



Estimating the burden of disease attributable to urban outdoor air pollution in South Africa in 2000

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Objectives. To quantify the mortality burden attributed to urban outdoor air pollution in South Africa in 2000.

Design. The study followed comparative risk assessment (CRA) methodology developed by the World Health Organization (WHO). In most urban areas, annual mean concentrations of particulate matter (PM) with diameters less than 10 μm (PM_{10}) from monitoring network data and PM with diameters less than 2.5 μm ($\text{PM}_{2.5}$) derived using a ratio method were weighted according to population size. PM_{10} and $\text{PM}_{2.5}$ data from air-quality assessment studies in areas not covered by the network were also included. Population-attributable fractions calculated using risk coefficients presented in the WHO study were weighted by the proportion of the total population (33%) in urban environments, and applied to revised estimates of deaths and years of life lost (YLLs) for South Africa in 2000.

Setting. South Africa.

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

Outcome measures. Mortality and YLLs from lung cancer and

cardiopulmonary disease in adults (30 years and older), and from acute respiratory infections (ARIs) in children aged 0 - 4 years.

Results. Outdoor air pollution in urban areas in South Africa was estimated to cause 3.7% of the national mortality from cardiopulmonary disease and 5.1% of mortality attributable to cancers of the trachea, bronchus and lung in adults aged 30 years and older, and 1.1% of mortality from ARIs in children under 5 years of age. This amounts to 4 637 or 0.9% (95% uncertainty interval 0.3 - 1.5%) of all deaths and about 42 000 YLLs, or 0.4% (95% uncertainty interval 0.1 - 0.7%) of all YLLs in persons in South Africa in 2000.

Conclusion. Urban air pollution has under-recognised public health impacts in South Africa. Fossil fuel combustion emissions and traffic-related air pollution remain key targets for public health in South Africa.

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Epidemiological research^{1,2} has found significant associations between outdoor air pollution (even at low ambient concentrations) and various morbidity and mortality 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.³

Anthropogenic ('man-made') air pollution is a complex mixture with many toxic components. The commonly found air pollutants are sulphur dioxide (SO_2), oxides of nitrogen (NO_x), ozone (O_3), volatile organic compounds and suspended particulate matter (PM). PM 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.⁴ Suspended PM is divided into three fractions, namely $\text{PM}_{2.5}$ which is PM with diameters less than 2.5 μm , PM with diameters less than 10 μm

(PM_{10}), and suspended PM with diameters greater than 10 μm (suspended PM > PM_{10}).

Epidemiological studies use several indicators of exposure to assess the effects of this complex mixture, including NO_2 , CO, PM_{10} , $\text{PM}_{2.5}$, total suspended particulates, and SO_2 . However, according to a study done by Sarnat *et al.*,⁵ these pollutants are strongly correlated in concentration, thus leading to a criticism that health effect estimates of air pollution may be confounded by gaseous co-pollutants. Hence, epidemiological studies cannot strictly allocate observed effects to single pollutants, and a 'pollutant-by-pollutant' assessment may overestimate the impact.¹ Many studies on health effects indexed exposure to this pollution mixture in terms of PM, a useful indicator of several sources of outdoor air pollution such as fossil-fuel combustion.¹ Samet *et al.*⁶ analysed the components of ambient air pollution (PM_{10} , O_3 , NO_2 , CO and SO_2) and daily mortality data from 20 cities and found PM_{10} to be a significant predictor of daily mortality, controlling for the gaseous co-pollutants. Schwartz⁷ examined 10 cities separately during summer and winter and reported identical associations between daily mortality and PM_{10} . For quantitative assessment of health effects, $\text{PM}_{2.5}$ and PM_{10} are most often selected as exposure metrics for epidemiological studies.³

A recent review of air pollutants in South Africa⁸ observed that the main anthropogenic sources of PM emissions are

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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 unelectrified areas, mainly underdeveloped rural areas and peri-urban settlements.

