Chapter 2

AIR POLLUTION EPIDEMIOLOGICAL STUDIES IN SOUTH AFRICA: NEED FOR FRESHENING UP

Background: The results of epidemiological studies obtained in developed countries cannot be extrapolated with complete confidence to developing countries. The objectives of this review were to examine the evidence from South African studies for associations between air pollution and adverse health along with a critical review for methodology limitations in order to indicate the need for improvement.

Methods: The literature search strategy and selection criteria involved a MEDLINE search up to June 2005. Of 267 journal articles, 14 were found that focused on air pollution epidemiology (excluding active smoking and internal dose as a proxy for health outcomes). Two studies were also located by word of mouth or through the references from the selected studies.

Results: The local studies provide some evidence of an association with a range of serious and common health problems. None of the studies established exposure-response curves for the criteria pollutants carbon monoxide, sulphur dioxide, nitrogen oxides, lead and ozone. Therefore, using the results of those studies in risk assessment studies is impossible. Most of the studies were fraught with systematic and random errors, which limit their validity and precision.

Conclusions: We recommend conducting a quantitative intervention study with an analytical study design in all major cities in the country, where residents are still using dirty fuels for cooking, lighting and space heating. Future studies must involve national and international multi-disciplinary stakeholders and must be planned well in advance.

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2.1 Background

The World Health Organisation (WHO) reports that 25% of all preventable diseases are due to a poor physical environment.[1] Furthermore, over 40% of the global burden of disease attributed to environmental factors falls on children below 5 years of age, who account for only ~10% of the world's population.[2] The term *burden of disease* is defined as lost healthy life years, which includes those lost to premature death and those lost to illness as weighted by a disability factor (severity).[3] The WHO estimates that the number of persons exposed to unsafe *indoor* air pollution levels exceeds those exposed to unacceptable *outdoor* air pollution levels in all of the cities of the world combined.[4] In most countries, air pollution is the largest single environment-related cause of ill health among children, whilst in others it is the second, after the scarcity of safe water.[1,5]

Globally, 2.6% of all ill-health is attributable to indoor smoke from dirty fuels (such as wood, animal dung, crop residues, coal, paraffin) – nearly all in poor regions.[1,4] Dirty fuels are also referred to in the literature as 'solid' fuels. A distinction is also made between biomass fuels or biofuels and fossil fuels. *Biomass fuels* comprise any material derived from plants or animals, which is deliberately burnt by humans. Wood is the most common example, but the use of animal dung and crop residues is also widespread.[5] *Fossil fuels* refers to any carbon-containing fuel – for example, coal, peat, petroleum and natural gas derived from the decomposed remains of prehistoric plants and animals.[6] Indoor air pollution is a serious global public health risk demanding significantly improved research and policy-making contributions. No case in support of environmental action is deeper than that of the need to eradicate health risks.

2.2 Environmental Epidemiology

Despite all their shortcomings, epidemiological studies are important in linking exposure to human health *directly*.[7–22] The ultimate endeavour of epidemiology is to identify modifiable determinants of disease occurrence and progression and to contribute toward testing the effectiveness and efficacy of interventions on such determinants, including health services. The evolving field of *environmental epidemiology* is the study of physical, biological and chemical factors in the external environment and their relation to human health by examining specific populations or communities exposed to different ambient environments. Environmental

epidemiology involves the distribution of health-related states or events in specified populations in relation to determinants/ hazards in their living environment and the application of this study to the control of such hazards.[23,24]

The term *environment* means everything that is not genetic, such as diet, smoking and even exercise that may have an impact on the development, action, or survival of an organism or group of organisms. Environmental epidemiology has a more restricted connotation, however, referring to the disease consequences of involuntary exposures that occur in the general environment and are outside the immediate control of the individual. A comprehensive introduction to the science of environmental epidemiology and environmental health is beyond the scope of this article. The reader may consult Yassi et al, Baker et al, Beaglehole et al and reports by the National Research Council (NRC) in this regard.[23-27]

2.3 Air Pollutants

2.3.1 Chemical Properties, Transport, Environmental Fate

Emissions from industry, traffic and domestic dirty fuel combustion contain variable complex mixtures of numerous air pollutants that are detrimental to health, including respirable particulate matter (RSP), such as particulate matter < 10 μm in aerodynamic diameter (PM_{10}) and $PM_{2.5}$ (< 2.5 μm in aerodynamic diameter), carbon monoxide (CO), sulphur dioxide (SO_2), nitrogen oxides (NO_x), formaldehyde, benzene, 1,3 butadiene, polycyclic aromatic hydrocarbons (PAHs), such as the carcinogen benzo[a]pyrene, B[a]P, many other volatile organic compounds (VOCs) and metals (such as lead, iron, copper), as well as secondary pollutants such as ozone (O_3). A detailed introduction to the physicochemical properties of each pollutant and its environmental fate and transport is beyond the scope of this article. For recent reviews on the exposure assessment of air pollutants, the reader is referred to Monn, the WHO *Air Quality Guidelines* and an article by Patterson et al.[21,28,29]

The physicochemical properties of a pollutant, its geographic distribution and the type of its emission sources (line source such as traffic, or point source such as an industry) determine its spatial variation along with physical processes (such as sedimentation and coagulation) and atmospheric conditions (wind speed, vertical temperature gradient and solar radiation).[30] Furthermore, the composition of smoke

derived from combustion of dirty fuels is determined by fuel type (for example, coal, wood, or gas), fuel quality (for example, low versus high grade coal), time since ignition, combustion device (for example, vented versus unvented devices) and various other factors. The physical and chemical characteristics of wood smoke mixtures in particular have been characterised from metal heating stoves used in developed countries.[31] The time-scale of the small-scale spatial variation can also be important; the size of short-term (for instance within minutes) spatial fluctuations is different from spatial fluctuations in annual means.

2.3.2 Exposure Assessment

The purpose of exposure assessment in environmental epidemiology is to facilitate the investigation of and to establish, a cause-effect relation between an environmental exposure and an adverse health outcome (see NRC, Chapter 3).[26] Exposure to a contaminant can be defined as the contact between a human and a chemical, physical, or biological agent in an environmental carrier medium at a specific contaminant concentration for a specified period of time; the units to express exposure are concentration multiplied by time.[23–25,32] The discipline of exposure assessment encompasses techniques to measure or estimate a contaminant and its source, environ-mental media of exposure, avenue of transport through each medium, chemical/physical transformation, route of entry to the body, intensity and frequency of contact and spatial/ temporal concentration patterns. In environmental epidemiology, exposure assessment has proved difficult (see NRC, Chapter 3).[26]

Exposure to a contaminant can be measured or modelled either directly (including personal sampling and use of biological biomarkers) or indirectly (microenvironmental monitoring, the measurement of contaminant concentrations in water or air).[33–35] Although descriptive studies in which no direct determination of exposure is carried out may imply causation, personal exposure measurements are deemed the most accurate approximation of true exposure for numerous air pollutants. Personal measurements are expensive, labour intensive, time-consuming and invasive.[36–39] Study participants have to carry the sampling equipment. Modelling requires a validated model and sufficient, representative, good quality input data. Once these requirements are met, however, a model can be repeated for a large number of individuals or populations.

A full description of personal exposure to an air pollutant requires the knowledge of the magnitude of pollutant concentration in the exposure environment and the duration and time pattern of exposure.[33] The microenvironmental approach— in which exposure is calculated as the sum of the partial exposures across the visited microenvironments—has been commonly used to model exposures.[34,37,38,40–43] In cases where no measured data are available for an indoor microenvironment, the concentration can be derived as a function of the outdoor concentration, the effective penetration factor and the contribution of indoor sources.[41,42] The latter two factors are dependent on many parameters, such as ventilation rates and time activity patterns. The effective penetration factor considers both infiltration and loss mechanisms (sinks).[42] A meticulous presentation to air pollution exposure assessment is outside the range of this article. Current reviews on the exposure assessment of air pollutants by Monn and the WHO *Air Quality Guidelines* can be consulted in this regard, along with an article by Patterson et al.[21,28,29]

The vast majority of detailed exposure assessment studies on air pollution have been conducted in Europe and North America. In these parts of the world, motorised traffic is the main source of outdoor air pollution generated in close proximity to people. Most indoor sources are due to environmental tobacco smoke (ETS) and unvented gas cooking and to a limited extent, vented space heating. Other indoor sources include pesticide spraying; household chemicals; and radon. The combustion of dirty fuels during cooking, heating and lighting results in high levels of various of air pollutants. Depending on which pollutant is studied, indoor and personal levels often correlate poorly with outdoor air levels.[33,38,39,41,42,44–50]

The results of many studies have indicated that short-term outdoor PM concentrations are adequate proxies for estimating personal exposure to PM of outdoor origin.[51–54] Time-series studies evaluate the short-term effects of air pollution on human health by linking the daily fluctuations in air pollution and daily fluctuations of health endpoints, such as mortality, hospital admissions, respiratory symptoms and lung function. One study in adults by Janssen et al provided support for the use of ambient PM_{10} concentrations as a measure of exposure in time-series epidemiological studies.[52] Conclusions from most time-series studies are that non-accidental mortality is associated with air pollution, especially with particulates.

