable to this risk factor as outdoor air pollution occurs in rural areas that would not be captured by the indoor pollution estimates.

Indoor air pollution from household use of solid fuels. Multiple fuel use and a degree of 'fuel switching' in poor households mean that households may use up to five fuels for cooking and heating. Hence, even if households reported 'clean fuel' as their main energy source for cooking, they may often have complemented this with other fuels, based largely on affordability. One study /96/ found that after being paid, people used paraffin for cooking and as the month progressed and funds diminished, they slid down the energy ladder to relying on wood (cheaper) and then cow dung (free) as the fuel source.

Considering exposure as a binary classification would also result in an underestimation of the burden. In reality, exposure to indoor air pollution from the use of solid fuels results in a wide range of exposures, which vary according to fuel type and quality as well as stove and housing characteristics (ventilation and size), cooking and heating methods, time spent within the household, close proximity to the pollution source, and the season. In future assessments, exposure would therefore best be characterized as a continuous outcome, or at least better characterized by multiple categories.

In this analysis, for COPD, 73.5% of the burden in males and 61.3% of the burden in females could be attributed to the joint effect of tobacco, indoor air pollution, and urban outdoor air pollution. Notably, the risks of tobacco in any individual are far greater than those from air pollution. Exposure to air pollution and tobacco smoking can also act synergistically in promoting lung cancer and COPD.

Indoor air pollution may also work jointly or synergistically with undernutrition and/or HIV to increase the incidence and effects of diseases such as ALRI. Some risks related to indoor smoke may be mediated through under-nutrition whereas, equally, some risks for undernutrition may be mediated through indoor smoke-related ALRI. HIV-positive children living in conditions of high exposure to indoor air pollution may be particularly vulnerable to consequent respiratory ill health effects. The extent to which this may occur is difficult to measure, however, and has not been assessed.

Growing evidence suggests that other important health outcomes, such as tuberculosis (of special concern because it is also closely related to the HIV/AIDS epidemic), ischemic heart disease, and asthma, which are among the leading causes of death in the country, may also be associated with exposure to indoor smoke from solid fuels. These outcomes were not included in this analysis, however, as the evidence was considered insufficient at this stage /76/, which could also result in an underestimate of the true burden attributable to this risk factor. The association between these priority diseases and indoor smoke needs further investigation in our local setting.

We also assumed that children of 6-14 years and adults of 15-29 years of age were not exposed to this risk factor because of the absence of related health outcomes in these age groups, although probably some exposure did occur in these groups. Although the related chronic diseases would not yet manifest in these age groups, note that the development of these diseases at older ages is a consequence of exposure in the younger age groups. As the levels are unknown, they could not be quantified in these age groups, possibly also leading to an underestimate.

Because of a lack of local epidemiologic data, the results of the meta-analysis by Smith and colleagues /76/ were used as the source of the relative risk estimates. This method is not ideal as extrapolating results of epidemiologic studies from one region to another does not take into account the potentially interactive risk factors, such as malnutrition or HIV, which were not addressed in all of the meta-analyses /76/ and would result in an unquantified uncertainty in our results. Collecting more epidemiologic data on the risks of indoor air pollution in the current South African setting would be important.

Unsafe water, sanitation and hygiene. The unsafe WSH analysis has a number of limitations. Firstly, while providing information on water and sanitation facilities, the Census does not indicate whether these facilities are operating. The termination of household water supplies for non-payment is also pertinent /97/. Although legislation establishing the right to a basic minimum free household water supply may have partly mitigated this problem, the residents of rented 'backyard' shacks may not have access to this water as they are often not registered with the local authority. Such uncertainty regarding the functioning of basic facilities is difficult to quantify and would result in an overestimate of the number of households meeting the criteria for exposure scenario II and an underestimation of the fraction of the burden of diarrheal disease attributable to unsafe WSH. The true attributable fraction for diarrheal diseases might therefore be higher than that calculated.

Secondly, assignment of households to exposure scenarios was not always straightforward. For example, do households using pit latrines without ventilation have 'poor' or 'intermediate' sanitation?