Living in a middle-income country, South Africans are simultaneously at risk of ill health related to industrialisation and to underdevelopment. People in industrially developed urban settings are exposed to urban air pollution and lead, while those in underdeveloped peri-urban settlements face additional environmental risks from inadequate access to water and sanitation and indoor smoke from solid fuels. The focus of this article is on quantifying the impact of urban outdoor air pollution. The burden attributable to exposure to indoor smoke from solid fuels, lead and unsafe water, sanitation and hygiene are quantified separately in related articles in this supplement. It is important to note that limiting the analyses to urban areas may underestimate the burden attributable to this risk factor as there is outdoor air pollution in rural areas that would not be captured by the indoor pollution estimates.

A legacy of apartheid-era town planning was the location of industry and working-class communities in close proximity. While this served rapid industrial growth in the 1960s and 1970s, population growth in those communities on the one hand and increasing production by the industries on the other has led to a major environmental dilemma for the country as a whole.⁹ In a number of 'hot-spot' areas, large industrial sources located close to poor communities result in high exposures. The SO₂ emissions from oil refineries in the South Durban industrial basin and Cape Town, dust emissions from mine tailings in Gauteng, and SO₂ emissions from steel and chemical plants in the Vaal Triangle are examples of industrial areas that pose a danger to the health of people living in close proximity to them.

White *et al.*¹⁰ conducted a study in the north-west quarter of the City of Cape Town on the basis of community concern that a petrochemical refinery in the area had an impact on their health. The petrochemical refinery produces approximately 18 tons of SO₂ daily, and the available monitoring data indicated a significant contribution by the refinery to ambient levels in the area. The study showed a measurable health effect, with more frequent asthmatic symptoms in schoolchildren associated with meteorologically estimated petrochemical emissions dose, indicating a substantive basis for the community concern. In this study area petrochemical refinery emissions were shown to be the most important risk factor for allergic disease symptoms in the ambient environment.¹⁰ A study of respiratory conditions in children living in areas exposed to higher levels of community air pollution found increased odds of 1.3 compared with areas with less pollution.¹¹

Opperman *et al.*¹² 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 µg/m³ in 1992). The large, recently completed South Durban Health Study⁹ by the Centre for Occupational and Environmental Health of the University of KwaZulu-Natal found that relatively moderate ambient concentrations of NO₂, NO, PM₁₀ and SO₂ were strongly and significantly associated with decrements in lung function among children with persistent asthma.

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 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 it difficult to assess overall exposure to urban air pollution. At this stage the national monitoring network is limited, uneven in distribution across the urban population, and not standardised. In general, it does not conform to recommended international practice.¹³

The aim of this study was to make use of the available data to estimate population exposure to urban outdoor air pollution (indexing this complex mixture in terms of PM₁₀ and PM_{2.5}) and the mortality burden attributed to this exposure by sex and age group in South Africa for the year 2000. Consistent with the global assessment,³ only attributable mortality estimates are included in this analysis since mortality effects of air pollution are the most important, and local incidence data required for morbidity estimates are lacking.

Methods

Comparative risk assessment (CRA) methodology was used, as developed by the World Health Organization.^{14,15} The disease burden attributable to exposure to this particular risk factor was estimated by comparing the current observed risk factor distribution with a counterfactual risk factor distribution, conferring the lowest possible population risk (the theoretical minimum distribution). The population-attributable fraction (PAF) requires estimation of the gradient of risk between the theoretical minimum level of air pollution exposure and the estimated observed national urban exposure.

PM₁₀ and PM_{2.5} were used as exposure metrics for the reasons mentioned earlier. In the absence of background air pollution data we used the counterfactual or theoretical minimum risk exposure annual average values of 7.5 µg/m³ for PM_{2.5} and 15 µg/m³ for PM₁₀, as estimated by the global urban outdoor air pollution risk assessment study.³ These values are also close to the lowest concentrations observed in epidemiology studies by Pope *et al.*,¹⁶ from which the concentration response functions used in the global assessment were derived. According to Cohen *et al.*³ this approach also avoids extrapolating the concentration response functions below the concentrations observed in the epidemiological studies, although health



benefits may well be gained from reductions below those concentrations.

