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Final Project Report

Multipoint Plan: Project 4 Health Study and Health Risk Assessment

South Durban Health Study

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Executive Summary

The industrial basin in the south of Durban is a legacy of the apartheid era town planning. The basic approach at that time was to locate industry and working class communities in close proximity to each other, which ensured adequate control of the disenfranchised in their resource challenged townships, but which simultaneously ensured the provision of labour to the burgeoning exclusively white industry. While this may have served rapid industrial growth in the 1960s and 1970s, the increasing size of the communities on the one hand -- and the increasing production of the factories on the other -- has resulted in a major environmental dilemma for the city specifically and for the country as a whole. Durban is seen as a key area for growth in post-apartheid South Africa: it has the busiest harbour on the continent; it is a regional hub of the chemical industry and a major motor and metal manufacturer centre; and Durban's population continues to grow at some 4% per annum.

In recognition of the potential for growth and the likelihood that growth will adversely affect the communities within the industrial basin, the government in 2000, at a national Cabinet level, proposed the implementation of the "Multipoint Plan" (MPP). This plan was intended to better understand the state of pollution in the area, to develop a system to monitor fluctuations in pollution levels, understand and restructure the policy framework on the environment, to characterise the health of the population in these areas, and to determine the extent to which pollution adversely impacts on health of the community members.

The Centre for Occupational and Environmental Health at the University of KwaZulu-Natal and the Department of Environmental Health Sciences at the University of Michigan were awarded the tender to conduct a health risk assessment and an epidemiological study in Durban. We proposed an epidemiological study on about 400 school children using a population-based sample of invited participants.

The two broad overall objectives of this study were:

1. To determine the health status of the Durban South residents, with specific reference to respiratory health outcomes and other chronic diseases, and to determine the relationship between environmental pollution, these health outcomes, and the quality of life within this community, particularly among susceptible populations ("the epidemiological study").
2. To describe the range of ambient exposures and to assess the potential risks posed by such exposures to the health of the community in the Durban South ("the health risk assessment").

These two objectives, to some extent, constituted two distinct projects which were necessarily closely integrated. The underlying approach to the study was to have a particular focus on respiratory health for a variety of reasons, including well established and validated methodologies for determining relationship between respiratory outcomes and air pollution, and the preponderance of diseases related to this organ system in low-middle class communities. Time and financial resources also dictated the selection of outcomes that could be considered, and for these reasons, chronic outcomes such as haematological disorders and cancers could not be studied adequately epidemiologically.

To achieve these objectives, four schools in south Durban (Assegai, Dirkie Uys, Nizam, Entuthukweni in Lamontville), and three schools in the north (Briardale, Ferndale, Ngazana) as comparison sites were selected on defined criteria as a source of representative participants for the health study, as well as being sites representative of community exposure. A systematic approach to the sampling of the study participants was undertaken to ensure that (1) a population-based sample was randomly selected to ensure some degree of generalisability of the results, and (2) that sensitive subpopulations (such as children with pre-existing asthma and the elderly) were present in sufficient numbers to allow for efficient analysis.

An equally systematic approach was undertaken in the ambient air monitoring to ensure (1) appropriate data to allow for the proper assessment of exposure for the epidemiological study, (2) adequate data to characterise exposures of the various communities to a variety of pollutants, and (3) specific data to allow the development of the health risk assessment.

Ambient and indoor exposure monitoring was conducted in south and north communities during the study period, May 2004 to February 2005. Additional sources of pollutant and meteorological data covering January 2004 to October 2005 were accessed. Pollutants monitored included sulphur dioxide (SO₂), carbon monoxide (CO), volatile organic compounds (VOCs), particulate matter in two size classes (PM₁₀ and

PM_{2.5}), ozone (O₃), metals, and semi-volatile organic compounds (SVOCs, including dioxins, furans, and polycyclic aromatic hydrocarbons). The health risk component of the study also utilized exposure data (nitrogen oxides, ozone, total reduced sulphur, particulate matter), and meteorological data (wind speed, wind direction, temperature, relative humidity, barometric pressure, and precipitation) from the eThekweni MultiPoint Plan monitoring sites (including Wentworth, Harbour, City Hall, Warwick, King Edward, Grosvener, Jacobs, Settlers, Alverston, Ganges, Southern Works, Prospecton, Illovo, Ispingo, Westville, Umhlanga and Turner) and stations managed by the Durban International Airport. Also accompanying the ambient exposure data are indoor pollution measurements in approximately 140 homes drawn from the seven communities (target of 20 homes of recruited pupils from each community), and biological monitoring of lead and manganese in blood drawn from over 400 of the children. Methods for participant selection are described elsewhere in the document. The indoor home environment was evaluated through an extensive walk through assessment and inspection, and monitoring for particulate matter (PM₁₀), bioaerosols, carbon monoxide, temperature, humidity, allergens, and VOCs.

The study consisted of four intensive phases of data collection. During each of these three week phases, air pollutant exposures were monitored either continuously or integrated over 24-hour periods simultaneously with bihourly assessment of the student participants while at school. The intensive phases (conducted in June, August and October/November 2004 and February 2005) were scheduled so as to ensure that seasonal variability could be assessed in the study. During these intensive phases air pollutants, SO₂ (UV detection method), PM₁₀ and PM_{2.5} (filter-based gravimetric methods) were measured using active monitoring techniques, and CO was monitored using a passive electrochemical method. During non-intensive periods, an every 6th day monitoring programme was instituted for PM and VOCs, and every 12th day for SVOCs. In addition, we utilized the monitoring conducted under the MultiPoint Plan, which utilised a variety of monitoring types, e.g., real time continuous active monitoring for the conventional pollutants and passive monitoring for VOCs.

Health assessments consisted of the administration of several standardised and previously validated interview instruments: a child interview, a child caregiver interview, head of household interview and adult interview. The 423 children who participated in the study additionally underwent baseline spirometric assessments, methacholine challenge testing, blood lead and manganese assessments, genetics profiling for asthma-related polymorphisms and skin prick testing allergen sensitization. Adults were also requested to participate in baseline spirometry and methacholine challenge. The intensive phase assessments consisted of bihourly measurements of lung function, including peak expiratory flow (PF) and forced expiratory volume in one second (FEV₁), using digital handheld peak flow meters. At each lung function assessment session (bihourly during the school day), the child also completed a symptoms and activity log for the preceding one and half hours.

Descriptive analysis of the exposure data revealed NO₂ concentrations lowest in Ferndale in the north (mean of 11 ppb), highest in the city centre and industrial areas (19 - 24 ppb), and lower at Southern Works and Wentworth in the south (12 - 14 ppb).

Average SO₂ concentrations varied widely; with low concentrations (1 - 3 ppb) at sites in the north; medium to high concentrations (6 - 10 ppb) at central and south-central sites and (12 - 20 ppb) in the south. The SO₂ spatial distribution reflects the location of emitting industries in the South Basin.

Average PM₁₀ concentrations measured using the continuous tapered element oscillating microbalance (TEOM) method were nearly identical, 38 – 39 µm m⁻³, at four sites representing both north and south communities, and slightly elevated, 46 µg m⁻³, at Ganges. The average filter-based concentrations at the seven school sites were in the same range, 41 – 57 µg m⁻³. Maximum 24-hr average concentrations approached or exceeded 150 µg m⁻³ at most sites. The highest concentrations were observed at Assegai (south), and Ngazana (north), two widely separated monitors. PM_{2.5} concentrations measured at three sites were nearly identical, 20 – 21 µm m⁻³, with maximum 24-hr concentrations ranging from 79 to 131 µg m⁻³. PM₁₀ and PM_{2.5} concentrations across the region were high relative to international norms and standards. While there is moderate correlation between PM_{2.5} and PM₁₀, the ratio between these pollutants is not constant and different emission sources contribute fine (PM_{2.5}) and coarse (PM_{2.5-10}) particles.