Some studies reported that outdoor PM_{10} concentrations were generally homogenously distributed across urban areas without major local point sources. However, other studies have recorded notable within-city variation of outdoor concentrations, particularly related to the proximity to busy roads and to the location within the city.[55-57] Such studies have documented a moderate association between multiple fixed-site outdoor and personal exposure PM_{10} measurements of adults and children.[51,52]

Janssen et al reported a strong correlation between personal $PM_{2.5}$ and multiple fixed-site outdoor $PM_{2.5}$ and PM_{10} concentrations.[53] Cross-sectional outdoor and personal exposure measurements exhibit a weaker connection.[58,59] Personal PM_{10} and $PM_{2.5}$ measurements are nevertheless higher than outdoor levels. Oglesby et al reported that personal exposures to $PM_{2.5}$ mass are not correlated with matching home outdoor levels.[60] Kousa et al found that outdoor nitrogen dioxide (NO_2) levels are a poor predictor for personal NO_2 exposure variation, but adding personal questionnaire information can significantly improve the predicting power.[50]

Population studies indicate that study participants living near major roads are more prone to chronic respiratory symptoms, lung function deterioration and hospital admissions for asthma. Most such studies used proxy measures, such as distance from major roads or traffic intensity in the surroundings of the home. Proxy measures are used due to a lack of *concurrently* performed measurements of outdoor, indoor and personal air pollution in urban streets having high and low-traffic density. Nevertheless, proxy variables for traffic-related air pollution exposure must be validated directly for their use as exposure measures in epidemiological studies. Yet only a handful of studies have communicated findings of *concurrently* performed measurements of air pollution in urban streets having high and low-traffic density.[51,61–63] Performing *concurrent* measurements of air pollution in urban streets having high and low-traffic density is important for investigating whether differences between these two exposure categories remain significant after adjustment for potential indoor sources (such as cooking and use of unvented heating appliances). If significant differences are found between high and low-traffic density homes (after adjustment for indoor sources), then the findings will provide

support for the use of the type of road as proxy measure for measuring a particular traffic-related air pollutant in epidemiological studies.

Even fewer studies have reported on the influence of traffic intensity on pollutant concentrations inside homes or on personal exposure measurements. Evidence of an influence of traffic-related air pollution in the indoor environment would significantly reinforce the credibility of the reported health effects associated with motorised traffic. Although persons living in Europe and North America spend a large proportion of their time indoors, linking exclusively home indoor traffic-related air pollution to health effects might bias the association.[64,65] Health effects of air pollutants are caused by the exposures to both outdoor and indoor sources that individuals experience during their daily activities.

Many epidemiological studies treat particulate matter as a single entity and very few have investigated the risk that the different physicochemical characteristics of PM can pose to human health.[22,66] The relation between PM_{10} mass and absorption coefficient measurements has been investigated for outdoor and indoor measurements only in the Netherlands, but not for personal measurements.[55,67] Reflectance measurements of PM collected on filters are easily transformed into absorption coefficients according to standard equations. Filter reflectance is highly correlated with the measurement of elemental carbon, a marker for particles produced by incomplete combustion.[68] One major source for carbonaceous particles is diesel exhaust.[69] Absorption coefficients can be converted into black smoke ('soot') concentrations using a regression equation of the relation between absorption of PM_{10} filters and black smoke concentrations measured simultaneously at the same site, as reported by Roorda-Knape et al.[70] Black smoke is also a good indicator of fine (<1 μm in aerodynamic diameter) and ultrafine (<0.1 μm in aerodynamic diameter) PM and these fractions have been shown to have serious health effects.[71,72]

Furthermore, only short-term associations have been investigated for PM_{10}, whereas long-term personal exposure measurements have been conducted for NO_2.[73] Personal NO_2 sampling is not as labour intensive and time-consuming as personal PM sampling and is therefore much easier and practical to perform.

Smith-Sivertsen et al highlighted that although outdoor sources often dominate air pollution *emissions*, indoor sources frequently dominate air pollution *exposures* because exposure is a function of both the concentration in an environment and the person-time spent in the environment.[74] Dirty fuels are at the bottom of the energy ladder regarding combustion efficiency and cleanliness. Such fuels are energy inefficient and typically burned in simple, inefficient and mostly unvented household stoves, which when combined with poor ventilation, generate large volumes of smoke indoors, often emitting 50 times more pollutant levels than energy equivalent natural gas.[31] Pandey et al reported indoor PM levels of dirty fuel using households to be 20 times greater than those due to cigarette smoking alone.[75] Even when the stoves are vented to the outside, the combustion of dirty fuels produces enough pollution to affect local neighbourhood pollution levels significantly, with implications for total exposures.[76]

Before epidemiological findings can be interpreted into efficient risk reduction policies, a better understanding of the associations between personal exposure to various air pollutants and outdoor levels and their relation to other significant exposure determinants (such as indoor sources, sinks and personal activities) are needed for both developed and developing countries.[77,78]

2.3.3 Adverse Health Effects

In developed countries, various epidemiological and toxicological studies have primarily linked relatively low outdoor air pollution exposure to various mortality and morbidity outcomes (for reviews, see *Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society*, Vedal and Lighty et al. [66,79-81] Cohort studies focusing on long-term health effects are scarce.[82,83]

The pathogenic mechanisms by which air pollution can increase the risk of adverse health outcomes are not fully understood. A thorough description of pathogenic mechanisms is beyond the scope of this article. The WHO *Air Quality Guidelines* and an article by Patterson et al can be consulted in this regard.[28,29] Table I lists some of the mechanisms by which certain key pollutants can increase the risk of respiratory and other health problems. In a statement aiming to provide a comprehensive review of the literature on air pollution and cardiovascular disease, Brook et al addressed several plausible mechanistic pathways for cardiovascular

diseases, including enhanced coagulation/thrombosis, propensity for arrhythmias, acute arterial vasoconstriction, systemic inflammatory responses and the chronic promotion of atherosclerosis.[115–122]

Table I
Mechanisms by which some key pollutants may increase the risk of respiratory and other health problems (modified from Bruce et al[84])

Pollutant	Mechanism	Potential health effects
PM_{10} and primarily $PM_{2.5}$	• Acute: bronchial irritation, inflammation and increased reactivity.[85–88] • Reduced mucociliary clearance[89] • Reduced macrophage response and reduced local immunity[88,90,91] • Fibrotic reaction[92]	• Wheezing, exacerbation of asthma • Respiratory infections • Chronic bronchitis and chronic obstructive pulmonary disease • Exacerbation of chronic obstructive pulmonary disease
CO	• Binding with hemoglobin to produce carboxy hemoglobin, which reduces oxygen delivery to key organs and the developing fetus[93–95]	• Low birth weight (fetal carboxy-hemoglobin—2% to 10% or higher) • Increase in perinatal deaths
PAHs, such as B[a]P	• Carcinogenic[96] • Immune suppression[97–99] • Absorption of toxins into lens, leading to oxidative changes[100,102]	• Lung cancer • Cancer of mouth, nasopharynx and larynx • Cataract
NO_2	• Acute exposure increases bronchial reactivity[103,104] • Longer term exposure increases susceptibility to bacterial and viral lung infections[105–108]	• Wheezing and exacerbation of asthma • Respiratory infections • Reduced lung function in children
SO_2	• Acute exposure increases bronchial reactivity[109–111] • Longer term: difficult to dissociate from effects of particles[112,113]	• Wheezing and exacerbation of asthma • Exacerbation of chronic obstructive pulmonary disease, cardiovascular disease
Metal ions, such as iron or copper	• Absorption of toxins into lens, leading to oxidative changes[114]	• Cataract

Thomas and Zeliko reported that exposure of animals to wood smoke significantly modifies the immune response to bacterial infection.[123] Supportive evidence is also available from studies reporting that chronic exposure to tobacco smoke (having a similar pollutant mix as dirty fuel smoke) decreases cellular immunity, antibody production and local bronchial immunity, as well as increasing susceptibility to infection and cancer.[124–128] Indeed, tobacco smoke has been associated with tuberculosis (TB).[129,130] With narrower respiratory passages and poorly developed immune systems, young children are particularly susceptible to air pollution that can impair clearing mechanisms of the respiratory tract and allow bacteria and viruses to enter the lower airways. Therefore, extended exposure to high levels of air pollution

can impair the pulmonary defence mechanisms, compromise the lung function and render individuals more susceptible to various adverse health outcomes.

Most international epidemiological studies have highlighted the significant health impact of PM_{10} and more recently $PM_{2.5}$. The strongest and most coherent associations have been found for outdoor particulate matter (PM_{10} and $PM_{2.5}$) when compared with other criteria air pollutants—CO, SO_2, NO_x, lead and O_3.[79,80] As outdoor particle levels are often correlated with outdoor concentrations of gaseous pollutants, possibly such associations between particles and adverse health effects could be due to confounding by these other correlated pollutants and not to the particles themselves.[33,36] If co-pollutants are surrogates, inserting them into a multivariate model could in fact lower the detection of an effect. Nearly all such studies are also based on exposure data from a single central outdoor monitor, which could introduce information bias.[131-133] The selection of the site is critical for most air pollutants as substantial spatial variations in concentration levels arise.