Urban areas in this study comprised the 6 metropolitan areas (metros) defined by Statistics South Africa¹⁷ and the Sasolburg district that falls in the Vaal Triangle. Metros are conurbations featuring high population density; intense movement of people, goods and services; extensive development; and multiple business districts and industrial areas.¹⁷ Annual mean PM₁₀ concentrations were calculated from continuous measurements (mostly hourly) taken in the years 2000 - 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.¹⁸ 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.⁸ The Tshwane metropolitan area, 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 Fig. 1 shows that monitoring networks and stations across the country are largely situated in metropolitan areas.

The PM₁₀ estimates were converted to estimates of PM_{2.5} using available information on geographical variation, factors influencing the ratio of PM_{2.5} to PM₁₀, as well as the observed ratio from local studies where monitoring data were available for both PM₁₀ and PM_{2.5}. For these areas the ratio was observed to be between 0.5 and 0.65.^{8,19} In areas without local data on PM_{2.5}/PM₁₀ ratios, we assumed a ratio of 0.5. A ratio of 0.35 was assigned to peri-urban areas with high fugitive emissions (e.g. 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.³

Population-weighted annual average concentrations of PM₁₀ 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 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 localised sources and the more distant air pollution sources. The 'small area level' dataset from Census 2001¹⁷ 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 Medical Research Council.

Variations across the impact zone are assumed to average across the area as well as with time over an annual period. Population-weighted mean PM₁₀ and PM_{2.5} concentrations for

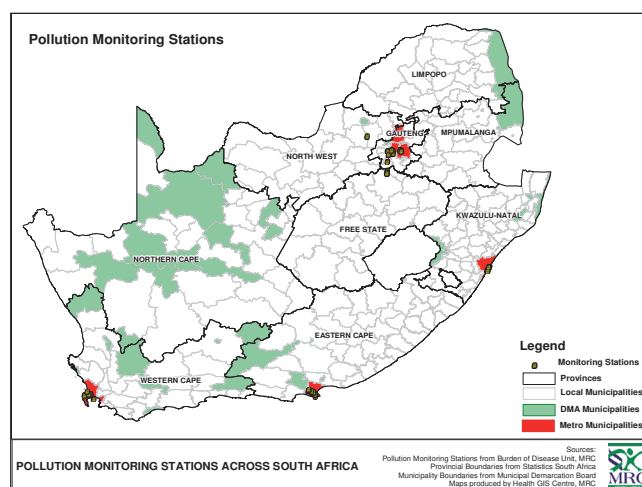


Fig. 1. Map showing monitoring stations (networks and studies) across the country.

all urban areas in South Africa in 2000 were also estimated (Table I). When compared with the estimates of PM₁₀ generated for South Africa by the Global Model of Ambient Particulates (GMAPs) developed by the World Bank,²⁰ the model tended to underestimate PM₁₀ concentrations by about 50%.¹⁸ It was felt that our estimates provided a more realistic picture since they were based on site-specific data (air pollution monitoring data) and the exposed population. The GMAPS model, on the other hand, is based on many factors (e.g. income per capita) which are averaged across the whole population. It is, however, common knowledge that there may be significant differences in these factors, particularly in semi-urban areas.

Exposure to outdoor air pollution is associated with a broad spectrum of acute and chronic health effects, ranging from eye irritation to death. The health effects associated with PM exposure include lung cancer and respiratory disease and some specific cardiovascular outcomes.²¹ The three health outcomes assessed by Cohen *et al.* in the 2004 global CRA study³ were included in this study, classified using ICD-9 codes²² (Table II): (i) mortality due to cardiopulmonary disease in adults aged 30 years and older; (ii) mortality due to lung cancer in adults aged 30 years and older; and (iii) mortality due to acute respiratory infections (ARIs) 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 hospitalisation for cardiovascular or respiratory disease, emergency room and urgent care visits, asthma exacerbation, acute and chronic bronchitis, respiratory symptoms and decreased lung function.³