The highest CO concentrations were observed at Warwick, the traffic-oriented site. 24-hr levels at this site averaged 2.1 ppm (maximum of 10 ppm). Measurements at other sites were much lower, averaging 0.7 ppm at two of the northern sites, 0.4 and 0.5 ppm at two southern sites, and <0.3 ppm at the remaining sites.

O₃ levels at Alverston (~30 km inland) averaged 26 ± 13 ppb, compared to 15 ± 11 ppb at Wentworth (south). A moderate correlation was observed between the two sites ($R^2 = 0.56$).

Metals analysis for 13 metals on filter-based PM_{2.5} samples revealed higher levels of lead, copper and iron at Ferndale (north), higher levels of vanadium and manganese at Nizam (south) and higher levels of chromium and manganese at Wentworth (south). Lead levels were lower than monitored elsewhere in earlier time periods in eThekweni Municipality, reflecting the distance of the school sites from high traffic areas as well as the decreasing trend of airborne lead attributable to the phase-out of leaded petrol. Concentrations of VOCs tended to be highest at the Settlers and Warwick sites, near refineries and traffic, respectively. For example, benzene levels at these sites averaged 7 – 9 µg m⁻³, compared to noticeably lower levels (2 – 5 µg m⁻³) at the other sites. Toluene was detected at central and south sites with the highest concentrations (35 – 39 µg m⁻³) while northern sites had noticeably lower concentrations (7 – 8 µg m⁻³). Ethylbenzene was found at south and central sites with the highest concentrations (9 – 14 µg m⁻³) while northern sites had low concentrations (2 µg m⁻³). Xylene was detected at south and central sites as the highest concentrations (24 – 43 µg m⁻³) while the northern sites had the lowest concentrations (6 – 7 µg m⁻³).

Numerous SVOCs in both gaseous and particulate phases at three sites (Nizam, Wentworth and Ferndale), were detected and quantified. The major compounds detected included: polycyclic aromatic hydrocarbons (PAHs) (levels of benzo[a]pyrene, the most potent PAH, were generally low); chlorinated hydrocarbons, mostly in the vapor phase (including pesticides lindane, aldrin, chlordanes, and DDT); polychlorinated biphenyls (PCBs); polychlorinated dibenzodioxins (PCDDs, dioxins; 0.4 – 0.6 pg m⁻³); and polychlorinated dibenzofurans (PCDFs, furans (0.5 – 1.1 pg m⁻³).

The indoor study found that the indoor PM₁₀ concentrations were elevated compared to outdoor levels, and that outdoor sources appeared to contribute about two-thirds of indoor concentrations. The highest indoor concentrations of PM₁₀, CO and VOCs tended to occur in homes where paraffin stoves were used. Indoor fungal levels were relatively high (average of 1148 CFU/m³), but this resulted from the still slightly higher levels in ambient air, the high penetration of outdoor pollutants into the homes, and lack of particle filtration.

Of the 422 children in the randomly selected classrooms for which all children were considered eligible to participate (referred to as the “type A classes”), 87% completed a screening questionnaire, which made them eligible to participate in the further aspects of the study. Over 93% of these participated in the full study. Additionally, of the 93 students from “type B” classes who were invited to participate because they had screening questionnaire responses consistent with persistent asthma, 87% participated in the full study. A total of 1391 adult members of households of the children who completed screening questionnaires participated in the study.

Because the type A classes form a population-based sample and their findings are more generalisable, the report focuses on this sub-sample. Mean age was 10.6 years; 57% were female; 43% were African, 22% Coloured, 28% Indian, and 7% White; 56% had a caregiver with education of Standard 9 or less; 16% had annual household income of R10000 or less, 45% of R10000-R75000, and 40% of R75000 or more. Caregivers rated child's general health as excellent, very good, good, and fair or poor at respectively 22.5, 38.4, 30.0, and 9.1%. Lifetime health consultation prevalence was 21.7%. Defined as skin test sensitivity to at least one of seven common allergens (house dust mite, cockroach, cat, dog, mould mix, Cladosporium, grass mix), 35.7% of the children had atopy. Sensitization was most common to house dust mite (29.5%) with all the other allergen sensitivity prevalences at 7% or less. Caregivers reported that 14.7% of children had been diagnosed with asthma by a doctor, 10.9% with hayfever, and 4.0% with chronic bronchitis. Based on symptoms described by the caregiver, 31.5% of children had some grade of asthma, with 7.9% having mild persistent asthma, and 4.1% having moderate to severe persistent asthma. Objective lung function assessments using methocholine challenge testing were well correlated with these reported assessments, with 7.6 % having marked bronchial hyperreactivity (BHR) ($PC_{20} \leq 2$ mg/ml) and a further 17% having either probable or possible BHR. The covariate-adjusted prevalence of marked BHR varied substantially between the south (8.0%) and north (2.8%). Logistic regression models adjusting for age, gender, race, caregiver education, household income and the presence of smokers in the household, found that attending a school in the south was statistically significantly associated with increased risk for persistent asthma (odds ratio [OR] and 95% confidence interval of 1.82 (1.05, 3.14)) and marked BHR (OR 2.55 (1.03, 6.28)).

Generalized estimating equations (GEE) were used to examine associations between daily mean levels of ambient air pollutants and lagged measures of pulmonary function. These models were adjusted for age, gender, race, school, caregiver smokes, caregiver education, household income, and phase (season). The

overall body of GEE results provided strong evidence that adverse effects on pulmonary function related to preceding exposures to NO₂, NO, PM₁₀ and SO₂, were occurring among the children who participated in the study. Statistically significant lagged decrements in pulmonary function (i.e., increased intraday variability and/or lower nadir values of FEV1 and peak flow) associated with higher ambient concentrations were present for a substantial proportion of the regression models evaluated for each of the four pollutants. Moreover these significant associations were stronger and much more frequent among those children expected *a priori* to be more sensitive to the effects of ambient air pollutants (i.e., those with persistent asthma as well as those with either of two genetic polymorphisms, GSTM1 null and GSTP1 (A/A), associated in the previous scientific literature with decreased capacity to address oxidative stress and related increase reactivity to pollutant exposures).

Mean age of the 1391 adult participants was 43.0 years; 61% were female; 49% were African, 24% Indian, 21% Coloured, and 6% White; 61% had Standard 9 or less education; 23 % were current smokers and 4% ex-smokers; 31% had ever consumed alcohol. Adults self-rated general health as excellent, very good, good, and fair or poor at respectively 29, 25, 24, and 23%. Doctor-diagnosed illnesses and reported symptom prevalences of 10% or higher included arthritis at 17%, diabetes 10%, hypertension 20%, cough 11%, phlegm/sputum/mucus 16%, wheezing 18%, stuffy runny nose 31%, watery itchy eyes 29%, skin problems 13%, and clothes too large due to weight loss 15%. The covariate-adjusted prevalence of reported doctor-diagnosed asthma and tuberculosis (TB) were, respectively, 7.0% and 3.5%. These rates did not vary markedly across communities, but were marginally higher in the south. Logistic regression models, adjusted for age, gender, race, education, smoking status, history of hazardous occupation, and more than one AIDS-like symptom, showed statistically significant increased reported doctor diagnosed hayfever (OR 3.95 (2.17, 7.20)) among those residing in the south. Doctor-reported chronic bronchitis and wheezing with shortness of breath also were somewhat elevated (ORs, respectively of 1.81 (0.92, 3.58) and 1.73 (0.98, 3.06) among those residing in the South, as was hypertension (OR 1.51 (0.99, 2.32)).