Thirdly, looking at the synergistic effects of risk factors, such as unsafe WSH and malnutrition including micronutrient deficiencies and underweight, and childhood mortality in poor households in this setting, is not within the scope of this framework. The burden of diarrheal diseases reported as attributable to these risk factors could therefore be an underestimate of the true attributable fraction. A recent evaluation suggests that approximately 50% of the disease burden due to malnutrition can be attributed to unsafe WSH /98/, indicating that a proportion of the burden attributable to underweight is linked to unsafe WSH and highlighting the complexity of these effects.

Fourthly, within this assessment, examining how the burden is distributed between rural and urban settings and between poorer and wealthier households is not possible. Nevertheless, the RRs associated with different exposure scenarios clearly indicate that households with poorer access to water and sanitation facilities are at substantially greater risk of developing diarrheal and other diseases. Since most of these households are likely to be located in poor rural or peri-urban settlements, we can assume that the burden attributable to unsafe WSH is borne largely by poorer households and, more specifically, by children within them. Research on water access in relation to the cholera epidemic of 2000-1 supports this assumption /99/.

Finally, we did not assess the attributable burden from all intestinal parasite infestations but rather focused on the major ones. We were also unable to include in our estimates other WSH-related diseases, such as hepatitis A, for which current knowledge does not allow the attributable fraction to be estimated.

Lead exposure. 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 could lead to an overestimation of exposure in rural adults because lead exposures are usually lower in adults than in children. Nationally representative blood lead surveys are necessary in our country to improve the accuracy of these estimates.

Our estimate of burden attributable to lead exposure is probably an underestimate of the true attributable burden because only mild mental disability caused by IQ reduction and CVD endpoints were considered in this analysis. The burden of gastrointestinal symptoms and anemia caused by lead was not assessed as the rate of illness for these related health outcomes was negligible. Several additional related health outcomes, such as increased delinquent behavior 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 in '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.

Recommendations

Urban air pollution. The assessment of exposure to outdoor air pollution for this study is based on data obtained from the available air pollution monitoring network, rather than from a network specifically designed to estimate population-weighted exposure. This situation made it necessary to assume that the underlying distribution of vulnerable groups is consistent across studies. It is important for South Africa to develop a national air-quality monitoring network that covers all significant urban settlements (populations > 100,000), standardized with respect to instrumentation, data quality assurance, and

reporting formats. The location of monitoring stations within each urban area should be in conformity with accepted international practice. The location of monitoring stations within areas of high population density and proximal to known significant sources of pollutant emissions ('hot-spots') will ensure an improved population-weighted estimate of exposure and of the impact of specific emission sources.

In addition, we must develop urban-scale air-quality mathematical models for all urban settlements. Air-quality modeling combined with monitoring data is capable of relating pollution sources and atmospheric processes of dispersion and chemical transformation to ambient concentrations, thus providing a potentially more accurate estimate of population-weighted exposure. Other recommendations include encouraging movement away from the use of dirty (highly polluting) fuels such as coal, wood and paraffin for domestic purposes to cleaner fuels such as liquefied petroleum gas (LPG) and electricity, and use of cleaner fuels as industrial fuel, as well as installation of air pollution control devices to minimise industrial emissions. In addition, expanding the use of renewable and environmentally friendly energy sources such as solar or wind power and upgrading combustion technology especially for diesel engines and stationary sources such as power plants, incinerators, industrial boilers and residential cooking and heating appliances is important. Strict regulations on open burning of waste and uncontrolled burning of forests and agricultural fields are also essential.

Indoor air pollution from household use of solid fuels. The most important interventions to reduce the impact of indoor air pollution include access to cleaner household fuels, improved stoves, and better ventilation as described in detail elsewhere /7/. The use of solid fuels has a negative impact on household economies due to the time spent harvesting, storing, and preparing such fuels. Thus, time is deducted from hours that could be spent on other tasks including childcare, education, domestic hygiene, commercial activities and rest and relaxation, particularly for women, thereby having a negative impact on health and well-being. Notably, other fuels carry health risks too. For example, households using paraffin and gas for cooking and heating can also be exposed to pollution, largely related to stoves' quality, and are also at risk of fire injuries and childhood poisonings associated with the use of paraffin. Access to electricity is therefore key to good health, breaking the cycle of poverty, and to promoting sustainable development. Yet, health risks are involved in providing electricity to households as well, including occupational hazards from coal mining, air pollution from power plants, and nuclear plant accidents /76/.

