Probing secondary exposure and health data as a tool to improve public health in South Africa

by

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DECLARATION

I, Janine Wichmann, declare that the dissertation/thesis, which I have submitted for the degree Doctor of Philosophy at the University of Pretoria, is my own work and has not previously been submitted by me for a degree at this or any other tertiary institution.

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Janine Wichmann Date

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Commissioner of Oaths Date

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ABSTRACT

The usefulness of secondary exposure and health data to improve public health in South Africa will be assessed. Given the tremendous health impact of air pollution exposure the focus of this thesis is on primary prevention, that is the identification of outdoor air pollution and the use of dirty fuels (wood, animal dung, crop residues, coal, paraffin) for cooking and heating as risk factors, whilst controlling for confounding. Hourly averaged outdoor PM_{10} mass, NO_2, NO, SO_2, O_3 data (1 August 1998 - 31 July 2003) from Cape Town were analysed in a nonparametric Spearman's Rho correlation analysis to determine the seasonal spatial correlation between the monitoring sites. Trend and descriptive analyses were conducted on the outdoor hourly and daily PM_{10} mass data to investigate the current and future health implications in the Khayelitsha sub-district, Cape Town. The 1998 South African Demographic and Health Survey (SADHS) data were analysed. The survey involved 13 826 individuals in 12 763 households. Univariate and multivariate logistic regression analyses generated crude and adjusted odds ratios and 95% confidence intervals in order to assess the influence of dirty fuel use for cooking and heating on adult (> 15 years) respiratory health, childhood (< 59 months) respiratory health and 1-59 month mortality. It was found that outdoor air pollution is not homogenously distributed in Cape Town during all seasons. Elevated PM_{10} mass concentrations are frequently present in the Khayelitsha sub-district. There is a strong case for acknowledging the large public health risk arising from air pollution exposure in South Africa, despite the limitations of the 1998 SADHS data. Not much progress has been made in air pollution epidemiology in the country during this investigation due to the identified limitations. Secondary exposure and health data are thus only useful in improving public health in South Africa by supplying baseline data for trend analysis or hypotheses generation. It is recommended that the country must develop environmental public health tracking networks, which incorporates various data sources from multi-sectoral collaborative intervention projects with analytic study designs, in all major cities in the country.

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SUMMARY

The hypothesis that air pollution (using secondary exposure and health data) poses a significant potential risk on human health (specifically respiratory health of people >15 years and <5 years as well as 1-59 month mortality) in South Africa will be tested. The focus of this thesis is on primary prevention, that is the identification of outdoor air pollution and the use of dirty fuels (wood, animal dung, crop residues, coal, paraffin) for cooking and space heating as risk factors, whilst controlling for confounding. Daily averaged (24-h) outdoor PM_{10} mass, NO_2, NO, SO_2, O_3 data (1 August 1998 - 31 July 2003) from Cape Town were analysed in a nonparametric Spearman's Rho correlation analysis to determine the seasonal inter-site correlation between the monitoring sites. Trend and descriptive analyses were conducted on the outdoor hourly and daily PM_{10} mass data to investigate the current and future health implications in the Khayelitsha sub-district, Cape Town. Limitations in the current air quality monitoring network of Cape Town were addressed. The 1998 South African Demographic and Health Survey (SADHS) data were analysed in more detail than reported in the SADHS report. The survey involved 13 826 individuals from 12 763 households. Univariate and multivariate logistic regression analyses were used to generate crude and adjusted odds ratios and 95% confidence intervals in order to assess the influence of dirty fuel use for cooking and heating on adult (> 15 years) respiratory health, childhood (< 59 months) respiratory health and 1-59 month mortality. Limitations, such as inadequate control of systematic and random errors, of the 1998 SADHS were compared to those from other local air pollution epidemiological studies. It was found that 24-h averaged outdoor air pollution is heterogenously distributed in Cape Town during all seasons. Elevated PM_{10} mass concentrations are frequently present in the Khayelitsha sub-district. There is a strong case for acknowledging the large public health risk arising from air pollution exposure in South Africa, despite the limitations of the 1998 SADHS data. Not much progress has been made in air pollution epidemiology in the country during this investigation due to the identified limitations. Secondary exposure and health data are thus only useful in improving public health in South Africa by supplying baseline data for trend analysis or hypotheses generation. It is recommended that the country must develop environmental public health tracking networks, which incorporates various data sources from multi-sectoral collaborative intervention projects with analytic study designs, in all major cities in the country.