2.4 Indoor Air Pollution And Health

2.4.1 The Evidence

Although high exposure to air pollutants in dirty fuel smoke has been associated with a host of adverse health outcomes, many uncertainties remain. Reports by the WHO, Smith, Ezzati and Kammen as well as Bruce et al highlighted international research needs to address the link between indoor air pollution, household energy use and human health.[84,134-136]

Strong evidence from studies of outdoor air pollution indicates that active and passive smoking and multiple studies in dirty fuel using households are risk factors for acute respiratory infections (ARI) and chronic obstructive pulmonary disease (COPD), a progressive lung disease characterised by difficulty in breathing, wheezing, chronic cough, eventually resulting in bronchitis, pneumonia, emphysema, or lung cancer.[75,137–147]

Consistent evidence that dirty fuel smoke exposure increases the risk of ARI in children (< 5 years) is based on studies from Zimbabwe, Nigeria, Tanzania, Gambia, Brazil, India, Argentina, Nepal and the United States (U.S.).[75,137-147] ARI is

the most important single cause of global burden of disease and is responsible for ~33% of all deaths among children under 5 years of age in developing countries.[1,84]

Globally, the prevalence of COPD has not been studied to the same extent as asthma. Although a number of studies of chronic bronchitis have been reported in selected populations in middle- and low-income countries, the overall burden and determinants of COPD in such countries are not well documented.[148–153] Bruce et al and Smith evaluated studies from Saudi Arabia, Columbia, Mexico, Nepal, India and Bolivia and reported an adjusted odds ratio (OR) range of 2–4 for women cooking for many years over biomass fires.[84,148,152-158]

Smith and Liu evaluated more than 20 studies conducted in China on the association between lung cancer in women and exposure to cooking smoke from open coal stoves.[159] The authors reported an OR range of 3–5 (95% confidence level) for nonsmoking women. In addition, certain coals produce large indoor exposures to arsenic and fluorine.[160]

Rural communities in poor countries still rely heavily or exclusively on biomass fuels, such as wood and dung, but evidence of a connection between lung cancer and biomass fuel is sparse. In Europe, where lung cancer rates are high, many residents have had a long tradition of burning unprocessed biomass fuel. Yet, only one study conducted by the International Agency for Research on Cancer in Central and Eastern Europe and the United Kingdom evaluated the contribution of combustion fumes from solid fuel (combined coal and biomass) used for cooking and heating at home to the development of lung cancer.[161] The results suggested a modest increased risk of lung cancer that is related to solid-fuel use for cooking rather than for heating.

In India, however, strong evidence emerged from a recent study indicating that biomass fuel exposure alone is an important risk factor in the causation of lung cancer among women.[162] Among non-smokers, of all the cooking fuels tested, the risk of development of lung cancer was highest for biomass fuel exposure with an OR of 5.33 (95% confidence interval (CI) = 1.7–16.7). In multivariate logistic regression analysis, biomass fuel exposure was still significant with OR of 3.59 (95% CI 1.07–11.97), even after adjusting for smoking and passive smoking.

Moderate evidence from studies of outdoor air pollution, smoking, laboratory animals and studies in biomass-using households can be found for links with cataracts, TB, asthma attacks, adverse pregnancy outcomes (stillbirth, low birth weight (< 2500 g), perinatal mortality (< 14 days), nasopharyngeal and laryngeal cancer and trachoma.[163–190] Pollution attributable to the use of biomass fuel causes eye irritation and may cause cataracts.[163] An analysis of over 170,000 inhabitants in India yielded an adjusted OR for reported partial or complete blindness of 1.32 (95% CI = 1.16–1.50) with respect to persons using mainly biomass fuel compared with other fuels and significant differences were found between men and women and between urban and rural residents.[164] The results were adjusted for socioeconomic, housing and geographic variables, but information was lacking on smoking, nutritional state, episodes of diarrhoea and other factors that might have influenced the prevalence of cataracts.

On the other hand, the crude method of classifying exposure could be expected to result in an underestimation of the effect. A Delhi clinical case-control study established comparable risks of 1.6 for cataract-caused blindness after adjustment, whereas another case-control study in Nagpur, India, conveyed an adjusted OR of 2.4.[165,166] Animal studies have revealed findings of cataracts from wood smoke exposure.[167] The growing evidence that environmental tobacco smoke causes cataracts is supportive.[114,168]

An analysis of data on 200,000 adults in India from the same national survey as mentioned above found an association between self-reported TB and exposure to wood smoke.[166,169] Persons living in households burning biomass fuels reported TB more frequently than did persons using cleaner fuels, with an OR of 2.58 (95% CI = 1.98–3.37) after adjustment for a range of socioeconomic factors. A clinical study in the north of India reported analogous risks (2.5), although not adjusted for potential confounders other than age.[158] A recent case-control study in Mexico City conveyed an adjusted OR of 2.4 for clinically confirmed TB cases in households using wood for cooking.[170] Animal studies demonstrated a decline of respiratory immune function from wood smoke exposure.[90]

Conflicting evidence has emerged on the effect of dirty fuel smoke on asthma, even though such smoke contains some of the same pollutants that are found in ambient air pollution or environmental tobacco smoke, both of which are associated with asthma.[84] Anecdotal association of asthma with cooking smoke is common, but few epidemiological studies seem to have been carried out.[135] Of the limited research that does exist on this subject, several studies found a positive association between cooking smoke and asthma[171-174], whereas others either found no relation[175–178] or found a protective effect[179,180]. In developing countries, three studies in biomass-using households reported adjusted ORs that range from 1.4 to 2.5.[171,174,175]

Low birth weight has been associated with both outdoor air pollution and active and passive smoking in developed countries.[181-184] Only one study has specifically reported the association with outdoor CO levels.[185] Very few studies conducted in developing countries (India[185] and Guatemala[186]) have investigated the impact of dirty fuel use and low birth weight. Low birth weight is a risk factor for several diseases in childhood and possibly in later life. To date, the data are too sparse to establish ORs. A recent study in São Paulo, Brazil by Gouveia et al, however, reported negative effects on birth weight following exposure to outdoor PM_{10} and CO during the first trimester.[187] This effect seemed to be more robust for CO, with a reduction of 23 g in birth weight per 1 part per million (ppm; 1 ppm = 1.15 mg m^{-3}) increase in mean CO during the first trimester. Additionally, the evidence shows that maternal smoking during pregnancy can lead to reduced early infant lung function, with potential long-term effects. One study, for example, found significantly lower forced expiratory volume after controlling for infant size and other relevant confounders.[188] Thus, even a relatively low risk would translate into a large attributable burden due to the possibly chronic impact of adverse pregnancy outcomes on later life.

Nearly 90% of 11.7 million global deaths occur before the age of 5 years.[1] Thus, investigating the association between neonatal (< 7 days), perinatal, infant (< 28 days) and child (< 59 months) mortality with modifiable risk factors is imperative. Only one study has reported an association between perinatal mortality and exposure to indoor air pollution in India, with an OR of 1.5 (95% CI = 1.0–2.1) for stillbirths, following adjustment for a wide range of factors.[185] A univariate

association with early neonatal deaths did not persist after adjustment. Supportive evidence has been provided by outdoor air pollution studies.

A time series study in Mexico City examined the relation between $PM_{2.5}$ and infant mortality.[189] The most robust impact was 3–5 days before death, when an increase of 10 mg m^{-3} was associated with a 6.9% (95% CI = 2.5–11.3) excess infant mortality rate. Infant mortality rates in the U.S. showed a surplus in perinatal mortality with elevated PM_{10} concentrations: adjustment OR of 1.10 (95% CI = 1.04–1.16) for the high pollution group (mean 44.5 mg m^{-3}) versus the low pollution group (mean 23.6 mg m^{-3})[190] High exposure was associated with respiratory mortality (OR = 1.40, 95% CI = 1.05–1.85) and sudden infant death syndrome (SIDS) (OR = 1.26, 95% CI = 1.14–1.39) in infants of normal birth weight. Conversely, in an ecological study of pollution and stillbirths in the Czech Republic, no association was established between any measure of pollution—total suspended particulates (TSP), SO_2, NO_x—and stillbirths, regardless of the association with low birth weight.[183]

Three-quarters of the global cardiovascular disease (CVD) burden lies in developing countries and its rapid growth is projected over the next 2 decades.[191] Mendis et al addressed the dearth of studies focusing on CVD in developing countries, but recent suggestive evidence has emerged from studies of outdoor air pollution and smoking and CVD—for example, a study by Chang et al in Taipei, Taiwan reported statistically significant positive associations on warm days (≥ 20 °C) between CVD hospital admissions and outdoor PM_{10}, NO_2, CO and O_3 levels in single-pollutant models, after controlling for weather variables, day of the week, seasonality and long-term time trends. On cool days (< 20 °C), all pollutants except O_3 and SO_2 were significantly associated with CVD admissions.[192,193] For two-pollutant models, CO, NO_2 and O_3 were significant in combination with each of the other four pollutants on warm days. On cool days, PM_{10} remained statistically significant in all the two-pollutant models. A review by Brook et al on the impact of outdoor air pollution on CVD in the U.S. states that epidemiological studies have demonstrated a consistent increased risk for cardiovascular events in relation to both short- and long-term exposure to present day concentrations of ambient particulate matter.[115] No known studies have yet been conducted in households using dirty fuels.

A preliminary study in Honduras conducted by Ferrera et al provided suggestive evidence on an association between invasive cervical cancer and exposure to wood smoke in human papillomavirus (HPV)-positive cases.[194] A follow-up study by this group (Velema et al) reported that burning wood in the kitchen increases the risk of cervical neoplasia in HPV-infected women, showing a significant linear dose-response relation ($p = 0.026$), independent of other risk factors.[195] The ORs were 5.69 for more than 35 years of exposure to wood burning in the kitchen.