In summary, we 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 and/or genetic polymorphisms associated with reduced ability to respond to oxidative stress. Moreover, attending primary school in south Durban, as compared to the north, was significantly associated with increased risk for persistent asthma and for marked airway hyperreactivity in covariate-adjusted regression models. For adults, residing in the south was significantly associated with hayfever, and marginally associated with chronic bronchitis, wheezing and shortness of breath, and hypertension.

This report supports many recommendations, which are summarized below. As noted below, the resolution of several concerns may require additional investigation. We recognize that this report was intended to answer many questions related to the significance of air pollutants to human health, and further that eThekweni Municipality invested significant resources into this study and other elements of the MultiPoint Plan. At the same time, it should be recognized that many more questions will arise, especially given the scope and complexity of the problem, than can be addressed in any single study. Recommendations to further study an issue should not impede actions aimed at reducing emissions, for example, where concentrations, risks, nuisance or other factors are of concern. Further, not all studies will be fruitful and feasible, for example, an epidemiological study examining the relationships of cancers to air pollutants is unwarranted for many reasons, e.g., the need for retrospective exposures (which are unavailable), the very large sample sizes required, and the difficulty in controlling the many factors that can confound results. Potential follow-up studies to this report, where appropriate, will further build the knowledge, understanding and capacity to address air pollutant problems in Durban.

Monitoring of conventional pollutants (PM₁₀, PM_{2.5}, NO₂, CO, O₃, SO₂ and Pb) should be continued and potentially expanded in order to: better determine compliance with standards, guidelines and targets; document the effect of emission controls; trend the performance of air pollutant management; and aid forecasting and dispersion modeling. A number of recommendations are made to better identify the sources of emissions, especially PM, which includes local and regional sources (e.g., traffic, industry, veld and biomass burning, entrained dust), and to better map O₃ levels. With respect to emission controls on sources of conventional pollutants, emission reductions from both new and old sources are necessary to attain ambient air quality guidelines and standards, and to accommodate further development and traffic. Strategies and timeframes for attaining compliance with standards, guidelines and targets should be developed.

With regard to toxics, a number of pollutants are designated contaminants of potential concern (COPCs) that warrant concern due to their concentrations and potency that together may cause potentially significant cancer and non-cancer health risks. Recommendations to address these COPCs include the establishment of an enhanced VOC monitoring network, monitoring of wider set of trace metals and monitoring in the Jacobs area, and VOC controls on industrial and vehicular sources. Elevated levels of semivolatile organic compounds (SVOCs) like PCDDs (dioxins) and PCDFs (furans) indicate a need to identify and control local emission sources. Airborne concentrations of SVOCs compounds are associated with some of the larger risks identified in this study, but these risks need to be verified, especially since most of the exposure and risk from these compounds are likely to occur via food consumption. An aggressive schedule to reduce TRS and H₂S emissions in the DSIB should be implemented given that odours are widely perceived as objectionable and the exceedence of concentration guidelines. Finally, we suggest a need for a toxics emission inventory.

The assessment of residential and school environments shows that indoor combustion sources, particularly paraffin stoves, create excessive levels of CO, PM and VOCs, and the potential for serious adverse health effects. The use of paraffin (and similar fuels) in unvented indoor applications should be discouraged and ideally phased out. Also, means to increase awareness and to improve the housing conditions would help to reduce exposures to indoor pollutants, including PM and bioaerosols.

Health based recommendations emanating from the findings of the study provide substantial support for rigorous environmental monitoring and control of ambient pollutants as the primary means of reducing morbidity, particularly arising from adverse respiratory outcomes among sensitive subpopulations, including children with persistent asthma. Asthma education and awareness could prove to be a useful adjunct to pollution control.

NOTE:

For reporting purposes, the results from the environmental monitoring/risk assessment study and the health study are presented in two sections. Section I provides the details of the exposure assessment and ambient air monitoring. Section II provides the report of the epidemiological study. Each section utilizes a variety of complex approaches and present considerable data. The separation is aimed at clarifying the methods and results used in each approach.

This report used screening analyses for the purpose of examining emissions, exposures and risks associated with dioxin and furans. The screening level analysis utilised a number of conservative assumptions, in part due to large uncertainties and data gaps. In particular, exposure estimates would be improved by:

- Better spatial/temporal coverage of concentrations
- Information describing current (and multimedia) exposures of bioaccumulative pollutants, e.g., dioxin measurements in milk
- Obtaining additional demographic and health data on the surrounding community, including health monitoring and surveillance activities
- Obtaining emissions data for toxics
- Obtaining measurements of hexavalent chromium
- Obtaining measurements of additional pollutants, e.g., aldehydes, mercury, cadmium, very volatile VOCs (formaldehyde, 1,3-butadiene)
- Uncertainties in the toxicity characterizations have been discussed for several pollutants, e.g., naphthalene and dioxin. These uncertainties can be large, e.g., an order of magnitude.

Risk estimates will change with additional information. We expect that risks will increase with consideration of additional pollutants (e.g., 1,3-butadiene) and consideration of spatial gradients (e.g., traffic-impacted populations). On the other hand, risks may decrease if exposures are overestimated.

We believe that this report does highlight priorities for risk management as the contaminants of principal concern. Due to inherent uncertainties and limitations of the health risk assessment, we anticipate that the communities in Durban may desire additional reassurance or confirmation of results in this report, particularly where risks are stated as being “minimal,” “low ” or “*de minimis*.” We believe that the lower risks stated for these pollutants are based on the best available information in a technical study designed to characterise risks, and that the results obtained are credible and applicable to the communities in the DSIB (and in North Durban). Thus, we urge that public health managers focus on the highlighted risks.

11.10 Summary

Health risk assessments have been performed in many contexts and much has been learned. As discussed in the previous section, assessments have a number of limitations. The interpretation of the calculated risks depends on a number of factors, including the reliability and representativeness of the exposure information, the weight of the evidence supporting the dose-response characterisation, the number of individuals potentially exposed, the magnitude of the risk, and the certainty and relevance of the supporting information. Because of these factors, there are generally not presumptive maximum limits in the quantitative outcomes of a health risk assessment. Instead, results should be interpreted with respect to risk guidelines, risk characterisations and weight-of-evidence analyses should be considered, and all of this information should be utilised in the assessment’s conclusions and subsequent mitigation/management decisions.

This risk assessment and additional supporting information in Section I identify a number of toxic contaminants of potential concern (COPCs) that warrant attention due to health risks, including cancer and non-cancer effects. Some of the largest cancer risks are posed by the following pollutants:

- VOCs, especially benzene. Concentrations were high at multiple sites in the DSIB as based on calculated risks and by comparison to levels measured in other urban/industrial areas. Additionally, several other VOCs not measured in this study are also likely to be carcinogenic COPCs (as has been seen in other cities), e.g., 1,3-butadiene and formaldehyde. Major sources of these VOCs include point and fugitive releases from petroleum refining, storage and distribution facilities, and vehicle emissions.
- Semivolatile compounds, including selected dioxins, furans, PAHs and naphthalene. These compounds are present at elevated levels based on risk calculations and compared to other urban areas, and these pollutants also represent COPCs in Durban. Sources of these compounds, suggested by the monitored concentrations and spatial gradients, likely include regional sources, e.g., biomass/waste burning, as well as large “point” sources, e.g., incinerators.

- Metals including chromium, nickel, lead, and manganese.
- Pesticides are not considered to pose significant inhalation risks at the ambient concentrations observed. However, the monitoring indicates that use of very hazardous and banned materials, e.g., lindane, is occurring locally.

A key finding is that the estimated lifetime cancer risk from the inhalation of all carcinogens measured at the three sites (where most toxic monitoring occurred) ranged from 1.8 E-4 (18 per 100,000 at Ferndale) to 2.5 E-4 (25 per 100,000 at Wentworth), well above guideline levels. Importantly, these are not worst-case estimates for several reasons: monitoring locations do not necessarily represent hot-spots; exposure estimates used averages, not upper confidence level estimates, and; inhalation unit risk factors used mid-point values.