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INTRODUCTION

I.I 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 five years of age, who account for about 10% of the world's population.[2] The 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 people exposed to unsafe indoor air pollution levels exceed those exposed to unacceptable outdoor air pollution levels in all of the world's cities collectively.[4] Air pollution is the largest single environment-related cause of ill health among children in most countries.[1,5] In other countries it is the second, after the scarcity of safe water. 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. Distinction is also made between biomass fuels or biofuels and fossil fuels. Biomass fuels comprised 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 refer to any carbon containing fuel e.g. 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.

Despite all the shortcomings of epidemiological studies, they are important in linking exposure to human health *directly*.[7-22] The ultimate endeavor of epidemiology is to identify modifiable determinants of disease occurrence and progression and to contribute in testing the efficacy and effectiveness of interventions on these determinants including the health services. Environmental epidemiology is specifically focused at linking environmental exposure to human health. It is the study of the distribution of health-related states or events in

specified populations in relation to determinants/hazards in the living environment of these populations and the application of this study to the control of such hazards.[23,24] The term "environment" comprises everything that is not genetic, such as diet, smoking and even exercise. However, environmental epidemiology has a more restricted connotation, referring to those environmental factors that are outside the immediate control of the individual, such as active smoking. 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. and Beaglehole et al. in this regard.[23-25]

The reader is referred to Chapter 2 for an introduction on the chemical properties of air pollutants and their environmental fate and transport, exposure assessment, adverse health effects from mainly outdoor low levels of air pollution, health evidence and research needs for indoor air pollution and the relevance of air pollution epidemiological studies in South Africa.

1.2 Impetus for this thesis

Much of what must be done to prevent various morbidity and mortality outcomes lies outside of the sphere of health care. Therefore interventions should be targeted at risk factors and determinants (primary prevention), rather than only diagnosing and providing medical treatment for those already affected (secondary and tertiary prevention). This is parallel with the ultimate endeavour of epidemiology research: to identify modifiable determinants of disease occurrence and progression and to contribute in testing the efficacy and effectiveness of interventions on these determinants including the health services.[25] Given the tremendous health impact of air pollution exposure, as identified in the previous section, the focus of this thesis is on primary prevention, that is the identification of outdoor air pollution and the use of highly polluting fuels for cooking and space heating as risk factors, whilst controlling for confounding. Motivation for the use of the selected health and exposure data along with the focus on the selected health outcomes in the data analyses will be deliberated in this section.

Unfortunately, despite the large and expanding literature (especially in developed countries), relatively few people have reaped the potential health benefits identified in environmental epidemiology. This situation is worse in many developing

countries, including South Africa, where indoor and outdoor environmental and/or occupational exposures exceed national and international standards or guidelines by a considerable amount. Regrettably very little is done to rectify these trends in the country. Possible reasons are the lack of risk management strategies (such as interventions), failure to extrapolate results from developed to developing countries with complete confidence, weak design and/or implementation of local studies leading to presence of systematic and random errors or even the complete lack of studies conducted in developing country settings.

In an ideal world with unlimited funds it will be possible to conduct the perfect air pollution epidemiological study, which will be able to follow a vast number of people up from the day they were conceived to the day they die with detailed quantitative exposure, health and confounder measurements, whilst implementing interventions derived from the study results during the follow-up period. However, we are living in a world with limited funds for research and more so in the case of a developing country like South Africa, where approximately 5% of the research budget is spent on health related research, compared to 30% in developed countries.[26] In this regard, developing countries face a double hurdle: they often do not have easy access to routinely collected data and they face considerable difficulties in disseminating the information they themselves generate. Thus it is the lack of funding that drives the lack of detailed, accurate and valid information to minimise measurement error and bias in epidemiological studies along with the lack of research capacity to disseminate information to the research community and policy makers.