2.4.2 Health Research Needs

Reports from Smith and the WHO emphasise that epidemiological studies must include case-control studies for TB and CVD in women, randomised intervention trials for childhood acute respiratory diseases and adverse pregnancy outcomes (stillbirth, low birthweight), as well as case-control and/or cohort studies to strengthen the evidence on outcomes for which very few studies currently exist: perinatal mortality, cataract and asthma (development/exacerbation).[134,135]

Systematic reviews and meta-analyses of the health risks of indoor air pollution are required to estimate a pooled relative risk from the available evidence. Existing and new evidence on the exposure-response relation for indoor air pollution have to be collated to produce improved exposure-response relation information for key outcomes like childhood ARI. Studies should also focus on the direct effects arising from the use of household energy, not just those resulting from indoor air pollution: burns, injuries, paraffin poisoning and house fires. Particularly lacking are good population studies of incidence and factors determining risk. Less-direct health consequences should also be targeted: opportunity costs of women's time; injuries from carrying large loads of wood; restrictions on opportunities for education (adult and child), leisure and economic activities in the home; other economic activity outside the home; issues arising from gender power imbalance and decision-making about the use of energy and appliances; impact of inter-relations between scarcity of fuel and stressed local environments.

Research on exposure assessment must cover the entire spectrum of exposure indicators, from indirect indicators like fuel use and house type to area and personal measurements and biomarkers. This approach also involves technique development for inexpensive equipment needed in large scale studies, including national level

surveys. Research on new and existing interventions is needed. The experience from existing household energy implementation efforts to identify, compile and disseminate lessons learned from both the technology employed and the implementation approaches taken (improved stoves, fuels, ventilation and behaviour) must be reviewed. Developing and evaluating a range of interventions and policies for implementation in a variety of settings is needed to reduce exposure economically.

Ezzati and Kammen stressed that when conducting intervention and/or epidemiological studies, zooming in only on accurate and valid quantitative personal, indoor and outdoor air pollution measurements and/or biomarkers without linking these measurements to the distal and proximal causes of disease, such as poverty, home design and socio-behavioural practices, is insufficient.[136] Epidemiological studies would then focus only on causation and miss its goal to introduce interventions to eliminate or reduce risk factors.

Lastly, studies must further develop methods required for economic studies, such as research to help understand and estimate secondary impacts of interventions on cooking time, fuel gathering and crop production. The complete set of the direct impact of the intervention must be clear for households to evaluate the desirability of the intervention. Research to understand the household benefits of risk reduction using cost-of-illness and willingness-to-pay valuations should allow for differences in household values for adult and child risks.

Further research is needed on cost-benefit and cost-effectiveness analyses of specific interventions in various settings. Macro-economic (national) consequences of policy options relating to the supply and uptake of cleaner household energy for the poor also have to be addressed. Risk communication should be improved by the development and assessment of appropriate means for conveying information on health risks and interventions to households.

2.5 Air Pollution Epidemiological Studies in South Africa

Various epidemiological and toxicological studies have linked relatively low outdoor air pollution exposure to mortality and morbidity outcomes in developed countries. South Africa, however, has a fragile population that might be more susceptible to

the adverse effects of air pollution. The country is confronted by air pollution caused by industry and traffic (a developed country state of affairs) and by domestic burning of dirty fuels (developing country situation). The statistics estimate that 10.5 million lives might be lost to AIDS by 2015.[196] Furthermore, the country has ~43.7 million inhabitants, of which one-third are younger than 15 years.[197] Depending on the poverty line and the methodology used, various estimates have been made about the extent of poverty in the country. StatsSA estimates that 52% of households were living in poverty in 1996.[197] The Gini-coefficient is a measure of income inequality, ranging from 0 to 1, with 0 representing absolute equality and 1 representing absolute inequality. The Gini-coefficient for South Africa is currently 0.58, the second highest in the world. In 2003, the unemployment rate was 30% and just over 25% of South Africans lived in informal housing.[198] During the same year, 41%, 48% and 23% of 11 million households used dirty fuels for cooking, heating and lighting, respectively.[197]

Bailie et al conducted an indoor exposure assessment study during winter in a poor urban environment in South Africa, where a range of fuel types was used, including paraffin.[199] The mean maximum hourly average was 28 μg m^{-3} (range 0–451 μg.m^{-3}) for NO_2, 1414 μg m^{-3} (range 0– 17 723 μg.m^{-3}) for SO_2 and 34 mg m^{-3} (range 0–388 mg m^{-3}) for CO.[199] The number of households for which NO_2, CO and SO_2 standards were exceeded by the maximum hourly averages was six (9%), 20 (30%) and 28 (42%) respectively. The hourly WHO standards are 200 μg m^{-3} and 30 mg m^{-3}, respectively for NO_2 and CO.[28] The hourly California standard is 655 μg m^{-3} for SO_2.[200] *Hourly* WHO or U.S. EPA standards do not exist for SO_2.[28,200] Concentrations of TSP ranged from 7–433 μg m^{-3}. The investigators did not compare the latter with international standards because TSP measurements now focus on PM_{10} and $PM_{2.5}$ particles. To date, hourly standards or guidelines do not exist for PM_{10}/$PM_{2.5}$.

Röllin et al provided scientific evidence that during summer, electrified homes in South African villages have lower levels of air pollution (RSP and CO) than do non-electrified homes.[201] Even in partially electrified homes, RSP levels were significantly lower (mean 77 μg m^{-3}, median 37.5 μg m^{-3}) in electrified areas than in non-electrified areas (mean 162 μg m^{-3}, median 107 μg m^{-3}) ($p = 0.012$).

Stationary (kitchen CO) levels in un-electrified and electrified dwellings ranged from 0.36–20.95 ppm (1 ppm = 1.15 mg m^{-3}) to 0– 11.8 ppm, respectively. The mean level of log (CO) in the kitchen was significantly higher in un-electrified areas (1.25 vs. 0.69) ($p = 0.0004$). The mean level of log (CO) for personal measurements in children (< 18 months old) was higher in un-electrified areas (0.83 vs. 0.34) ($p < 0.0001$). Such CO levels are comparable with those associated with indoor exposure to environmental tobacco smoke (8-h average 20–40 ppm).

A local study by Thomas et al reported that on average, 14% of households had children (<6 years) who were usually or always present when their mothers were cooking.[202] This percentage increased to 18.3% of households in the lowest wealth quintile. In lower-wealth quintile homes, paraffin stoves were burning on average 4 h per day, with the evening meal taking over half of this time to prepare. Muller et al established in another local study that during both winter and summer individuals spend on average 2 h cooking indoors.[203]

The consequent lack of infrastructure and inadequate living conditions in many areas of South Africa means that millions of people are routinely exposed to noxious smoke emitted through the combustion of dirty fuels. Yet, very few comprehensive local studies have attempted to measure the extent and consequence of public exposure to indoor air pollution. The country cannot rely merely on results (risk estimates and exposure-response relations) deduced from studies conducted in developed countries for estimating burden of disease indicators. The three global factors that directly or indirectly impact on health—community and social environment, physical environment and family and individual environment—differ between/among developed and developing countries.[22, 204,205]

2.6 Literature Search: Methodology

This review focuses exclusively on the chemical component of air pollution, thereby excluding the biological component. The review is also restricted to the environmental epidemiological field and excludes studies related to occupational epidemiology. The objectives of this review are (a) to examine the evidence from studies conducted in South Africa for possible associations between air pollution and ill health and (b) to critically review these studies for methodological limitations in order to stress improvement of future studies. The possible reasons for the lack of

air pollution epidemiological studies in the country are also addressed. Finally, recommendations are made in this regard.

The literature search strategy and selection criteria involved a MEDLINE search until June 2005 for key words *air pollution* or *smoke* or *smoking* or *environment* and *South Africa* and *health* and *child* or *adult* or *elderly*.** The search revealed 267 journal articles in total. Fourteen studies were found that focused on air pollution epidemiology. Two studies were also located by word of mouth or through the references of the selected studies (Klopper et al, cited in Zwi et al and Thomas et al by word of mouth).[202,206,207] All studies investigating active smoking were excluded because such studies do not fit the restricted connotation of environmental epidemiology, meaning involuntary exposure. Furthermore, this review will only focus on manifested health outcomes and not on internal dose as an outcome.

2.7 South African Air Pollution Epidemiological Studies

Eight of the sixteen studies were not designed specifically to investigate air pollution indicators and associated health outcomes. In such studies, air pollution indicators were treated as confounders.[202,208–213,215,216]

The aim of the first published South African environmental epidemiology study was to investigate whether smoke exposure from cooking or heating fires was significantly greater in a group of children with severe lower respiratory disease than in a group of children who had no respiratory signs or symptoms.[217] The study commented on the long hours of exposure of Zulu children to high levels of smoke from cooking or heating fires and linked clinical evidence of respiratory diseases to self-reported high levels of exposure to smoke. In this study, 70% of 132 children with respiratory diseases had smoke exposure whereas only 33% of 18 children with non-respiratory diseases had been exposed to smoke ($p < 0.005$). The sample size, however, was too small to show a significant difference in parental smoking in the two groups of children.

*** Including conference proceedings or any other type of grey literature (for example, theses, dissertations or unpublished technical reports) would have been impracticable due to the difficulty in tracking this kind of literature.*

The study by Thomas et al covered a wide range of social, health and environmental issues and possible ways to address the problems were identified.[202] The authors documented a significant inverse correlation between ARI symptoms and wealth ($p = 0.017$). Tobacco smoking in the home was significantly correlated with ARI symptoms in children ($p = 0.023$). Notwithstanding the common use of pesticides in the home, no significant association was found between pesticides and ARI ($p = 0.693$). Children living in overcrowded conditions were more likely to suffer from symptoms of ARI than were control subjects ($p = 0.001$). Damp in the house was related to child ARI ($p = 0.021$). Dust and the burning of refuse were noted as a major cause of air pollution.