A second key result is that a small number of pollutants contribute the bulk (~90%) of the total estimated cancer risks, specifically, PCDFs, PCDDs, benzene, and naphthalene, with additional but smaller contributions from nickel, ethylbenzene, chromium, PCBs and styrene.

A third result is that chronic non-cancer risks for most of the measured toxic compounds measured fall below the significance level (e.g., hazard quotient of 0.25). The most important air toxics for chronic non-cancer effects include manganese, benzene, p,m-xylene, phenanthrene, and naphthalene. Lead also remains a COPC since a higher than desirable fraction of children has blood lead levels that exceed guidelines.

For the most part, we did not examine acute non-cancer health effects (given the study design). An exception, however, is the total reduced sulfur/hydrogen sulfide. This pollutant clearly exceeds standards and guidelines, and poses a nuisance to a very large fraction of individuals in the DSIB.

With regard to conventional pollutants, the evaluation in Part II of this report discusses the epidemiological evidence for acute respiratory effects. Here, we note that health-based standards and guidelines are exceeded for multiple pollutants:

- Particulate matter. Both PM₁₀ and PM_{2.5} frequently exceeds short- and long-term standards and guidelines at most or all sites in eThekweni, including the 5 sites operated by the Metro and the 7 school-based sites established for this study. PM is associated with a range of adverse acute and chronic health effects including respiratory hospital admissions, bronchodilator use, cough and lower respiratory symptoms, changes in peak expiratory flow, cardiovascular stress, and mortality.
- SO₂. While concentrations have come down from historical highs, SO₂ continues to frequently exceed short-term standards and guidelines in the DSIB area. SO₂ peaks of 1-hr duration can adversely affect lung function (e.g., reduce FEV1, increase airway resistance, increase likelihood of wheezing and shortness of breath). SO₂ exposure over a 24-hour period has been shown to affect mortality (total, cardiovascular and respiratory) and hospital emergency admissions for total respiratory causes and chronic obstructive pulmonary disease.
- CO at traffic-impacted sites exceeds short-term standards.
- NO₂ at traffic-impacted sites exceed short- and long-term standards.
- O₃ is presently at levels below standards and guidelines, based on the limited monitoring data available (Wentworth in the DSIB), but O₃ levels occasionally exceed guidelines and standards at the inland site, Alverson.

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Ambient pollution and respiratory outcomes among schoolchildren in Durban, South Africa

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Objective. To examine associations between ambient air pollutants and respiratory outcomes among schoolchildren in Durban, South Africa.

Methods. Primary schools from within each of seven communities in two regions of Durban (the highly industrialised south compared with the non-industrial north) were selected. Children from randomly selected grade 4 classrooms were invited to participate. Standardised interviews, spirometry, methacholine challenge testing and skin-prick testing were conducted. Particulate matter (PM), sulphur dioxide (SO₂) and carbon monoxide were monitored at each school, while nitrogen oxides (NO_x) and other pollutants were monitored at other sites.

Results. SO₂ was significantly higher in the south than in the north, while PM concentrations were similar across the city. The prevalence of symptoms consistent with asthma of any severity was 32.1%. Covariate-adjusted prevalences were higher among children from schools in the south than among those from the north for persistent asthma (12.2% v. 9.6 %) and for marked airway hyperreactivity (AHR) (8.1% v. 2.8%), while SO₂ resulted in a twofold increased risk of marked AHR (95% confidence interval 0.98 - 4.66; *p*=0.056).

Conclusions. Schoolchildren from industrially exposed communities experienced higher covariate-adjusted prevalences of persistent asthma and marked AHR than children from communities distant from industrial sources. Our findings are strongly suggestive of industrial pollution-related adverse respiratory health effects among these children.

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A substantial body of literature provides evidence for the adverse effects of ambient pollution on respiratory health, particularly among children with pre-existing respiratory disease.^[1] Increased ambient air pollution levels have been reported to precipitate symptoms of asthma.^[2] Against the reported background of a worldwide increasing prevalence of childhood asthma,^[3] environmental pollution has received scrutiny as a cause of increased respiratory morbidity.

The study area, south Durban, South Africa, is recognised as one of the most highly industrialised and heavily polluted areas in southern Africa.^[4] Residential and industrial areas mingle in the region, which has a population of over 400 000.^[5] A previous study reported a high prevalence of asthma among children at a primary school in south Durban, with pollutants having a significant association with increased respiratory symptoms and decrements of pulmonary function in asthmatic children.^[6] However, the generalisability of the findings is uncertain, given the closeness of the school to large sources of pollution. Further description of adverse respiratory health among children in communities with varying proximity to industrial pollution was necessary.

This study investigated the possible relationships between ambient pollution and respiratory health among schoolchildren in the metropolitan area of Durban, comparing covariate-adjusted prevalences of chronic respiratory symptoms and conditions of those residing in industrialised (south) and non-industrialised (north) areas.

Methods

Selection of the study communities and schools

Four communities in south Durban in close proximity to industrial areas (Merebank, Wentworth/Austerville, Bluff and Lamontville) and three communities in north Durban (Newlands East, Newlands

West and KwaMashu) were selected. This study design permitted description of health outcomes among the various communities exposed to industrial pollution in the south. The northern communities were selected as comparison areas based on their similar socio-economic and race/ethnicity profiles to those of the southern communities, together with their greater distance from major industry and expected lower exposure to industrial emissions. The design increased the ability to separate effects of race/ethnicity and socio-economic position from effects of ambient air pollutant concentrations, with regard to both short-term exacerbations and prevalence of specific respiratory diagnoses and symptoms.

All primary schools in the selected communities were assessed by location, geography and potential sources of exposure. Only schools where bussing of students from surrounding communities was minimal (<15%) were eligible, to ensure that exposure measurements at the schools were reasonably representative of residential exposures of the study sample. One school was randomly chosen from each community. None was selected on the basis of the health status of children at the school.

Student recruitment

At each of the seven schools, all children in one or two randomly selected grade 4 classrooms were invited to participate in the study ('type A' classrooms). In addition, all students from all other classrooms ('type B' classrooms) in grades 3 - 6 with known or probable persistent asthma, based on parent/caregiver responses on a screening questionnaire, were invited. Inclusion of these additional students augmented statistical power to address the hypothesis that students with persistent asthma are at increased risk for adverse health effects associated with exposures to ambient air pollutants. The questionnaire was adapted from an instrument used in a study of asthma among children in Detroit, USA, and had been used previously in south Durban.^[6]

The legal guardians of the children who participated gave written informed consent, and the children participated voluntarily and had the right to withdraw at any stage. Ethical approval was obtained from the Institutional Review Board of the University of Michigan and the Ethics Committee of the University of KwaZulu-Natal, Durban. The study was conducted over an 8-month period, with continuous environmental monitoring during this time.

Child and caregiver interviews

Survey instruments were written in English, then translated and back-translated into Afrikaans and isiZulu. Interviews were administered in the respondent's language of choice. Survey instruments utilised standardised and validated questions addressing presence and severity of respiratory and other relevant symptoms.^[7] Participating children and caregivers were interviewed at school and at home, respectively. Caregiver responses categorised the children as having moderate to severe persistent, mild persistent, mild intermittent or no asthma based on the US National Asthma Education and Prevention Program (NAEPP) guidelines.^[8] Information about the child's household, residential history, use of biomass fuels at home, smoking in the home and household income was obtained from an interview of the head of the household.