Unsafe WSH. A substantial proportion of disease burden due to unsafe WSH in South Africa could be prevented if water supplies, sanitation services, and hygiene behaviors were further improved. Significant headway has already been made in improving water supplies. Since 1994, an additional 18.6% of the population has gained access to improved water within 100 m of their home. These upgrades are largely the result of the Department of Water Affairs and Forestry's capital works program, which provided new water services for approximately seven million people between 1994 and 2002 /100,101/. Access to safe sanitation also improved, from 48.7% to 63.7% between 1994 and 2004 /52/, suggesting that exposure to unsafe WSH in South Africa decreased over the last decade /102/.

Nevertheless, rural-urban and intra-urban differentials in access to safe WSH remain a concern. The residents of inner-city, low-income, high-density areas and urban informal settlements struggle to gain access to basic services or share these with many households. Variation in access between urban and rural areas is also stark:

although only 5% of urban households did not have access to purified water and any toilet in 2001, 37% and 28% of rural households did not have access to purified piped water or any form of toilet, respectively /52/.

Reducing the risks to which children are exposed through unsafe WSH is particularly important, given the large disease burden attributable to this risk in children under five years of age. Reducing the unsafe WSH risk includes decreasing diarrhea risks in the household and in local environments. A study in Port Elizabeth, Eastern Cape Province, showed that diarrhea levels were significantly higher in children under six years of age who shared a tap with more than six other households, and also in those who shared a toilet with more than five households /103/. Informal preschool and child-care facilities have also been shown as an important environment for the transmission of diarrheal diseases /104/.

Although improving access to adequate water and sanitation facilities is key, the impact of both personal and domestic hygiene behaviors should not be neglected. In the same Port Elizabeth study, diarrhea levels were 4.8 times higher in caregivers who stored water in their kitchens /103/—a risk that could be mitigated by improved domestic hygiene behaviors.

Reviews have suggested that good hygiene may result in 33% reduction in diarrheal mortality /46/, and that hygiene education is a highly cost-effective intervention for reducing childhood diarrheal diseases /105/. A systematic review of the impact of hand-washing with soap showed that this cheap intervention could reduce diarrheal risk in the community by 42% to 44% /45/. Improvements in hygiene behavior should also reduce intestinal parasite infections, yet how best to change hygiene behaviors in different settings remains unclear /45, 106/. The secondary prevention of diarrheal diseases is also important. Impacts can be mitigated by prompt and appropriate treatment at the household and health facility level. Providing hygiene information to caregivers of children with diarrheal disease could also contribute to reducing disease spread within households and the likelihood of further episodes.

Improving WSH is also likely to have positive impacts on household economies due to the time saved in water collection /107/. As discussed under indoor air pollution, this saving would also free time for other tasks, including education, domestic hygiene, and commercial activities /108, 109/, particularly for female children and women /103, 110/. All these activities are likely to contribute to improved health. Improving WSH is therefore key to breaking the cycle of poverty and disease and to promoting development.

Note that in the same way as indoor air pollution was discussed previously, unsafe WSH may work jointly or synergistically with other risk factors, like underweight/malnutrition (including iron deficiency anemia and vitamin A and/or zinc deficiency), to increase the incidence and effects of such diseases as diarrhea and intestinal parasites. Some risks related to unsafe WSH can be mediated through underweight whereas, equally, some risks for underweight may be mediated through WSH-related diseases like diarrhea /48,111/. In South Africa, HIV/AIDS may also be an important moderator of the risk of diarrheal disease from unsafe WSH, particularly in children living in informal settlements /112/

The synergistic effects, however, are not within the scope of this analysis. Nevertheless, policies to reduce disease burden due to diarrheal diseases have to be cross-sectoral to reduce exposure to multiple risks. The increased demand for water for providing care for a household member living with AIDS also highlights the importance of improved access and multisectoral developmental approaches /113/. Government programs, such as those focusing on informal settlement eradication and integrated rural development, as well as the Extended Public Works Programme, provide opportunities for this approach.