A recent review of South African-based studies of the health effects of air pollution concluded that none were able to provide local estimates of the risk.²³ To estimate the relative risk (RR) of mortality from cardiopulmonary disease and lung cancer in adults aged 30 years and older, Cohen *et al.*³ used the



results of the American Cancer Society (ACS) study, a large cohort study that links data from 500 000 cohort members with data on air pollution from metropolitan areas throughout the USA.¹⁶ In these analyses, we used estimates from Cohen *et al.*'s³ base-case analyses and results from the linear regression model in which annual average concentrations measured from 1979 - 1983 ACS data were used as estimates of exposure.¹⁶

Estimates of coefficients of concentration-response functions are presented in Table II, adjusted for confounding factors. To estimate the relationship between exposure to PM and mortality from ARIs among children aged 0 - 4 years, Cohen *et al.*³ computed a summary estimate from five published time-series studies. The five studies were summarised as a weighted average of the estimates from individual studies,

Table I. Population-weighted mean PM₁₀ and PM_{2.5} concentrations for urban areas, South Africa, 2000

Metro/urban area	Population*	Mean PM ₁₀ concentration (µg/m ³)	PM _{2.5} /PM ₁₀ ratio	Estimated PM _{2.5} concentration (µg/m ³)	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
Population-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 2001.¹⁷
 CJ = City of Johannesburg; SS = Scientific Services Cape Town; EM = Ethekwini Municipality (Durban);
 F = Fridge Study;⁸ NMM = Nelson Mandela Metro (Port Elizabeth).

Table II. Estimates of relative risk of mortality, coefficients of concentration-response functions and data sources for related health outcomes

Health outcome	ICD-9 codes ²²	Data source	PM exposure metric	Concentration-response slope* per µg/m ³ (SE)	Relative risk per 10 µg/m ³ (95% CI), from data source
Mortality from cardio-pulmonary disease, adults ≥ 30 yrs	401-440, 460-519 (ill-defined cardiovascular causes of death proportionally re-distributed across all specified causes except stroke)	ACS study ¹⁶	PM _{2.5}	Linear [†] 79 - 83 0.00575 (0.002160) Log-linear average ^{‡§} 0.155148 (0.050460)	1.059 (1.015 - 1.105)
Mortality from lung cancer, adults ≥ 30 yrs	162, 166 (trachea/bronchi lung cancer combined in SA NBD list)	ACS study ¹⁶	PM _{2.5}	Linear 79 - 83 0.00789 (0.003447) Log-linear average 0.232179 (0.074770)	1.082 (1.011 - 1.158)
Mortality from acute respiratory infection, children aged 0 - 4 yrs	460-466, 480-487, 381-382	St George's Hospital meta-analysis of five time-series studies of daily mortality ³	PM ₁₀	0.0010 (0.0010)	1.010 (0.991 - 1.031)

Adapted from Cohen *et al.*, 2004.³
 *Slope of the concentration-response function for air pollution and mortality.
 †Base-case scenario: Results from regression models in which annual average concentrations measured from 1979 to 1983 were used as estimates of exposure (Pope *et al.*, 2002¹⁶).
 ‡Alternative scenario (case 6):³ Results from regression models in which the average of annual average concentrations measured from 1979 - 1983 and 1999 - 2000 were used as estimates of exposure (Pope *et al.*, 2002¹⁶), and where exposure (i.e. annual average PM_{2.5}) is specified on the log scale.
 §RR per 10 µg/m³ will depend on the specific concentrations calculated and hence is not presented.
 SE = standard error; SA NBD = South African National Burden of Disease Study.



with the weights determined by the inverse of the reported variance in the concentration-response function (Table II). After deriving the concentration-response functions for the three endpoints, they 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} which were used in this study:

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

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

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, from Table II. Cohen *et al.*³ limited the risk of mortality in any city to no greater than that attained at a $PM_{2.5}$ concentration of $50 \mu\text{g}/\text{m}^3$, and assumed that the risk of death increases linearly over a range of annual average concentrations of $PM_{2.5}$, between a counterfactual concentration of $7.5 \mu\text{g}/\text{m}^3$ and a maximum of $50 \mu\text{g}/\text{m}^3$. Similarly, concentrations of PM_{10} were truncated at $15 \mu\text{g}/\text{m}^3$ and $100 \mu\text{g}/\text{m}^3$.³ In our local study the estimated annual average concentration of $PM_{2.5}$ and PM_{10} did not exceed $50 \mu\text{g}/\text{m}^3$ and $100 \mu\text{g}/\text{m}^3$ respectively, in any urban area (Table I).

Since there is considerable uncertainty regarding the timing of exposure with regard to the risk of mortality,²⁴ Cohen *et al.*³ also calculated alternative estimates using the reported ACS coefficients, based on the average of past (1979 - 1983) and more recent (1999 - 2000) annual average concentrations using a log-linear (case 6) extrapolation (also presented in Table II). Given the current lack of knowledge concerning both the relevant induction time for exposure and chronic effects and the shape of the concentration-response curve, this may be more justifiable (A Cohen, Health Effects Institute – personal communication, 2006), and hence a sensitivity analysis was carried out in this study using the case 6 alternative scenario.

Population-attributable fractions (PAFs) for the 3 endpoints were calculated in MS Excel using the formula:

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

where P is the prevalence of exposure (indexed as population-weighted mean PM_{10} or $PM_{2.5}$ concentrations (depending on the health outcome) for urban areas in South Africa), and RR is the relative risk of mortality 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 residing in the six metropolitan areas (including Sasolburg) using Census 2001 data (33%),¹⁷ and assuming that the non-metropolitan areas were not at risk. To calculate attributable burden, these national PAFs were applied to the number of deaths and years of life lost (YLLs) due to

premature mortality for each selected outcome extracted from the revised burden of disease estimates for South Africa 2000, with methods and assumptions described elsewhere.²⁵ Since it was not possible to estimate the impact of PM on the incidence of disease, disability-adjusted life years (DALYs) quantify only YLLs.

We used Monte Carlo simulation-modelling techniques to present uncertainty ranges around point estimates that reflect all the main sources of uncertainty in the calculations. We used @RISK 4.5 for Excel,²⁶ which allows multiple recalculations of a spreadsheet, each time choosing a random value from distributions defined for input variables. 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,^{3,8} 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 the RR estimates we specified a normal distribution based on the published standard errors for the slope of the concentration-response function for each of the three endpoints (using the base-case analyses).^{3,16} We calculated 95% uncertainty ranges for our output variables, namely attributable burden as a percentage of total burden in South Africa in 2000 bounded by the 2.5th and 97.5th percentiles of the 2000 iteration values generated.

Results

The annual average concentrations of PM_{10} and $PM_{2.5}$ in metropolitan and other urban areas of South Africa are presented in Fig. 2. The annual population-weighted average concentration of PM_{10} was estimated at $46.9 \mu\text{g}/\text{m}^3$ (95% uncertainty interval $44.9 - 48.8 \mu\text{g}/\text{m}^3$) and $PM_{2.5}$ at $26.6 \mu\text{g}/\text{m}^3$ (95% uncertainty interval $24.8 - 28.5 \mu\text{g}/\text{m}^3$) for all urban areas in South Africa, 2000. The highest annual concentrations of PM_{10} and $PM_{2.5}$ were estimated for the Vaal Triangle, followed by Orange Farm. Although PM_{10} concentrations in the mining town of Rustenburg were third highest, the $PM_{2.5}$ concentration was relatively low. Both PM_{10} and $PM_{2.5}$ concentrations were also high in the peri-urban areas of Khayelitsha and Soweto. The Cape Town Metropole had the lowest concentration of both PM_{10} and $PM_{2.5}$.