Local researchers emphasised in 1990 the necessity for policies to be enlightened by research that highlight the connection between environment and health.[27] Other local researchers stressed the fact that most health research concentrates on the identification and description of the problem, whilst action orientated research is neglected.[28] A study conducted in 1991 by the South African Medical Research Council (MRC) highlighted the deficiencies in public health research, particularly with regard to policy-directed health systems research.[26,29,30]

Recognising the importance of health research, the White Paper for Transformation of the National Health Systems in South Africa formally adopted the Essential

National Health Research (ENHR) strategy in 1997 as a mechanism to drive the health research agenda setting.[28,31] The 1996 ENHR Priority Setting workshop was the first attempt in South Africa to develop criteria for priority setting for the country's health research. Recommendations for implementing the ENHR action included promotion and advocacy, a mechanism, priority setting, capacity building, networking, funding and evaluation.

In order for the public to benefit from epidemiological results, these must be translated from theory into public health practice more efficiently. This process requires the epidemiologist to be involved in the process of addressing the solutions to the problems they study. Given the finite resources available to protect health, there is a need to weight different risks and to allocate preventive resources to get the maximum benefit. Population attributable fractions (PAFs), also known as population attributable risks, are useful for estimating the proportion of disease cases that could be prevented if one or more risk factors for that disease were reduced or eliminated.[32-39]

Furthermore, the new National Health Act (Act 61 of 2003) may offer relief to the lack of political commitment to environmental health and improve the dilapidated function – along with air quality measurements - in South Africa.[40] The Act has been developed from the 1996 Draft National Environmental Policy.[41] This policy stipulated the paradigm shift of the old 'health inspector' model to a community development approach in addressing environmental health issues by transferring accountability for most environmental health services from provincial level to metro and district municipalities. Regrettably environmental health is not presently a main concern in municipality budgets.

The science of environmental epidemiology is essential to implement Clause 70 of the new National Health Act (Act 61 of 2003).[40] It stipulates that health research priorities should be identified in the country on the basis of burden of disease, cost-effectiveness of interventions aimed at reducing the basis of burden, the availability of human and institutional resources for the implementation of an intervention at the level closest to the affected communities, the health needs of vulnerable groups such as women, older persons, children and people with disabilities and the health needs of communities.

In order to prioritise on health research needs a country should ideally use risk estimates projected from local epidemiological studies in the calculation of the attributable risk due to a specific risk factor and consequently the computation of the disease burden indicators. Disease burden indicators are expressed as daily adjusted life-years lost due to ill health (DALYs) or years of life lost (YLLs).[3] South Africa, a middle income country, is faced by health risk factors from a First World situation (such as industry, traffic, aging population) along those from a Third World situation (such as domestic burning dirty fuels, poor sanitation, overcrowding). Results deduced from epidemiological studies conducted in developed countries and even other developing countries are not merely applicable in this country. In the South African context economical, social and cultural factors may render the population more vulnerable to increased air pollution exposure, due to factors such as poor hygiene, overcrowding, dusty environments, poor nutrition, open dwellings, outdoor lifestyles and the escalating HIV/AIDS epidemic. It is estimated that 10.5 million lives might be lost to AIDS by 2015 in the country.[42] The reader is referred to Chapter 2 for more information on the vulnerability of the South African society as well as published indoor air pollution concentrations due to dirty fuel use.