Ehrlich et al measured the associations between current asthma or wheezing and factors such as household smoking, damp and dietary salt preference in a questionnaire-based prevalence study of Cape Town schoolchildren (aged 7–9 years).[208] In a random sample of 15 schools, questionnaires were completed by the parents of 1 955 children, from whom 368 cases and 294 controls were selected, based on reported asthma diagnosis or symptoms. Concentrations of urinary cotinine (a biomarker of ETS exposure) were measured and the parents were interviewed. The results revealed an exposure-response relation between the urinary cotinine creatinine ratio and asthma/wheeze. In multivariate analysis, predictors of asthma/wheeze were hay fever (OR = 5.30; 95% CI = 3.16–8.89), eczema (OR = 2.19; 95% CI = 1.33–3.62), parental asthma (OR = 1.77; 95% CI = 1.11–2.84), absence of paternal contribution to income (OR = 1.72; 95% CI = 1.17–2.54), maternal smoking during pregnancy (OR = 1.87; 95% CI = 1.25-2.81) and each additional household smoker (OR = 1.15; 95% CI = 1.01–1.30). When the group was restricted to children with parent-reported asthma, the findings were similar, with higher ORs for most variables, except for the number of household smokers. The findings confirm that household smoking is an important modifiable risk factor in asthma/wheeze among young schoolchildren and suggest that maternal smoking in pregnancy and current household exposure are independent contributors to this effect.

Ehrlich et al analysed data from the 1998 South African Demographic and Health Survey (SADHS) to determine the prevalence and predictors of adult chronic bronchitis, defined as chronic productive cough.[209,218] A stratified national

probability sample of households was selected. All adults in the selected households were interviewed. Socio-demographic predictors were wealth, education, race, age and urban residence. Personal and exposure variables included a history of TB, domestic exposure to smoky fuels, occupational exposures, smoking and body mass index. The overall prevalence of chronic bronchitis was 2.3% in men and 2.8% in women. The strongest predictor of chronic bronchitis was a history of TB (men OR = 4.9; 95% CI = 2.6–9.2; women OR = 6.6; 95% CI = 3.7–11.9). Other risk factors were smoking, occupational exposure (men), domestic exposure to smoky fuels (women) and being underweight (univariate analysis only). Wealth and particularly education were protective. The pattern of chronic bronchitis in South Africa thus suggests a combination of risk factors.

The main aim of a project based in the major urban and peri-urban areas of the country was to examine the impact of environmental risk factors associated with housing on diarrhoeal disease and ARI.[210] Study results were reported on a national level. Significant risk factors for coughing and breathing problems were found when more than one adult smoked in the household (OR = 2.0; 95% CI = 1.3–3.3); not using electricity (OR = 1.7; 95% CI = 1.1–2.5); gas, paraffin, coal, or wood used as cooking fuel (OR = 1.7; 95% CI = 1.3–2.5) or heating fuel (OR = 2.0; 95% CI = 1.7–3.3); not owning a refuse bin (OR = 2.4; 95% CI = 1.1–5.0); no chimney in the home (OR = 1.8; 95% CI = 1.3–2.5); presence of a child younger than 2 y of age (OR = 1.3; 95% CI = 1.0–1.8), low income per household (OR = 1.5; 95% CI = 1.1–2.2) and low maternal school education level (OR = 1.7; 95% CI = 1.2–2.4).

The overall aim of the Birth to Ten (currently known as Birth to Twenty, BTT) study was to determine biological, environmental, economic and psychosocial factors associated with the survival and health of children living in an urban environment.[211,212] The objectives relevant to air pollution epidemiology were to look at the:

- incidence rates of respiratory symptoms and illness in a subcohort of Soweto children;
- effect of the indoor environment, particularly factors such as domestic fuel usage, smoking, crowding as well as the outdoor environment; and

- management of children with acute respiratory illnesses.

The results from the study indicated that at 6 months of age, 50% of children who live in homes with an open fire experience respiratory-related symptoms such as sneezing or a runny/stuffy nose compared with 24% of a control group ($p = 0.065$).[211] The caretakers' perception of air pollution as a problem significantly influenced the report rate of respiratory symptoms (OR = 1.35, $p = 0.004$). Having a pet in the home was also a significant risk factor (OR = 1.38; $p = 0.004$) and living with a smoker posed a risk for breathing difficulties and fever (OR = 2.5; p-value not quoted).

Wesley and Loening examined factors that might increase the severity of ARI and monitored subsequent respiratory symptoms during a 2-year follow-up.[213] No significant differences were found between cases and controls when comparing overcrowding in homes, occupancy of sleeping rooms and nutritional status. Indoor pollution risks were similar for both groups, with a respective incidence of adult smoking of 75% and 69% for cases and controls. Wood or coal fires were used in 19% and 14% of the homes of cases and controls, respectively.

Dudley et al investigated the impact of indirect indicators of air pollution on human health, focusing on vitamin A levels as a risk factor for respiratory infection.[214] The results revealed differences in risk between severe and mild cases of ARI with respect to housing conditions (OR = 4.2; 95% CI = 1.3–14.5) and possession of clinic report cards (OR = 3.4; 95% CI = 1.0–11.5). Mild cases were more likely to have had a prior ARI than were controls (OR = 3.2; CI = 1.0–10.1). The mothers of severe cases were more likely to be under 20 years old (OR = 9.9; 95% CI = 1.1–228) and severe cases were more likely to have had a hospital admission during the last 6 months (OR = 5.5; 95% CI = 1.2–33.4), poorer housing conditions (OR = 7.9; 95% CI = 2.2–29.9) and no electricity (OR = 4.9; 95% CI = 1.6–16.2).

Nriagu et al determined the prevalence of respiratory and asthma symptoms and evaluated the degree of under-diagnosis of asthma in the population in Durban.[215] Results indicated that cigarette smoking, ambient industrial pollution, insecticide

use and home ownership were strongly associated with a high prevalence of asthma and respiratory symptoms.

Mzileni et al measured the risk of developing lung cancer related to tobacco smoking, fuel use and residential/occupational exposure to dust and asbestos.[216] The authors reported a significant increase in the risk of developing lung cancer through tobacco smoking when compared with never smokers. In men, ORs were 2.2 (95% CI = 1.0–4.6) in ex-smokers, 9.8 (95% CI = 5.9–16.4) in light smokers (0–14 g/day) and 12.0 (95% CI = 6.5–22.3) in heavy smokers. In women, ORs were 5.8 (95% CI = 1.3–25.8) in ex-smokers and 5.5 (95% CI = 2.6–11.3) in current smokers. Work in a dusty industry constituted an elevated risk (OR = 3.2, 95% CI = 1.8–5.8) for lung cancer only in men. Males resident in areas where asbestos was shipped for distribution (moderately polluted asbestos area) had a 2.5-fold increased likelihood (95% CI = 1.0–4.4) of developing lung cancer. Residents of areas where asbestos was mined (heavily polluted asbestos area) had a 2.8-fold increased likelihood (95% CI = 0.7–10.4) of developing lung cancer. Female residents of heavily polluted asbestos areas showed a 5.4-fold increased likelihood (95% CI = 1.3–22.5) of developing lung cancer.

A 1986 study conducted in the industrialised Sasolburg area performed spirometry but did not collect the appropriate exposure data and the results were generally inconclusive.[219] Children from primary schools at Sasolburg were compared with those from neighbouring rural towns having negligible air pollution levels. Although no significant differences in the incidence of respiratory illness emerged from the questionnaire, the FEV_1 of the 174 boys in the study area differed significantly from that of the 81 boys in the control area. No significant differences in the other lung function tests (FVC, PEV, FEV_{50}) were found for either boys or girls, even when comparing children with smoking and non-smoking parents. The FEV_1 of girls from a higher social class was significantly different (n(study area)=27; n(control area)=16).

Spirometry is the most basic and frequently performed test of pulmonary (lung) function. A device called a spirometer is used to measure how much air the lungs can hold and how well the respiratory system is able to move air into and out of the lungs. Spirometry records the entire forced breathing capacity against time. This test

is used to determine the cause of shortness of breath, to rule out any kind of obstructive disease that blocks breathing, or restrictive disease that limits the expansion and capacity of the lungs. Spirometry is most often used to diagnose and monitor lung problems, such as chronic bronchitis, emphysema, pulmonary fibrosis, COPD, or asthma. Common parameters that spirometry measures mentioned in this review are the following:

VC	Vital Capacity: volume change of the lung between a full inspiration and a maximal expiration.
FVC	Forced Vital Capacity: maximum volume of air that can be forcibly and rapidly exhaled.
FMEF	Forced Midexpiratory Flow: average expiratory flow over the middle half of the FVC maneuver
FEV_1	Forced Expiratory Volume: volume of air expelled in the first second of a forced expiration.
FEV_{50}	Forced Expiratory Flow at 50% of FVC
$FEF_{(25-75)}$	Forced Expiratory Flow during mid expiratory phase, in which 25%–75% of FVC is expired
PEF	Peak Expiratory Flow measured in liters min^{-1}
PEV	Peak Expiratory Velocity
PIV	Peak Inspiratory Velocity
FMFT	Forced Mid Flow Time
PNIF	Peak Nasal Inspiratory Flow

Spirometry results are expressed as a percentage and are considered abnormal if less than 80% of the normal predicted value.

Another study determined (a) specific health conditions that manifest themselves in clearly recognisable forms, (b) whether excess morbidity and mortality existed in those areas where the local authority had allegations that air pollution related health problems were said to exist and (c) to compare the data with similar data obtained from appropriately selected control areas.[206] The authors reported that the age-adjusted morbidity rates for upper respiratory tract infections varied between 4.38 and 16.51 for the control areas (n = 4). The study area had a rate of 12.69 (n = 1).