Chronic respiratory symptoms were defined on the basis of responses from the caregiver interview. Symptoms included chronic cough ('yes' to 'usual cough on most days for 3 consecutive months or more during the year'); chronic phlegm ('yes' to 'bringing up phlegm on most days for as much as 3 months each year'); chronic bronchitis ('periods or episodes of (increased) cough and phlegm lasting for 3 weeks or more each year'); wheeze ('yes' to 'chest sounding wheezy or whistling on most days and nights'); and wheezing with shortness of breath ('yes' to 'ever having an attack of wheezing that has made the child feel short of breath'). 'Doctor-diagnosed' outcomes were based on the responses from the caregiver interview.

Pulmonary function assessments

Spirometric assessments and methacholine challenge tests were conducted by experienced respiratory technicians on all participants. American Thoracic Society guidelines for conducting spirometry were followed.^[9] Participants were instructed not to take any anti-asthmatic inhalants from 12 hours before the test, or oral asthma medications from 48 hours before the test, unless this was necessary (in which case testing was delayed appropriately). Participants with an obstructive pattern at baseline (the ratio of forced expiratory volume in one second/forced vital capacity (FEV₁/FVC) <0.75) were administered an inhaled bronchodilator and had testing repeated. Those without a baseline obstructive pattern underwent methacholine challenge testing according to an abbreviated protocol.^[10] Precautionary measures and medical personnel were available at all times during the tests. Students were assessed during school hours. Results of the methacholine challenge tests were classified, based on PC₂₀ (dose of methacholine causing a 20% fall in baseline FEV₁), as follows: marked airway hyperreactivity (AHR): PC₂₀ ≤4 mg/ml; probable AHR: PC₂₀ ranging from 4 to 8 mg/ml; possible AHR: PC₂₀ ranging from >8 to 16 mg/ml; none: PC₂₀ >16 mg/ml.

Assessment of allergic status

Students underwent skin-prick testing for allergic sensitisation at school on a different day from methacholine challenge testing. Health personnel assessed each participant immediately before skin testing, and were equipped to respond in the unlikely event that the child had a severe reaction to a skin test. Antigens tested included mixed cockroach, mixed dust mite, mould mix (*Aspergillus*, *Cladosporium* and *Penicillium*), cat, dog, mouse, rat and mixed grasses, plus histamine as a positive control and saline

as a negative control. Participants were told to stop taking any antihistamines and any other reactive medication (H₂ antagonists, tricyclic antidepressants, corticosteroids, etc.) at least 24–48 hours before the test. Test solutions were applied to the volar surface of the forearm and read 15–20 minutes later. A positive test was defined as a wheal ≥2 mm greater than the saline control.

Environmental monitoring

Ambient pollutants (nitrogen dioxide (NO₂), nitric oxide (NO), sulphur dioxide (SO₂), and particulate matter ≤10 μm (PM₁₀) and ≤2.5 μm (PM_{2.5}) in aerodynamic diameter) were measured throughout the 8-month study period. Monitoring sites were established at all schools to monitor SO₂ and PM₁₀. Continuous data collected at eight Durban municipal-operated sites were utilised to estimate exposures to NO and NO₂. The environmental monitoring strategy is detailed elsewhere.^[11]

Statistical analysis

Analyses were performed using Statistical Analysis Software (SAS) version 8.1. The primary independent variable of interest was school location (i.e. south versus north), and the primary outcome variables of interest were doctor-diagnosed respiratory diseases (e.g. asthma, chronic bronchitis), symptom-defined respiratory conditions (e.g. persistent asthma, chronic bronchitis, wheezing with shortness of breath) and AHR. Potential covariates/confounding variables examined included age, gender, race/ethnicity (as reported by the caregiver), previous history of respiratory disease, education level of primary caregiver, smoker in the household, atopy status and annual household income.

All prevalence outcomes are restricted to students in the randomly selected grade 4 classrooms (type A). Given the high participation rates (93.4%), the responses can be considered true population-based estimates, i.e. the prevalence rates obtained for the various outcomes, and therefore generalisable to this school pupil population. To increase statistical power, the regression models examining associations between pollutant levels and daily measures of pulmonary function included all participating children (type A and type B).

Covariate-adjusted prevalences of health outcomes by school and covariate-adjusted logistic regression models of associations of school location with health outcomes were constructed.

Asthma severity was categorised as a binary variable: probable (or known) persistent asthma (including mild and moderate to severe persistent cases) v. no asthma or mild intermittent asthma.

Atopy was defined as a positive reaction to the skin-prick test greater than that of the response to the histamine for any one of the tested allergens.

Results

Exposure evaluation

Annual average pollutant levels differed significantly across the study region (Table 1). SO₂ levels were much higher in the south than in the north. PM₁₀ levels showed much less geographical variability. NO and NO₂ showed significant differences between north and south Durban, reflecting local sources.

Participation rates

Of the 422 students in the type A classes, 366 (86.7%) completed the screening questionnaire. Of these, 341 (93.2%) participated in the full study. The non-participants were similar to the participants in respect of age, gender, and number of adults living in their household, but were more likely to speak English at home. From the type B classes, 451 completed the questionnaire and 93 known or probable persistent asthmatics were identified based on their responses. Of these identified asthmatics, 81 (87.1%) participated in the full study.

Table 1. School and geographical averages for the pollutants measured in the study

	Schools in the south				Average schools		Schools in the north		
	Nizam	Enthu	Assegai	Dirkie Uys	South	North	Briardale	Ferndale	Ngazana
PM₁₀ (µg/m³)									
Mean (±SD)	50.1 (±25.2)	51.6 (±26.3)	57.9 (±34.6)	45.6 (±24.2)	51.3 (±28.0)	48.5 (±35.4)	40.9 (±23.8)	45.3 (±30.0)	59.0 (±45.9)
Range	14.0 - 179.4	9.6 - 173.8	1.2 - 208.0	1.4 - 170.6	1.2 - 208.0	0.7 - 266.6	1.9 - 133.8	4.5 - 178.7	0.7 - 266.6
PM_{2.5} (µg/m³)									
Mean (±SD)	-	-	-	-	20.7 (±15)	20.0 (±23.6)	-	-	-
Range	-	-	-	-	3.6 - 78.6	4.4 - 131.4	-	-	-
SO₂ (ppb)*									
Mean (±SD)	8.3 (±8.4)	6.7 (±8.0)	11.9 (±10.1)	7.0 (±5.5)	8.7 (±8.2)	1.9 (±2.0)	1.6 (±2.6)	2.5 (±2.2)	1.2 (±1.2)
Range	0.01 - 61.9	0.05 - 46.8	0.43 - 73.4	0.17 - 32.2	0 - 73.4	0 - 16.8	0 - 24.1	0 - 16.3	0 - 6.0
NO₂ (ppb)[†]									
Mean (±SD)	-	-	-	-	17.2 (±8.8)	10.9 (±6.2)	-	-	-
Range	-	-	-	-	3.7 (±63.8)	0 - 47.5	-	-	-
NO (ppb)*,†									
Mean (±SD)	-	-	-	-	40.9 (±30.5)	22.1 (±22.2)	-	-	-
Range	-	-	-	-	3.2 - 192.2	0 - 115.7	-	-	-

PM₁₀ and PM_{2.5} = particulate matter ≤10 µm and ≤2.5 µm in aerodynamic diameter, respectively; SD = standard deviation; SO₂ = sulphur dioxide; ppb = parts per billion;

NO₂ = nitrogen dioxide; NO = nitric oxide.

*p<0.05 (t-test comparing north v. south).

[†]These pollutants were monitored only at single sites in the north and south respectively, and the table reflects data from the municipal monitoring sites.

Demographic data

The mean age of the students (± standard deviation) was 10.5 (±0.9) years, with 58.2% being female (Table 2). Most (40.9%) of the participants were black. English was reported as being the first language by 50.3% of the participants.