However, although locally derived risk estimates is desired for application in local policy decisions, Yach et al addressed the methodological difficulties in undertaking epidemiological studies in developing countries.[43] They pointed out the use of ecological and cross-sectional studies in determining the relationship between risk factors and disease and consequently applying detailed analytical studies to determine the reasons for these relationships. In South Africa, detailed analytic epidemiology studies will have to compete with the demands on limited public and research funds for work on common diseases of pressing current importance, such as HIV/AIDS, malaria, TB. Therefore analytical studies should not merely redocument the impact of known risk factors, but should provide a basis for designing interventions, albeit technical or socio-behavioural. Innovative research options, should therefore be explored to enhance air pollution epidemiology in the country, which in turn may improve public health and inform policy.

Considering the immense health impact of air pollution exposure, the need for local risk estimates to apply in burden of disease calculations and the lack of funding for air pollution epidemiology studies in the country, it is important to prioritise on which health outcomes to focus. On the one hand there are indoor air pollution research gaps identified by a WHO report, Smith, Ezzati et al and Bruce et al and on the other hand the local prevalent health outcomes as identified by the ENHR.[44-47] South Africa, as a developing country, also has an obligation to enhance understanding of health issues related to other developing countries.[48-50] Twenty health priority areas were identified by the ENHR, of which 5 correspond with indoor air pollution research gaps, namely injuries, TB, cancer, respiratory infections and perinatal conditions.[28]

The new National Health Act (Act 61 of 2003)[40] transfers accountability for most environmental health services from provincial level to metropolitan councils and district municipalities. It was therefore logical to approach a local metropolitan council for in depth statistical analysis of its air quality data. The main air pollution hotspots in the country are Durban, Johannesburg, Cape Town, Pretoria, Richards Bay, Witbank and the Vaal Triangle. Cape Town was selected as the city adheres to stringent quality assurance guidelines from the US-EPA when monitoring outdoor air pollution.[51]

Air quality monitoring in the cities of South Africa has been in place for decades, for example in Cape Town it commenced during 1958 with the introduction of the first monitoring station collecting SO_2 and smoke concentration data.[51] Bailie et al pointed out the deficiencies in the Cape Town monitoring equipment and lack of information on trends in photochemical smog levels.[52] They called for an upgrading of monitoring of air pollution in the city and for appropriate steps to prevent its further increase. Progress was made in the mean time.

Various environmental challenges confront the Cape Town area. These are primarily the consequence of the growing population of over 3.15 million people and their concurrent need for infrastructure, housing, employment and education.[51] Outdoor air quality remains a key issue in Cape Town, largely because of the visible air pollution, particularly during March to August - known as the 'brown haze'. The brown haze is associated with calm atmospheric conditions and low level

temperature inversions. It occurs over most of the City and is typically most severe in the morning.

A few years ago the National Association for Clean Air (NACA) commenced a pilot study executed by the Energy Research Institute (ERI) of the University of Cape Town.[53] The key objective of the study was the source apportionment of the brown haze. Conclusions were that small particles are the single largest cause of the visible brown haze; vehicular emissions are accountable for 65% of visible degradation, of which 49% is caused by diesel driven vehicle emissions; industry is a notable source, in particular low level emitting industries, the industrial contribution estimated to be 22%; wood burning and natural sources, such as wind-blown dust and sea salt, contribute very little towards the brown haze and assuming a laissez faire approach, air pollution is projected to escalate by 48% from 1997 to 2007.

Due to the lack of a computerised health data management system in the country, it was not feasible to conduct a time-series study using the high quality outdoor air pollution data from Cape Town. However, as will be addressed in Chapter 2, indoor and personal air pollution concentrations often correlate poorly with outdoor air levels.[54-65] Indoor, personal and outdoor correlations are dependent on the pollutant under investigation. Nevertheless, time-series studies are at least steppingstones to address air pollution epidemiology in the country.

The local MRC was contacted to provide the 1998 South African Demographic and Health Survey (SADHS) data free of charge. The data were analysed in more detail than reported in the SADHS report.[66] It was the first national adult and child health survey conducted across the entire country. Data from this survey provided the opportunity to examine the prevalence and determinants of various diseases in a representative national population rather than a selected high risk population, as has been the case in most previous studies in developed countries.