Outdoor air pollution in urban areas in South Africa was estimated to cause 3.7% of the total mortality from cardiopulmonary disease in adults aged 30 years and older, 5.1% of mortality attributable to cancers of the trachea, bronchus and lung in adults, and 1.1% of mortality from ARIs in children under 5 years of age. This amounts to an estimated 4 637 deaths or 0.9% (95% uncertainty interval 0.3 - 1.5%) of all deaths and 42 219 YLLs or 0.4% (95% uncertainty interval 0.1 - 0.7%) of all

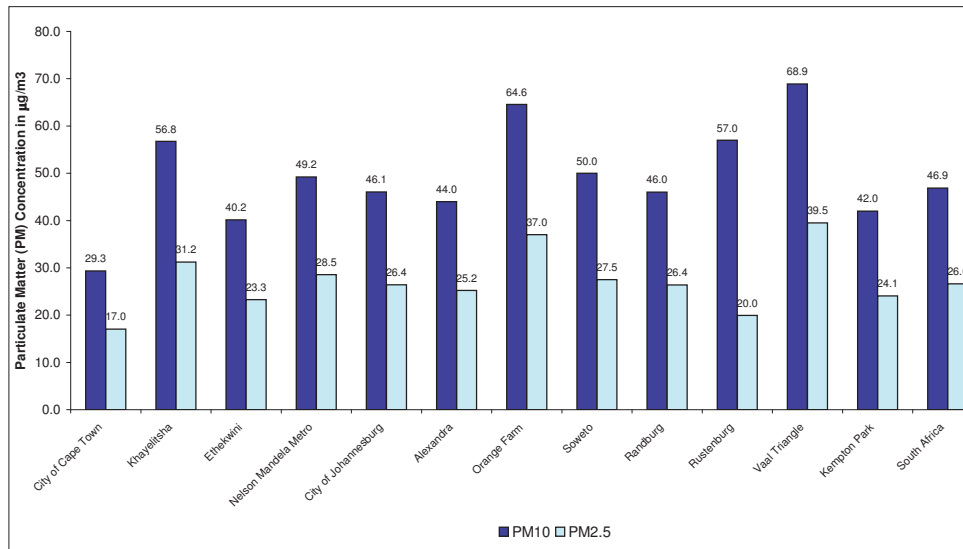


Fig. 2. Population-weighted annual average PM₁₀ and PM_{2.5} concentrations for South African urban areas in 2000.

Discussion

This study suggests that in the urban areas of South Africa, the average annual exposures to ambient PM₁₀ (46.9 µg/m³) and PM_{2.5} (26.6 µg/m³) are at levels well above those considered to be without increased risk of mortality (15 µg/m³ and 7.5 µg/m³ respectively). This exposure to urban air pollution using base case analyses by Cohen *et al.*³ has been estimated to cause some 4 637 deaths or 0.9% of all deaths in 2000. The uncertainty range is fairly wide (95% uncertainty interval 0.3 - 1.5%), but estimates of mortality attributable to urban outdoor air pollution based solely

on the effect of annual average exposure to PM_{2.5} and PM₁₀ are probably an underestimate of the actual burden. If it were possible to identify, accurately measure and include exposure to all known pollutants in the country, the burden attributable to this risk factor would be higher although it is noted that there may be interrelated effects of certain pollutants.^{1,27} 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. Sensitivity analyses using the case 6 alternative scenario or log-linear extrapolation of the larger coefficients from the ACS study^{3,16} doubled the number of attributable deaths and YLLs from cardiopulmonary disease and lung cancer relative to base-case estimates.

YLLs in persons in South Africa in 2000 (Table III). Although the attributable fractions for cardiopulmonary mortality were identical for men and women, the high number of deaths caused by hypertensive disease and cerebrovascular diseases in females led to more female than male attributable deaths.

Fig. 3 shows that most of the YLLs (86.3%) attributable to exposure to urban outdoor air pollution are due to cardiopulmonary mortality in adults aged 30 years and older. Lung cancer mortality in adults (8.5%) and ARIs in children under 5 (5.2%) accounted for much smaller proportions of the total attributable burden.