A study conducted in the former Eastern Transvaal Highveld (now known as Mpumalanga Highveld) was undertaken to determine whether exposure to community air pollution resulted in detectable effects on children's respiratory health.[207] The authors reported that cough, wheeze, asthma and chest illnesses were significantly more prominent in the study area (controlling for parental smoking and

home cooking fuel) compared with the control area (OR = 1.34, 95% CI = 1.14–1.56; OR = 1.20, 95% CI = 1.03–1.39; OR = 1.26, 95% CI = 1.02–1.55 and OR = 1.88, 95% CI = 1.18–3.00, respectively). No differences were found in lung function tests (VC, FVC, FEV_1, FMEF, FMFT, FEF and PEF) after controlling for height, age, parental smoking and home cooking fuel. In general, the study was inconclusive.

The key purpose of the Vaal Triangle Air Pollution Health Study (VAPS) was to assess the exposure and effects of indoor and outdoor air pollution on the human health.[220–223] The results reflected that during 1992 65.9% of children aged 8–12 years suffered from upper respiratory illnesses and 28.9% from lower respiratory tract illnesses. The risk of upper respiratory tract infections was 34 times higher among those using coal and/or wood for cooking and heating (n = 4713) relative to those using electricity (n = 2433). The asthma rates of the groups were similar. The risk for lower respiratory tract illnesses and symptoms (such as coughing and chronic phlegm) were 103 and 97 times higher, respectively for children living in the polluted Vaal Triangle compared to the control area (Klerksdorp).

A study by Richards et al investigated the effects of passive cigarette smoke exposure in the home on the levels of two plasma anti-oxidative nutrients, vitamins C and E and the development of smoke-mediated pulmonary, immunological, or haematological abnormalities.[224] The investigators also used data derived from a questionnaire and related this to domestic smoke exposure. The prevalence of respiratory illness before and after 2 years of age, pneumonia ever, croup ever, cough first thing in the morning, earache over the past year, low birth weight and learning difficulties were significantly increased in children exposed to parental smoke, especially those exposed to maternal smoking. The ORs (95% CI) were 2.18 (1.25–3.78); 3.62 (2.30–5.70); 3.23 (1.54–6.80); 4.68 (2.58–8.50); 2.95 (1.44–6.03); 2.07 (1.39–3.07); 2.63 (1.61–4.31) and 2.08 (1.21–3.56), respectively. Cotinine levels, spirometric (FMEF and FEV_1), immunological and haematological tests were not significantly affected by parental passive smoking.

Sanyal and Maduna determined the levels of indoor gaseous pollutants and their impact on the respiratory health of children.[225] The study established that high levels

of recurring ARI among children were most prevalent in very low and low income households using wood and coal as the main source of heating.

2.8 Study Design

From Table 2 follows that most South African epidemiological studies have focused on children and teenagers.

Under Apartheid, South Africans were categorised into one of four socially defined groups: White (mainly European ancestry), Asian (Indian sub-continent ancestry), African or Black (descent primarily from one of a number of Bantu language groups in Southern Africa) and Coloured (general grouping, including a mixture of Black, Malay, European and indigenous Khoisan ancestry). Race is very much linked to past access to resources, socioeconomic status and educational status. Local researchers Steyn et al stressed that it is also important to identify groups that have different biological as well as environmentally determined risk profiles and to target these groups for appropriate intervention.[226] Many of the local studies did not specify the race of the study population (Table 2). Approximately an equal number of studies focused on the African/Black and White population groups. Fewer studies focused on the Coloured or Indian/Asian population groups. Three of the sixteen studies were conducted in the Western Cape Province; one in the Limpopo Province; two in the Eastern Cape Province; three in Gauteng; three in KwaZulu-Natal and one in the Free State Province (Table 2). One study was conducted in four provinces (Gauteng, Mpumalanga, Northwest Province, Limpopo Province). One study was conducted nationwide and another in the major urban areas of the country (unspecified). No study was conducted in the Northern Cape Province. Nine studies had a cross-sectional design, two a case-control design and five were prospective cohort studies of which one only reported cross-sectional results (Table 2).[211] No time-series or any ecological study was located.

All studies investigated the impact of air pollution on respiratory health. The studies also focused on birthweight, learning difficulties, immunological, haematological conditions, gastrointestinal, dermal and ocular conditions. Eight studies addressed chronic respiratory health effects, whereas fifteen studies investigated acute respiratory health effects.

Health effects were assessed mainly by an indirect approach using questionnaires. The health and exposure questionnaire used by Terblanche et al and Richards et al consisted of elements similar to those adopted by the American Thoracic Society, the Harvard Six Cities Air Pollution Health Study and the Canadian Health and Welfare questionnaires.[220-224,227,228] The VAPS questionnaire was pilot tested and evaluated by several international and national experts before distribution.[220] Sanyal and Maduna used a questionnaire similar to that of the VAPS study.[225,228] The BTT health and exposure questionnaire was based on the European Longitudinal Study of Pregnancy and Childhood.[211,212,229] Appropriate questions were added to the questionnaire. Nriagu et al used a modified version of the questionnaire recommended by the WHO for asthma studies.[215,230,231] Coetzee et al used a modified version of a British questionnaire.[219,232,233] Klopper et al used an adapted version of the American Thoracic Society and the National Heart and Lung Institute Division of Lung Diseases.[206,234] The Port Elizabeth Household Environment and Health study was part of a four city comparative household environment and health series involving Jakarta, São Paulo and Accra.[202] The survey instrument used was based on questionnaires used in the other international cities, with adaptations to the local context.[202]

Ehrlich et al used the internationally standardised questionnaire from the International Study of Asthma and Allergies in Childhood (ISAAC) study.[208,235] In another study, Ehrlich et al used the data collected during the 1998 SADHS.[209,218] The 1998 SADHS questionnaires were translated into nine of the eleven official languages of South Africa, checked by back-translation and pretested as part of a pilot study.[218] Many studies did not use a standardised questionnaire.[207,210,214, 216,217]

Some studies conducted the following lung function tests: FEV_1, FVC, PEV, FEV_{50}, VC, FMEF, FMFT, FEF, PEF and PNIF (Table 2). Wesley and Loening used laboratory tests for the specific investigation of the severity of pneumonia infection.[213] Richards et al measured cotinine levels and conducted immunological and haematological tests.[224] Other studies used data from general practitioners and hospitals in their health assessment.[216,219]

Table 2
Summary of South African air pollution epidemiological studies

References	Study design	Study population *Age, race group, province*	Exposure assessment	Health outcomes	Results
Thomas et al[202]	Cross-sectional	<6 years, African/Black, White, Indian/Asian and Coloured, Eastern Cape Province	Indirect using standardised questionnaires	Acute respiratory health, gastrointestinal, dermal and ocular conditions	Significant
Klopper et al[206]	Cross-sectional	All ages, Race not specified, Western Cape Province	Outdoor quantitative measurements	Chronic and acute respiratory health	Insignificant
Zwi et al[207]	Cross-sectional	10–11 years, White, Gauteng, Mpumalanga, Northwest Province, Limpopo Province	Indirect using non-standardised questionnaires	Chronic and acute respiratory health, lung function tests (FEV_1, FVC, VC, FMEF, FMFT, FEF, PEF)	Significant
Ehrlich et al[208]	Cross-sectional	7–9 years, Coloured, Western Cape Province	Indirect using standardised questionnaires along with biomonitoring	Chronic and acute respiratory health	Strong significant
Ehrlich et al[209]	Cross-sectional	≥ 15 years, African/Black, White, Indian/Asian and Coloured, Nationwide	Indirect using standardised questionnaires	Chronic and acute respiratory health, lung function tests (PEF)	Strong significant
Von Schirnding et al[210]	Cross-sectional	<5 years, Coloured, Major urban areas of South Africa	Indirect using non-standardised questionnaires	Acute respiratory health	Significant
Von Schirnding and Mokoetle[211]	Cross-sectional analyses	6 months, African/Black, White, Indian/Asian and Coloured, Gauteng	Indirect using standardised questionnaires	Acute respiratory health	Insignificant
Wesley and Loening[213]	Prospective cohort	<3 years, African/Black, KwaZulu-Natal	Indirect using non-standardised questionnaires	Acute respiratory health	p-value or CI not quoted
Dudley et al[214]	Case-control	<5 years, Race not specified, Western Cape Province	Indirect using non-standardised questionnaires	Acute respiratory health	Significant

Table 2 *(continues)*
Summary of South African air pollution epidemiological studies

References	Study design	Study population *Age, race group, province*	Exposure assessment	Health outcomes	Results
Nriagu et al[215]	Cross-sectional	Children (<17 years) and adults (≥ 17 years), African/Black, White, Indian/Asian and Coloured KwaZulu-Natal	Indirect using standardised questionnaires	Chronic and acute respiratory health	Significant
Mzileni et al[216]	Case-control	>45 years, African/Black, Limpopo Province	Indirect using non-standardised questionnaires	Chronic respiratory health	Significant
Kossove[217]	Cross-sectional	<13 months, African/Black, KwaZulu-Natal	Indirect using non-standardised questionnaires	Acute respiratory health	Insignificant
Coetzee et al[219]	Cross-sectional	9–11 years, Race not specified, Free State Province	Indirect using standardised questionnaires and outdoor quantitative measurements	Chronic and acute respiratory health, lung function tests (FEV_1, FVC, PEV, FEV_{50})	Insignificant
Terblanche et al[220–223]	Prospective cohort	8–12 years, African/Black and White, Gauteng	Indirect using standardised questionnaires along with outdoor, indoor and personal quantitative measurements	Chronic and acute respiratory health, lung function tests (PNIF)	Strong significant
Richards et al[224]	Prospective cohort	14–18 years, Race not specified, Gauteng	Indirect using standardised questionnaires along with biomonitoring	Acute respiratory health, birthweight, learning difficulties, immunological, haematological conditions, lung function tests (FEV_1, FMEF)	Significant
Sanyal and Maduna[225]	Prospective cohort	<14 years, Race not specified, Eastern Cape Province	Outdoor quantitative measurements	Acute respiratory health	p-value or CI not quote

Coetzee et al and Zwi et al placed more emphasis on health assessment than on exposure assessment.[207,219] Fifteen studies used questionnaires to collect indicators of air pollution exposure (Table 3).