Only 44.0% of caregivers had matriculated from high school. A wide wage gap existed within the study population. Approximately 37% of households had annual incomes exceeding US\$9 375 (US\$1=R8), in contrast to 19.5% with incomes under US\$1 250 (Table 2).

Reported symptoms and doctor-diagnosed diseases

Prevalences of doctor-diagnosed asthma among schoolchildren were higher in the schools in the north (16.5%) than in the south (13.0%) (Table 3). This contrasts with asthma severity reporting. Students attending schools in the south were more likely to report symptoms consistent with moderate to severe persistent asthma (5.3% v. 2.9%), persistent asthma of any severity (15.3% v. 9.1%), and any asthma (35.4% v. 29.1%) than those at schools in the north (Table 4).

Among children diagnosed with asthma (n=45), the reported age of onset for a substantial proportion (39.8%) was before the age of 2, while 25.3% were diagnosed after the age of 5. Of these diagnosed asthmatics, 62.9% were reported to have current asthma. The interpretation of school-stratified data must be done with caution, because of the small numbers of children with asthma in the various schools. Of note is the relatively high prevalence of doctor-diagnosed chronic bronchitis (4.1%) in this young population. The prevalence of symptom-based chronic bronchitis was considerably lower, 1.5% of caregivers reporting their child to have symptoms of both chronic cough and chronic phlegm (Table 3). This contradictory finding may reflect a poor understanding of the diagnosis made by the doctor.

Over 24% of children were reported to have wheezing symptoms, 41.5% reporting attacks of wheezing with dyspnoea. Children in the south had their first attack of wheezing at an average age of 3.5 years, compared with 6.8 years in the north. However, more children in the north than in the south were likely to have repeated episodes of wheezing, and more had experienced attacks requiring treatment (Table 3).

Lung function outcomes

Age-, height- and gender-adjusted mean FEV₁ was not statistically different between the north and the south (data not shown).

Differences in respiratory health between children at the northern and southern schools were highlighted by AHR testing: an 11.9% prevalence of marked AHR was found in the south, compared with 4.1% in the north. Over 32% of the children in the south presented with some degree of AHR (possible to marked grades), compared with 21.9% in the north (Table 4).

Allergy and atopy

Prevalences of atopy in north (36.1%) and south Durban (36.7%) were very similar, although the prevalence at one school (Enthuthukweni Primary in the south) was much higher (54.6%) than at the other schools. The overall prevalence of sensitisation to house-dust mite allergen was very high (31.0%). Enthuthukweni and Briardale (in the north) showed much higher prevalences of sensitisation than the other schools, particularly for house-dust mite and cockroach allergens (Table 4). Among children reporting hay fever, 71.1% had atopy. There was no association between the doctor-diagnosed outcomes or AHR and atopy: 48.7% of doctor-diagnosed asthmatics were atopic, compared with 42.9% of those with marked AHR (data not shown).

Table 2. Demographic variables for participating children (type A classrooms)

	Grand total	Schools in the south				Average schools		Schools in the north		
		Nizam	Enthu	Assegai	Dirkie Uys	South	North	Briardale	Ferndale	Ngazana
Age (years), mean (\pm SD)	10.5 (\pm 0.9)	10.5 (\pm 0.7)	10.9 (\pm 1.9)	10.6 (\pm 0.5)	10.3 (\pm 0.6)	10.6	10.4	10.3 (\pm 0.5)	10.5 (\pm 0.6)	10.5 (\pm 0.9)
Females, %	58.2	50.0	63.2	60.5	57.1	57.4	58.8	51.9	55.9	69.8
Race/ethnicity, %										
Black	40.9	15.2	100.0	30.6	-	32.2	48.7	12.0	38.5	100.0
Coloured	22.4	-	-	66.7	-	21.0	23.7	-	61.5	-
Indian	25.6	84.8	-	2.8	-	23.4	27.5	88.0	-	-
White	11.1	-	-	-	100.0	23.5	-	-	-	-
Language, %										
English	50.3	85.7	2.7	67.7	3.7	45.4	54.6	88.9	69.6	-
Zulu	35.0	14.3	81.1	32.4	-	29.0	40.3	11.1	26.1	88.9
Xhosa	3.4	-	16.2	-	-	3.0	3.8	-	2.9	8.9
Afrikaans	11.4	-	-	-	96.3	22.6	1.2	-	1.5	2.2
Caregiver education, %										
\leq Grade 11	38.4	40.0	46.7	40.6	40.0	41.4	35.6	39.5	30.2	38.5
Matric	44.0	35.0	33.3	31.3	60.0	39.4	48.3	51.2	49.1	44.2
Some tertiary	17.6	25.0	20.0	28.1	-	19.2	16.1	9.3	20.8	17.3
Annual household income, %										
\leq R10 000	17.9	18.9	34.5	19.1	-	17.4	18.4	14.3	26.2	12.8
R10 000 - R30 000	19.4	18.9	27.6	23.8	10.0	20.0	18.8	17.1	16.7	23.4
R30 001 - R75 000	21.8	29.7	13.8	14.3	10.0	17.3	26.0	22.9	23.8	31.9
\geq R75 001	40.8	32.4	24.1	42.9	80.0	45.3	36.8	45.7	33.3	31.9
Body mass index, mean (\pm SD)	18.3 (\pm 3.7)	16.7 (\pm 4.5)	18.0 (\pm 3.1)	19.2 (\pm 3.3)	19.2 (\pm 3.6)	18.3	18.5	18.1 (\pm 3.8)	18.6 (\pm 3.6)	18.7 (\pm 3.0)

Covariate-adjusted predicted prevalences and risk of respiratory health outcomes

The adjusted prevalences of respiratory health outcomes show substantial variability across schools, often exceeding the variability when comparing the average across schools in the south and north regions (Table 5). All the doctor-diagnosed conditions and chronic respiratory symptoms were more common in the north than in the south, although these differences were not statistically significant. However, the prevalence of persistent asthma was higher among schools in the south (12.2% v. 9.6%), as was the objective measure of marked AHR (8.1% in the south compared with 2.8% in the north).

The adjusted odds ratios (AORs) from logistic regression models contrasting students attending schools in north and south Durban were elevated ($p < 0.05$) for children in the south (from types A and B classrooms) for 5 of the 13 outcomes investigated: doctor-diagnosed chronic bronchitis (AOR 3.5, 95% confidence interval (CI) 1.6 - 7.7) (not shown in tables), as well as bronchitis by symptom definitions; watery/itchy eyes; wheezing with shortness of breath; and marked AHR (Table 6). In addition, marked AHR was associated with SO₂ exposure. While several of the other outcomes showed an increased risk for both PM₁₀ and SO₂ (i.e. ORs > 1), these were not statistically significant (Table 6).

Discussion and conclusions

This study compared Durban children exposed to industrial pollution with those who had less exposure, and documented prevalences of symptom-defined asthma and nonspecific AHR

that are at the higher end of the ranges described in the published literature. Among the population-based sample, 32.1% presented with some grade of asthma, 12.0% with persistent asthma, of which 4.0% was marked to moderate, and 7.8% with marked AHR. The prevalence of doctor-diagnosed asthma was much lower (14.8%). These prevalences fall within the range found in previous reports of paediatric populations in other countries that are likely to be at relatively high risk or in populations with lower socio-economic status, and that use similar and well-validated instruments such as those from the International Study of Allergy and Asthma in Children (ISAAC) projects. South American studies show asthma prevalences from 3.9% to 33.1%,^[12] while prevalences among children from lower socio-economic communities in the USA ranged from 6.2% to 15.2%.^[13]

Schoolchildren aged 7 - 8 years from Cape Town, South Africa, had a relatively high prevalence of wheeze in the past 12 months (26.8%) and asthma diagnosis (10.8%), as reported by parents.^[14] In south Durban children aged under 17 years, parents reported that 16.3% had experienced attacks of shortness of breath with wheeze during the last 12 months, and that 10% had ever been diagnosed with asthma.^[15] The ISAAC-based prevalences show wide variation between countries in Africa, prevalences of wheeze symptoms ranging from 4.0% to 21.5%.^[7] Our rates of 14.8% for doctor-diagnosed asthma and 24.5% for ever wheezing reported by the caregivers are within the range of other South African and international studies.