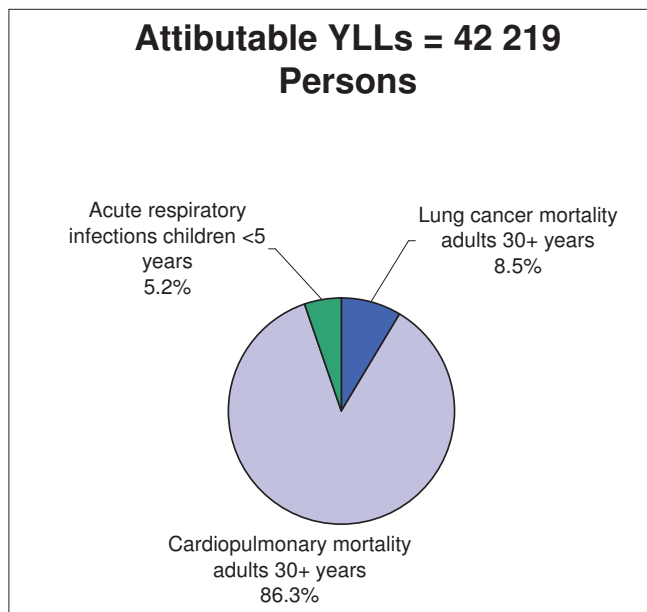


Fig. 3. Years of life lost attributable to urban outdoor air pollution, South Africa, 2000.

The results of the global risk assessment study³ indicated that the impact of air pollution on the burden of disease in cities around the world was large, but also likely to be an underestimate of the actual burden, on the basis of an assessment of sources of uncertainty. The study also revealed a 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 developing countries. The global study estimated similar PAFs, 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. This amounted to 0.8 million deaths, representing about 1.4% of the total global deaths.³ The highest proportions of the total burden occurred in Western Pacific Region-B and European Region-B, where urban air pollution caused 0.7 - 1.0% of the burden of disease.³





Table III. Summary of mortality burden attributable to urban outdoor air pollution, South Africa, 2000

Related health outcomes	Attributable deaths			Attributable YLLs		
	Males	Females	Persons	Males	Females	Persons
Lung cancer (adults 30+ yrs)	237	113	350	2 449	1 155	3 604
Cardiopulmonary disease (adults 30+ yrs)	1 936	2 286	4 222	18 031	18 391	36 423
Hypertensive disease	189	419	608	1 754	3 239	4 993
Ischaemic heart disease	651	613	1 264	5 694	4 088	9 782
Stroke	483	742	1 225	4 479	6 144	10 623
Inflammatory heart disease	85	92	177	944	899	1 843
Other cardiovascular*	59	80	139	665	846	1 510
COPD	287	179	466	2 385	1 415	3 799
Asthma	119	118	237	1 344	1 300	2 644
Other respiratory diseases*	64	42	106	765	462	1 227
Acute respiratory infections (children 0 - 4 yrs)	34	32	65	1 130	1 062	2 193
Lower respiratory infections	33	31	64	1 103	1 041	2 144
Upper respiratory infections	1	1	1	25	20	45
Otitis media	0	0	0	2	1	4
Total	2 207	2 430	4 637	21 610	20 609	42 219
95% uncertainty interval	714 - 3 699	675 - 4 174	1 432 - 7 884	7 185 - 35 736	6 036 - 34 845	13 405 - 70 741
% of total burden	0.8%	1.0%	0.9%	0.4%	0.4%	0.4%
95% uncertainty interval	0.3 - 1.3%	0.3 - 1.7%	0.3 - 1.5%	0.1 - 0.6%	0.1 - 0.7%	0.1 - 0.7%

*These disease categories were included because certain ICD-9 codes listed in Table II appeared in these categories. COPD = chronic obstructive pulmonary disease; YLL = years of life lost.