Table 3
Indicators for air pollution exposure in South African air pollution epidemiological studies

Indicator	Reference
Household fuels	
Type of fuel used	Dudley et al[214], Mzileni et al[216], Wesley and Loening[213], Kossove[217]
Access to electricity	Von Schirnding et al[210]
Type of cooking fuel used	Von Schirnding et al[210], Von Schirnding and Mokoetle[211], Terblanche et al[220–223], Thomas et al[202], Ehrlich et al[208,209]
Type of heating fuel used	Von Schirnding et al[210], Von Schirnding and Mokoetle[211], Terblanche et al[220–223], Ehrlich et al[208,209]
Duration of exposure to fuel smoke	Kossove[217]
Place where meals are usually cooked	Thomas et al[202]
Cooking time in minutes by fuel type	Thomas et al[202]
Home design	
Presence of a chimney	Von Schirnding et al[210]
Ventilation in cooking area	Thomas et al[202]
Tobacco smoke	
Smoking status of respondent	Mzileni et al[216], Nriagu et al[215], Richards et al[224]
ETS exposure	Nriagu et al[215], Kossove[217], Von Schirnding and Mokoetle[211], Thomas et al[202], Ehrlich et al[208,209], Wesley and Loening[213], Coetzee et al[219], Zwi et al[207], Richards et al[224]
Types of tobacco smoked	Terblanche et al[220–223]
Other indicators	
Asbestos exposure	Mzileni et al[216]
Occupational exposure	Mzileni et al[216], Ehrlich et al[208,209]
Use of insect coils and pump stray insecticides	Nriagu et al[215]
Pets in home	Nriagu et al[215]
Home dampness	Nriagu et al[215]
Residence of the participant in the control or study area	Zwi et al[207], Richards et al[224]
General housing conditions	Von Schirnding and Mokoetle[211], Terblanche et al[220–223]
Burning of garbage	Thomas et al[202]

Four studies measured outdoor air pollution directly. Klopper et al measured 72-h averages for SO_2 and particulates (soiling index) at 14 stations in Cape Town and 1-h values of SO_2, NO_x, O_3, wind speed and wind direction in Edgemead.[206] The authors also monitored SO_2 at Tableview. What was monitored at Lakeside was unclear. Sanyal and Maduna conducted 6-h continuous measurements of CO, NO_2 and SO_2.[225] Coetzee et al measured smoke and SO_2, but did not stipulate the

frequency and duration of the measurements.[219] Terblanche et al measured SO_2, NO_x, CO, O_3, non-methane hydrocarbons (NMHC), hydrogen sulphide (H_2S), TSP, $PM_{2.5}$ and PM_{10}.[220-223] Most of these pollutants were measured at six fixed sites—namely Makalu South of Sasolburg, Sasolburg (in a residential and an industrial area), Vanderbijlpark, Three Rivers and Sharpeville.

The first study to integrate direct and indirect measurements was the VAPS study, which monitored indoor and personal air pollution exposure.[220-223] In that study, Terblanche et al measured TSP indoors in 10 electrified homes, consisting of 12-h TSP personal measurements. Two studies conducted biomonitoring.[208,224] Biomonitoring included measurements of urinary cotinine levels and vitamins C and E levels in plasma, along with immunological and haematological measurements.[224]

Three studies established a strong significant relation between air pollution indicators and deteriorated human health (Table 2). Seven studies established a significant link between air pollution indicators and disease. Four studies found no significant link between air pollution indicators and disease. The lower limit of many of the association measures of some of the reviewed studies is more or less one.[207,210,214] Two studies mentioned a correlation between air pollution indicators and disease, but did not quote the p-value or the CI of the association measure.

2.9 Discussion

The majority of the studies reviewed above are fraught with systematic and random errors, hence limiting their validity and precision. Most studies to date have been observational rather than interventional studies, which could ultimately result in stronger evidence on the nature of the relation between air pollution and health. Other limitations include a lack of detailed and systematic pollution and/or exposure measurement, as well as variations among studies regarding the way that disease outcomes are defined and cases found.

Health effects were assessed mainly by an indirect approach, using a non-standardised questionnaire in one-third of the studies, which can be laden with information bias. Such bias can be minimised by using standardised questionnaires that would enforce uniform definitions upon participants of different studies. Neglecting to control for information bias limits the internal validity of a study.

Most studies that included lung function tests did not consider the temporal variability of lung function measurements (for example, morning, afternoon, evening PEF readings). Quality assurance was very low for most studies, resulting in high measurement variability.

Most studies administered questionnaires to collect information on indicators of exposure to air pollution. For this reason, no study has established exposure-response curves for the criteria pollutants (PM_{10}, lead, SO_2, NO_2, O_3, CO), therefore, using the results of such studies in risk assessment studies is impossible. Only four studies measured outdoor air pollution directly, but did not use the exposure data optimally. The exposure data were categorised and then linked to health data. Smith-Sivertsen et al reiterated that a careful assessment of pollution exposure is vital because it (a) enables assessments of dose-response relations, (b) reduces the chance of misclassification due to differences in air pollution sources and time-activity patterns and (c) enables comparisons to exposure in other parts of the world.[74]

Some studies have dealt inadequately with confounding factors, such as fertiliser use and outdoor air pollution sources. The preferred method for controlling confounding in the studies was during statistical analysis by stratification. In general, very few studies used multivariate analysis as a method for controlling confounding—for example, the two studies by Ehrlich et al.[208,209] Morbidity and mortality are caused by a complex network of risk factors, such as malnutrition, rapid urbanisation, HIV/AIDS and TB. It may thus be tricky to recognise the signal attributable to air pollution beside a myriad of opposing causes of disease and death.

Although the lower limit of many of the association measures of some of the studies reviewed here is more or less one, the impact might still be relevant due to the vast number of people exposed to high air pollution levels in the country, the possible synergy with numerous risk factors and the increased vulnerability of particular subpopulations.[207,210,214]

Virtually no study stipulated the selection process of participants, which limits the external and internal validity of the results. Most studies did not include sample size calculations nor did they discuss the limitations of their sample sizes. It is

anticipated that the sampling variability will be quite high and consequently the random error. The influence of these errors on the association measures is unpredictable.

Some studies were not designed specifically to investigate air pollution indicators and associated health outcomes. Air pollution indicators were then treated as confounders in the statistical analyses. Having reviewed the studies conducted in South Africa, we found it obvious that air pollution epidemiological studies must be better planned and executed to notice the health impacts of air pollution in this country. This deficiency was highlighted by local researchers more than a decade ago.[236] It is also necessary to build capacity in air pollution epidemiology in the country while addressing identified research gaps and priorities. A country's ability to develop more sustainably depends on the capacity of its people and institutions to understand complex environmental and development issues to make the right development choices. The Foresight Series by the South African Department of Arts, Culture, Science and Technology pointed out a general lack of highly trained experts in environmental epidemiology in the country.[237]

Given the tremendous health impact of air pollution exposure and the need for local risk estimates to be applied in burden of disease calculations, the lack of funding for air pollution epidemiology studies in the country, priorities must be assigned regarding which health outcomes should be the primary focus. Internationally, approximately 30% of the research budget is spent on health-related research, compared with 5% in South Africa.[238] In South Africa, detailed air pollution epidemiological studies are competing for limited funds against common diseases of pressing current importance (such as HIV/AIDS, malaria, TB). Local researchers Thomas et al further emphasised the necessity for policies to be enlightened by research highlighting the connection between environment and health.[239] A study conducted in 1991 by the South African Medical Research Council (MRC) for the Henry J. Kaiser Family Foundation highlighted the deficiencies in public health research, particularly with regard to policy-directed health systems research.[238,240,241]

On the one hand, we have indoor air pollution research gaps that have been identified in the international literature and on the other hand, local prevalent health outcomes. Twenty health priority areas were identified by the South African

Essential National Health Research (ENHR), of which five corresponded with those identified by a WHO report, Smith, Ezzati et al, Bruce et al and Chimere-Dan et al — injuries, TB, cancer, respiratory infections and perinatal conditions.[84,134-136,238] Yach et al, however, addressed the methodological difficulties in undertaking epidemiological studies in developing countries.[242] The authors pointed out the use of ecological and cross-sectional studies in determining the relation between risk factors and disease and consequently applying detailed analytical studies to determine the reasons for these relations.