Lead exposure. Several social factors assume importance in predisposing children to lead in the environment /64/. Exposure to lead tends to be higher in the lower socioeconomic groups of the population /114/. Poorer populations are forced to live in areas that are more exposed to industrial pollution, closer to busy roads and highways, or in degraded housing with old, flaking lead-based paint and lead water pipes, which has serious implications for inequalities in health.

Albeit in a piecemeal fashion, some significant developments have occurred in South Africa in recent years that hold considerable promise of reducing children's exposure to lead. In 2006, the use of leaded petrol was phased out. In addition, following the discovery by Medical Research Council researchers that lead is still being added to certain paints in South Africa, and in some instances at extremely high concentrations, legislation was drafted to prohibit this practice /115/. A lead hazard awareness initiative was also launched by the Department of Health and the Medical Research Council of South Africa. These steps are likely to lead to significant reductions in lead exposure among South African children. The burden attributable to lead in South Africa is, in principle, preventable through the implementation of a comprehensive, intersectoral lead poisoning prevention program. Such a program should include research to identify emerging risk factors for elevated blood lead concentrations in children as well as in adults (especially with regard to 'cottage industries' and the use of lead in cultural/traditional practices), significant scaling up of awareness programs to address the 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 must 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. Given the possible associations between lead exposure and poor school performance as well as delinquent behavior, 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 is extremely important.

CONCLUSION

As many policy-makers are not aware of the array of health effects associated with exposure to the environmental factors discussed here, the quantification of health risks associated with exposure can be an effective guide, as well as an educational tool. Such quantification of risks will also provide an indication of the level of effort that is necessary in a given city, region, or control strategy. For this reason, this first quantification of the impact of exposure to environmental risks on public health in South Africa is such a crucial first step in developing successful policies and strategies in the control of these environmental factors.

This study estimated that about 24,000 deaths in 2000 were attributable to exposure to four elected environmental risks, highlighting the significant public health impact of exposure to environmental risks and the significant burden of preventable disease attributable to exposure to these environmental risk factors in South Africa. The study identified that the major contribution to the disease burden attributable to these environmental factors arises from the diarrhea burden related to unsafe WSH. While clear improvements have been made in access to water and sanitation during the past 15 years, extending these services further will be essential to reduce this burden. In terms of the other environmental factors, macro-economic policy reforms to promote growth, employment, equity, trade, and reducing inflation, must be carefully planned to ensure that

these issues do not also encourage environmentally unsustainable practices. Such planning involves full cost accounting (e.g. by removing distortions from the economy such as energy subsidization), taxing pollution and waste generation, managing interest rates so that harmful land-use practices are discouraged, and providing alternatives to informal sector activities that use environmental resources unsustainably. South Africa is a disproportionately large producer of carbon emissions, with much of its relatively cheap electricity produced by coal-fired power stations. Considering the impact of climate change on the future burden of disease will be important so that the full disease burden can be assessed. A need exists for the development and implementation of evidence-based policies and programs to jointly address these environmental risk factors through an integrated approach toward improving environmental health at the individual, household, community, and societal levels. A considerable body of evidence has already emerged regarding effective environmental health interventions /116/ and how to implement them, If marshaled, this evidence and experience could result in considerable improvements in the health of South Africans.

ACKNOWLEDGMENTS

The other members of the Burden of Disease Research Unit of the South African Medical Research Council—Pam Groenewald, Michelle Schneider, Desireé Pieterse, Jané Joubert, Beatrice Nojilana, Karin Barnard and Elize de Kock—are thanked for their valuable contribution. We also thank Angela Mathee, Brendon Barnes, Elizabeth Thomas, Jongikhaya Witi, and the South African Comparative Risk Assessment Collaborating group. Ms. Ria Laubscher and Dr. Lize van der Merwe of the MRC Biostatistics Unit made contributions via their statistical expertise and assistance. Our sincere gratitude is also expressed for the valuable contribution of Associate Professor Jan Barendregt of the School of Population Health, University of Queensland for providing technical expertise and assistance. Dr. Shalomie Shadrach is gratefully acknowledged for editing the manuscript.

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