It is important to note that 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, there is a greater degree of uncertainty when morbidity findings are extrapolated to developing countries, because the estimation requires both a concentration-response function and a baseline incidence rate.²¹ Due to 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 morbidity outcomes such as asthma exacerbation. However, this will depend on further research into the health effects of air pollution in developing 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: SO₂, NO₂, O₃, CO and volatile organic compounds such as benzene, a well-known haematopoietic carcinogen.

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 a network specifically designed to estimate population-weighted exposure. This 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 (with populations > 100 000), standardised with regard 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. 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 need to develop urban-scale air-quality mathematical models for all urban settlements. Air-quality modelling 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.

Since many policy-makers are not aware of the array of health effects associated with exposure to outdoor air pollution, 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. That is why this first quantification of the impact of air pollution on public health in our country, which can be used to create awareness of the associated health risks, is such a crucial first step in developing successful policies and



strategies on the control of air pollution. Further sensitivity analyses using a more feasible counterfactual indicate that if we could achieve an improvement in PM_{2.5} concentrations in the future and successfully reduce local levels to the new WHO Air Quality Guideline²⁸ of 10 µg/m³ in urban areas, then more than 600 deaths and 5 300 YLLs could be prevented in a year.

Recommendations

The current system of regulation for the control of all sources of air pollution in South Africa is inadequate, a situation that the recently promulgated Air Quality Management Act (Act 39 of 2004), which became effective in September 2005, promises to address.²⁹ As the South African economy continues to develop and the urban populations grow, it is essential to implement strategies to control air pollution. One of the policy principles for air-quality management is environmental justice, and one of the objectives of the air-quality management plan is to consider air quality in land use and transport planning.

Possible control and intervention strategies that need to be considered by all spheres of government include the following.

1. Monitoring strategies for volatile organic compounds, especially benzene, an international priority pollutant which is not currently being monitored routinely in South Africa.

2. A reassessment of the current revised fuel specifications that have not dealt adequately with benzene and total aromatics. Petrol can contain up to 5% benzene and 55% aromatics by volume³⁰ while European specifications (Euro IV)³¹ are considerably more stringent and stipulate a maximum of 1% and 35%, respectively, since January 2005.

3. Even though the evidence base for the toxicity of benzene is very strong, there has been a limited public health response due to lack of knowledge of its toxicity³² by town planners and the public. Residential petrochemical exposure has been found to be a significant risk factor for leukaemia.³³ Furthermore, several studies have shown that benzene levels in the vicinity of petrol stations adjacent to residential housing is of concern with regard to human health.³⁴⁻⁴¹ Consideration should also be given to banning the location of petrol stations and other hazardous activities in residential areas.

4. Air pollution control regulation to reduce emission of volatile organic compounds from petrol filling stations during bulk storage tank filling and vehicle filling operations.

5. Promotion of the use of public transport instead of private cars and long-term strategies to provide an alternative to cars and diesel buses, including rail, electric- or alternative fuel-powered buses, and cycling/walking networks.

6. Land use strategies that emphasise compact urban design around public transport and/or pedestrian and cycle networks. Indirect benefits that may accrue from these include traffic injury prevention, noise reduction, and creation of spaces for exercise and recreation.

7. Encourage 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.

8. Expanding the use of renewable and environmentally friendly energy sources such as solar or wind power.

9. Improvement or upgrading of combustion technology especially for diesel engines and stationary sources such as power plants, incinerators, industrial boilers and residential cooking and heating appliances.

10. Regulations on open burning of waste and uncontrolled burning of forests and agricultural fields.

Typically, mobile sources contribute between 24% and 47% of PM concentrations in urban areas, while biomass burning may be the largest source in rural areas.⁸ In peri- or semi-urban environments a combination of fugitive emissions from unpaved roads or loose soil, mobile sources and biomass burning are significant contributors. Industrial sources also play a major role in PM concentrations. To select the most suitable interventions, an inventory of the principal local and regional sources of PM is essential. At individual level, reducing air pollution can be achieved by use of non-motorised transport, conserving energy, and using appliances with cleaner technologies.

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