Local researchers Sitas and Thompson discussed the value of ecological epidemiological studies in developing countries, pointing out that although retrospective case-control or prospective (follow-up) studies are important epidemiological tools and have provided useful information on exposure disease associations, their application is inadequate in developing countries with limited research funds.[243] These study designs are also sometimes implemented without acknowledging their limitations. These limitations are exacerbated when measures of exposure and disease are based on single measurements and when the population under investigation is homogenous with regard to exposure. The former is responsible for regression-dilution bias (under-estimation of effect) and the latter for a lack of contrast between exposure groups. Both limitations would attenuate any possible exposure disease relation. Ecological epidemiological studies, which are weaker in design, might offer advantages when conducted in a number of areas of varying exposure proportions and disease rates. Time-series studies, given their limitations, are still relatively easy and economical to conduct, especially in a resource-stricken country like South Africa. The lack of this design in local air pollution epidemiological studies is most likely the absence of an electronic health data management system.

Given the endeavour of epidemiology, it is important for epidemiologists to be vociferous about the ultimate roots of exposure to risk factors, which has a negative impact on wellbeing. Such distant causes include the drivers (such as poverty) and pressures (use of low cost, low efficiency, highly polluting cooking and heating fuels) that influence the state of the environment, ultimately leading to detrimental health effects. The driver of why so many people are not using available electricity in the country is poverty. As alleviating poverty completely in South Africa in the near

future as well as providing everyone with formal housing are economically impossible, other more realistic and financially feasible interventions should be sought, such as communicating the appropriate health promotion messages to the public. Such messages should address feasible technical and socio-behavioural interventions.

Analytical studies should not merely re-document the impact of known risk factors but also should provide a basis for designing technical or socio-behavioural interventions, like the study by Smith-Sivertsen et al.[74] This group conducted the very first ever published randomised control intervention trail in a poor rural community in Guatemala. The intervention involved replacing open fires with new chimney stoves burning the same wood fuels. The authors also reiterated that if the randomisation is successful, then the problem of confounding is eliminated. Randomised trials have the one design potentially providing the strongest evidence of causation. This approach will define the relation between exposure and disease more completely and show the benefit of potential interventions more convincingly.

Rothman, Smith and Ezzati et al, however, debated the applicability and ethical issues of the randomised assignment of individuals into groups having different types of environmental exposures.[11,135,136] Households can be randomised on an intervention (such as improved stoves, cleaner fuels or socio-behavioural changes) and an acute health outcome rate can be monitored to track any reduction. Smith pointed out, however, that envisioning a double-blind study (placebo stoves, cleaner fuels, or socio-behavioural changes) is difficult.[135] Such intervention trials are not practical for health outcomes having a long latency period (such as TB, lung cancer, COPD, CVD). Furthermore, the benefits of randomisation are drastically reduced when the number of randomly assigned units is small, such as when communities rather than individuals are randomised for ethical reasons. Also noteworthy is that acceptance of causation and the need for action has not depended on such randomised trials in the case of most environmental pollutants of issue today, such as active tobacco smoking.

Local researchers Barnes and Mathee briefly reviewed the sustainability of technical interventions and summarised main thoughts on socio-behavioural actions that hamper the use of cleaner fuels for cooking and heating.[244] Globally, technical

interventions to reduce exposure to indoor air pollution have dominated strategies. Technical interventions have either focused on improving existing appliances (for example, provision of improved chimneys), introducing new technologies (for example, new braziers for space heating), or promoting cleaner, more efficient fuels (for example, electricity). The latter type of intervention has been given prominence because it has the potential for improving health. Nevertheless, many of these technical interventions have proven to be unsustainable mainly because of irregular, low, or non-existent household income along with the secondary costs of electrical appliances.[84,245-247] Households generally use electricity for lighting, refrigeration and entertainment appliances, but less for cooking and space heating, which have the greatest implications for indoor air pollution and respiratory health outcomes.[248] In a local survey, continuing use of coal for space heating and cooking was reported for 48% and 45% of electrified households, respectively.[223] Another local study reported that ~4 years after being supplied with electricity, 89% of households had never used electricity for space heating and 61% of households had never used an electric stove.[248] Banks et al have also identified certain social and cultural determinants as important reasons for the delay in the use of electricity, such as socialising, communal cooking, sharing of resources when buying fuels and the chauvinistic perception that the presence of electricity in the home makes women lazy.[249]

Promoting the Basa Njenga Magogo (BNM) method (literally meaning 'make fire like the granny') is part of the low-smoke fuel strategy of the South African Department of Minerals and Energy.[250] This intervention is part of a 10-year project targeting 1 million homes in the winter coal-burning areas. BNM is the local name for the so-called Scotch method of lighting a coal fire by inverting the contents, so that the volatiles are burned off first. The method involves starting a fire from the top-down compared with conventional methods (bottom-up). This approach dramatically reduces the time during which a fire produces smoke and creates a slower-burning fire in a matter of minutes, reducing energy consumption by up to 30% or more. Furthermore, a recent report documented that implementing the BNM method saves a household ZAR26/month (~US$4/month), a considerable saving for the unemployed.[251] Furthermore, the very first Energy Efficiency Strategy for the country sets an overall national target for energy efficiency improvement of 12% by 2014 and 10% by 2014 for the residential sector.[250]

The BNM approach reduces only particulate emissions, however. Particulate emissions from such fires were between 8% and 28% lower than from fires using the conventional method of lighting the fire (using the same coal).[251] Sulphur dioxide emissions from both methods (using low-grade coal) were identical.[252] Comparing the particulate and SO_2 emissions to health guideline values is not possible, due to the method of determining the emission rates. Nevertheless, the health effects are anticipated to be fewer because particulate exposure is lower and risk is a function of exposure. Conventional bottom-up ignited fires have a longer period (~10 minutes) before peak CO concentration is reached (~650 ppm). The concentration then remains for ~30 minutes between 300 and 450 ppm before decreasing to ~150 ppm. With the BNM method, the CO concentration peaks within ~3 minutes at ~500 ppm, remains at this concentration for another 2 to 3 minutes, after which it decreases rapidly to ~150 ppm.[251] This procedure thus lowers the exposure time considerably, therefore it can be considered as having lower risk to human health. In both methods, however, the hourly WHO standard for CO of 35 ppm was still exceeded.[28] Thus, it is imperative that other interventions, such as building homes with chimneys or socio-behaviour changes, must be implemented with the BNM method to reduce exposure to gaseous pollutants (such as CO, SO_2, VOCs).

Besides a few studies that have used education strategies as part of broader programs to reduce acute lower respiratory infections, very little is known about the effectiveness of behavioural interventions to reduce childhood exposure to the dangerous pollutants produced by the indoor burning of wood, coal and paraffin.[253-256] Based on these research gaps, local researchers recently conducted a socio-behavioural intervention study to identify possible target behaviour change interventions to reduce child exposure to indoor air pollution.[257,258] The group recommended four behavioural interventions, namely

- to improve stove maintenance practices,
- to increase the duration that two ventilation sources are opened while a fire is burning,
- to reduce the time that children spend close to burning fires and

- to reduce the duration of dirty fuel burning.

The group consequently investigated the acceptability (willingness to try) and feasibility (ability to perform) of the four indoor air pollution reduction behaviours.[257] The study further aimed to identify the motivations for and barriers against modifying the behaviours, the perceived impact of the behaviours on children's respiratory health and the family's intention to continue with such behaviours. Thirty families in a rural village of South Africa practiced one or more of the behaviours over a 4-week trial period during winter 2002. Improving stove maintenance and reducing the duration of dirty fuel burning proved to be very difficult for most families. The researchers therefore recommended that the main intervention should focus on improving child location and ventilation practices. Nevertheless, as pointed out previously, in 2003, 25% of 44 million South Africans lived in informal housing and ~50% of 11 million households used dirty fuels for space heating.[197] Thus focusing only on child location and ventilation practices would be inefficient.

Many factors influence the perception of risk, such as latency of health effects, media awareness and the level of certainty.[24] The multi-cultural dimension of the South African society represents a particular challenge for developing culturally appropriate health promotion messages for intervention implementation. A significant 4.6 million South Africans aged 20 years and older have no formal schooling, with an additional 4.1 million having some primary school education.[196] Thus, more than 8 million South Africans may not be able to benefit from health promotion material that is designed for the more-educated population. A lack of appropriate health promotion messages may be a reason why the BNM method was promoted in the 1980's with limited success.

The intervention study by Smith-Sivertsen et al highlights the importance of planning well in advance.[74] This study took 8 years of planning, cost over US$ 1.8 million and involved collaboration from various research institutions.

2.10 Conclusion and Recommendations

The case is strong for acknowledging the large public health risk arising from indoor and outdoor air pollution exposure in South Africa. Nevertheless, the majority of

the 16 local air pollution epidemiological studies that have been reviewed, are fraught with systematic and random errors, thus limiting their validity and precision. Yet, the studies do provide some evidence of associations with a range of serious and common health problems.

Research is one of the most important tools for health improvement. We recommend conducting a quantitative intervention study with an analytical study design in all major cities in the country where people are still using dirty fuels for cooking, lighting and space heating. Such a study could address any of the following health outcomes: injuries, TB, cancer, respiratory infections, or perinatal conditions. Ideally, the study should include a comprehensive exposure assessment with outdoor, indoor and personal measurements for CO, SO_2, NO_2, O_3 and $PM_{2.5}$. From these measurements, local exposure models can be derived and validated in future studies. Interventions that could be addressed include technical or socio-behavioural, such as the BNM method coupled with opening windows/doors when cooking, thereby preventing vulnerable groups (such as children and pregnant women) from being exposed to smoke from the dirty fuels. More research is needed on establishing socio-behavioural interventions when using dirty fuels for lighting and space heating and not just for cooking. Future studies must be planned far in advance.

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