A striking finding in the present study is the substantial differences between respiratory health indicators among children in

Table 3. Respiratory and related outcomes for participating children as reported by caregivers

	Grand total	Schools in the south				Average schools		Schools in the north		
		Nizam	Enthu	Assegai	Dirkie Uys	South	North	Briardale	Ferndale	Ngazana
Range of Ns across the variables presented	246 - 313	40 - 48	18 - 32	25 - 36	12 - 19			41 - 61	44 - 63	46 - 54
Doctor-diagnosed asthma, %	14.8	12.8	15.6	5.6	21.1	13.0	16.5	18.6	23.3	5.6
Among children diagnosed with asthma (N=45)										
Age of child (years) when first diagnosed with asthma, %										
<2	39.8	-	-	100.0	-	31.5	47.2	18.2	55.6	66.7
2 - 5	35.0	100.0	50.0	-	33.3	43.6	27.1	27.3	22.2	33.3
>5	25.3	-	50.0	-	66.7	24.9	25.6	54.6	22.2	-
Current asthma, %	62.9	60.0	100.0	50.0	75.0	67.8	58.6	75.0	38.9	66.7
Ever treated for asthma, %	89.3	100.0	80.0	100.0	75.0	90.4	88.3	91.7	76.5	100.0
Doctor-diagnosed chronic bronchitis, %	4.1	6.8	-	-	10.5	4.3	4.0	8.5	3.4	-
Doctor-diagnosed hayfever, %	11.5	11.1	3.1	2.9	26.3	10.6	12.4	18.6	17.0	-
Chronic cough, %	5.2	6.3	6.3	2.8	5.3	4.9	5.5	8.2	4.8	3.7
Chronic phlegm, %	3.0	-	-	-	10.5	2.5	3.5	1.6	6.4	1.9
Symptom-based chronic bronchitis, %	1.5	-	-	-	5.3	1.2	1.8	-	3.2	1.9
Ever had ear infection, %	37.1	38.1	26.3	22.6	62.5	36.8	37.4	45.5	30.2	38.2
Ever sound wheezy, %	24.5	19.2	56.3	2.9	31.6	23.8	25.2	25.4	23.0	27.8
Among those who ever sound wheezy (N=78)										
Ever had an attack of wheezing with SOB, %	41.5	55.6	17.6	-	100.0	41.5	47.9	64.3	46.1	33.3
Stuffy, itchy, runny nose during past 12 months, %	41.5	42.6	28.1	28.6	68.4	41.5	46.4	59.3	41.7	38.9
Watery, itchy eyes during past 12 months, %	28.1	36.2	18.8	22.9	33.3	28.1	30.1	33.9	28.8	27.8
Among those reporting attack of wheezing with SOB (N=32)										
Age (years) at first wheezing attack, mean (±SD)	6.5 (3.5)	4.8 (2.7)	3.6 (3.7)	-	8.0 (1.6)	3.5	6.8	8.4 (4.1)	7.6 (2.8)	4.2 (2.3)
≥2 such episodes, %	57.9	60.0	100.0	-	100.0	57.9	73.3	77.8	80.0	60.0
Required treatment for attacks, %	50.6	100.0	66.7	-	50.0	50.6	96.5	88.9	100.0	100.0
Breathing normal between attacks, %	47.3	20.0	100.0	-	100.0	47.3	38.7	22.2	66.7	20.0

SOB = shortness of breath.

communities affected by ambient industrial pollution compared with those without such exposures. While the prevalences of symptoms and AHR varied across the schools, sometimes with higher prevalences in the north (for example, a 20.5% prevalence of probable AIHR in the northern school Ferndale), AORs comparing children in the south and north of Durban were 1.33 and 3.53 for doctor-diagnosed asthma and

chronic bronchitis, respectively. The AORs of having symptoms defined as persistent asthma or AHR were 1.14 and 2.49, respectively. These results imply a greater risk for the children exposed to ambient pollution. There was a suggestion of a twofold SO₂-associated increased risk for marked AHR, but this was not statistically significant ($p=0.056$; 95% CI 0.98 - 4.66).

Similar intra-city findings have been

reported in several studies in the USA^[16] and elsewhere.^[17] These differences have largely been explained by socio-economic and ethnic differences between communities. In the present study, comparison communities had similar socio-economic profiles, but differed in ambient pollutant exposure.

There were several limitations to this study. To ensure community representivity,

Table 4. Smoking, atopy and asthma measures

	Grand total	Schools in the south				Average schools		Schools in the north		
		Nizam	Enthu	Assegai	Dirkie Uys	South	North	Briardale	Ferndale	Ngazana
Any smoker in the household (N=287), %	26.4	19.4	12.5	20.9	29.0	20.8	31.3	15.2	26.8	53.9
Allergic status, skin-prick test (N=312), %										
House-dust mite	31.0	16.0	54.6	38.5	25.8	32.5	29.6	47.5	28.6	12.2
Cockroach	15.0	4.0	45.5	15.4	6.5	15.8	14.2	20.0	14.3	8.2
Cat	7.3	4.0	9.1	12.8	-	6.8	7.7	10.0	10.2	2.0
Dog	4.7	-	-	-	3.2	0.8	8.3	10.0	10.2	4.1
Mould	4.0	-	9.1	5.1	6.5	4.8	3.2	-	2.0	8.2
<i>Cladosporium</i>	2.2	-	9.1	-	-	1.7	2.6	-	2.0	6.1
Grass	0.7	-	-	-	-	-	1.4	-	2.0	2.0
Atopy*	36.4	20.0	54.6	43.6	32.3	36.7	36.1	47.5	32.7	28.6
AHR [†] (N=315), %										
Marked	7.8	13.0	14.3	5.6	17.4	11.9	4.1	2.6	6.8	2.2
Probable	8.4	13.0	7.1	8.3	-	7.4	9.2	-	20.5	4.4
Possible	10.6	8.7	7.1	16.7	17.4	13.0	8.6	13.2	4.6	8.9
None	73.2	65.2	71.4	69.4	65.2	67.7	78.1	84.2	68.2	84.4
Caregiver's report of asthma severity (N=301), %										
Moderate to severe	4.0	-	-	7.9	12.0	5.3	2.9	2.2	4.1	2.0
Mild persistent	8.0	-	11.1	13.2	16.0	10.0	6.2	8.9	4.1	6.0
Mild intermittent	20.1	20.0	22.2	13.2	28.0	20.1	20.0	17.8	26.5	14.0
None	67.9	80.0	66.7	65.8	44.0	64.6	71.0	71.1	65.3	78.0

AHR = airway hyperreactivity.

*Allergic to any of the listed allergens.

[†]Marked = PC₂₀ ≤4 mg/ml; Probable = PC₂₀ ranging from 4 to 8 mg/ml; Possible = PC₂₀ ranging from >8 to 16 mg/ml; None = PC₂₀ >16 mg/ml.

we had to select study participants from multiple communities. Resources limited us to randomly selecting a single school in each community. Although we have no reason to believe that the schools selected were not representative of the communities themselves, the generalisability of the findings must be treated with caution. Our study includes reporting symptoms from caregivers and child participants. We used instruments that have been well standardised, both internationally and in South Africa, to ensure comparison and limitation of bias. Despite the apparent differences in reporting of these symptoms, our findings are not due to over-reporting by residents living close to the polluting industries. Prevalences of respiratory outcomes reported by the caregivers were supported by the findings of objective lung function assessments, the methacholine challenge: 12.0% had some grade of persistent asthma, and 16.2% had either marked or probable AHR (Table 4). As an objective marker of airway disease, the overall rate of any grade of AHR found, a rate of 26.8%, is strikingly high, and at the high end of the range reported in other population samples in the international literature.¹¹⁷ Differing protocols, definitions and population selection across studies make such comparisons difficult. Population-based studies of children of a similar age show rates of positive responses to methacholine challenge tests (defined as <8 mg/ml methacholine) that range from 14% to 32%.¹¹⁷

In conclusion, this population-based sample of children attending schools in north and south Durban showed substantial differences in the prevalences of key respiratory outcomes, particularly grades of asthma, persistent asthma and AHR. Children living in the industrially exposed communities had higher risks of these outcomes than those living in non-exposed communities. These findings are consistent with a negative impact of industrial pollution on the respiratory health of schoolchildren.

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Table 5. Covariate-adjusted prevalences* of children's health outcomes by school

	Schools in the south					Average schools			Schools in the north		
	Grand total (N=341)	Nizam (n=51)	Enthu (n=38)	Assegai (n=43)	Dirkie Uys (n=30)	South (n=162)	North (n=179)	Briardale (n=56)	Ferridale (n=69)	Ngazana (n=54)	
Reported doctor diagnosed											
Doctor-diagnosed asthma [†]	13.7 (±9.9)	13.2 (±5.4)	15.8 (±8.4)	4.7 (±2.5)	10.3 (±9.8)	11.0	16.3	22.2 (±8.4)	20.0 (±10.7)	5.5 (±2.8)	
Symptom-based chronic bronchitis	3.5 (±4.9)	5.7 (±4.3)	-	-	6.9 (±5.0)	3.1	4.0	9.3 (±5.8)	2.9 (±2.9)	-	
Doctor-diagnosed hayfever [†]	9.1 (±10.5)	9.4 (±7.1)	2.6 (±2.7)	2.3 (±2.2)	17.2 (±9.8)	7.4	10.8	18.5 (±12.4)	12.9 (±10.6)	-	
Ever had ear infection [†]	25.2 (±14.8)	28.3 (±14.3)	13.2 (±10.3)	16.3 (±9.9)	34.5 (±10.7)	22.7	27.6	40.7 (±14.1)	21.4 (±12.1)	21.8 (±9.9)	
Past year symptom-defined outcomes											
Persistent asthma [‡]	10.8 (±8.27)	17.0 (±8.2)	21.1 (±8.5)	0.0 (±0.0)	10.3 (±3.8)	12.2	9.6	13.0 (±6.6)	8.6 (±4.6)	7.3 (±3.4)	
Chronic cough [‡]	5.3 (±5.0)	5.7 (±3.8)	5.3 (±4.6)	2.3 (±2.1)	3.5 (±6.3)	4.3	6.2	9.3 (±5.7)	4.3 (±4.9)	5.5 (±3.9)	
Chronic phlegm [‡]	2.3 (±4.2)	-	-	-	6.9 (±7.1)	1.3	3.3	1.9 (±2.0)	5.7 (±5.6)	1.8 (±1.8)	
Chronic bronchitis [‡]	1.2 (±3.7)	-	-	-	3.5 (±8.5)	0.6	1.7	-	2.9 (±4.3)	1.8 (±3.9)	
Ever sound wheezy	26.2 (±16.0)	19.5 (±7.2)	56.1 (±13.8)	2.7 (±1.5)	32.0 (±7.0)	25.9	26.5	26.7 (±8.6)	24.4 (±9.6)	29.1 (±8.8)	
Ever had an attack of wheezing with SOB [‡]	9.4 (±5.9)	9.4 (±2.8)	7.9 (±2.8)	-	13.8 (±6.6)	7.4	11.3	16.7 (±5.1)	8.6 (±3.5)	9.1 (±3.0)	
Stuffy, itchy, runny nose [‡]	39.2 (±15.3)	50.9 (±13.1)	23.7 (±7.8)	25.6 (±7.9)	41.4 (±8.7)	36	42.2	57.4 (±11.7)	34.3 (±9.8)	36.4 (±9.7)	
Watery, itchy eyes [‡]	25.2 (±12.6)	32.1 (±15.7)	15.8 (±7.6)	18.6 (±10.2)	20.7 (±5.8)	22.6	27.5	33.3 (±14.6)	22.9 (±9.8)	27.3 (±7.8)	
Test-based outcomes											
Marked AHR [‡]	5.3 (±5.0)	3.9 (±2.1)	10.5 (±6.0)	9.3 (±4.4)	10.3 (±7.4)	8.1	2.8	1.9 (±1.1)	4.3 (±2.6)	1.8 (±0.9)	

SD = standard deviation; SOB = shortness of breath; AHR = airway hyperreactivity.

*Prevalences (±SD) have been adjusted for the following covariates: age, gender, race, education and annual household income.

[†]p<0.0001 (F-test for differences between north and south).

[‡]0.0001 ≤ p ≤ 0.05 (F-test for differences between north and south).

Human subjects declaration. All the legal guardians of the child participants in this study gave written informed consent. Participation was voluntary, and the children had the right to withdraw at any stage. Ethical approval was obtained from the Institutional Review Board of the University of Michigan and the Ethics Committee of the University of KwaZulu-Natal.

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Table 6. Covariate-adjusted* logistic regression models of associations of living in communities in south Durban compared with living in north Durban with child outcomes (N=423)

Health outcome	South/north			PM ₁₀ [†]			SO ₂ [†]		
	OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value
Symptom-defined outcomes									
Chronic cough	0.81	0.47 - 1.40	0.45	1.03	0.41 - 2.58	0.96	1.02	0.47 - 2.19	0.97
Chronic phlegm	1.55	0.76 - 3.15	0.23	0.56	0.16 - 1.93	0.36	0.88	0.32 - 2.42	0.80
Chronic bronchitis	3.53	1.30 - 9.55	0.01	1.02	0.21 - 5.01	0.98	1.34	0.34 - 5.22	0.67
Wheezing	1.00	0.63 - 1.57	0.99	1.27	0.75 - 2.15	0.37	1.13	0.73 - 1.74	0.58
Wheezing with SOB	1.12	1.01 - 1.24	0.04	0.98	0.88 - 1.10	0.72	0.86	0.49 - 1.53	0.61
Stuffy, runny nose	1.22	0.80 - 1.87	0.36	0.91	0.58 - 1.43	0.68	0.96	0.66 - 1.40	0.85
Watery, itchy eyes	2.29	1.48 - 3.55	0.00	1.34	0.81 - 2.23	0.25	1.02	0.67 - 1.53	0.94
Persistent asthma	1.14	0.75 - 1.74	0.54	1.09	0.56 - 2.12	0.79	1.37	0.80 - 2.35	0.26
Test-based outcomes									
Marked AHR	2.49	1.13 - 5.5	0.024	1.08	0.44 - 2.66	0.867	2.14	0.98 - 4.66	0.056

PM₁₀ = particulate matter ≤10 µm in aerodynamic diameter; SO₂ = sulphur dioxide; SOB = shortness of breath; AHR = airway hyperreactivity; OR = odds ratio; CI = confidence interval.

Bold text denotes statistically significant or borderline significance.

*Covariates included age, male gender, race (Indian, coloured, black African (reference group)), education, smoker in home, household income (<R10 000, R10 000 - R29 999, R30 000 - R74 999, ≥R75 000 (reference group)), glutathione-S-transferase M1 polymorphism (GSTM) genotype negative.

[†]Mean school exposure level.

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