Studies addressing cardiovascular diseases were identified as one of the indoor air pollution research gaps.[44-47] The 1998 SADHS collected information on the prevalence of heart attacks and various risk factors on demographics, diet, lifestyle and the environment. A preliminary analysis was conducted on the 1998 SADHS data as a pilot study during June 2004. The preliminary results indicated that too

few cases of lung cancer, still births and perinatal deaths were reported. Birth weight data were also missing for the vast majority of participants. However, respiratory ill health is the main reason for use of the health services in the country and is also one of the research priorities identified by the ENHR.[67]

A recent report by Statistics South Africa, listed the ten leading underlying natural causes of death for different age groups during 1997, 1999 and 2001.[68] Infant deaths were mostly due to causes related to the perinatal stage, as also reported in the literature.[69-82] However, between 22.6% and 18.7% of infant deaths were due to unexplained causes. It is likely that some of these premature deaths may be attributed to high indoor air pollution exposure. A local study conducted 15 years ago reported that acute respiratory infections (ARI)(such as pneumonia) were then the principal cause of death amongst young children in large parts of country.[83] This is supported by international findings that ARI are a leading cause of childhood illness and death worldwide, accounting for an estimated 6.5% of the entire global burden of disease[4]. In addition there is no simple and rapid treatment for ARI as is the case with diarrhoeal disease and oral rehydration therapy.

A series of 5 papers on child survival appeared in *The Lancet* during 2003 and described a major public health challenge: more than 10 million children dying each year because they have not been reached by known and effective interventions.[84-88] One of the most challenging Millennium Development Goals (MDGs) is addressing this global public health dilemma, to achieve a 66% reduction in child mortality by 2015 (Goal 4, Target 5).[89] The MDGs adopted by the United Nations in 2000 provide an opportunity for concerted action to improve global health. They place health at the heart of development and establish a novel global compact, linking developed and developing countries through clear, reciprocal obligations. Over the past 20 years, the hazards of indoor air pollution has been documented by a growing body of literature[44-47] but very few studies focused on its impact on infant and child mortality. Another MDG is addressing the proportion of dirty fuel use in countries (Goal 7, Target 9, Indicator 29).[89]

Additionally, asthma and other chronic diseases were the main cause of death of South Africans in 2000.[90] Recent publications highlighted that prevalence rates of chronic respiratory diseases are escalating in developing countries.[91-97] Most

environmental epidemiological studies in South Africa focused on children health.[98-112] TB is also one of the diseases targeted by the MDGs (Goal 6, Target 8): start to reverse the incidence of TB and eradicate the disease by 2015.[89] This thesis therefore placed priority on determining risk factors of adult and childhood respiratory health and 1-59 month mortality.

Demographic and health surveys (DHS) are usually of cross-sectional design. As there is no follow-up, these surveys are less time-consuming and costly than more rigorous prospective cohort studies. Due to the inherent characteristics of health surveys, they do not adhere to some of Hill's causation guidelines (Table 1): temporal relation, reversibility and strong study design.

Table 1 Hill's causation guidelines[25]

Temporal relation	Does the cause precede the effect (essential)
Plausibility	Is the association consistent with other knowledge (e.g. regarding mechanism of action, evidence from experimental animals)
Consistency	Have other studies had similar results?
Strength	What is the strength of the association between the cause and the effect?
Dose-response	Is increased exposure to the possible cause associated with increased effect?
Reversibility	Does the removal of a possible cause lead to reduction of disease risk?
Study design	Is the evidence based on a strong study design?
Judging the evidence	How many lines of evidence lead to the conclusion?

However, the function of a DHS is not to prove causation, but to answer specific questions about the population related to measurements taken at a point in time, to provide information on intercorrelations among variables in the population at that current point in time, to detect high-risk groups, to give hints about causal relations, to generate hypotheses, to provide a baseline for comparisons with future measurements and to measure changes in health and risk factor prevalence rates

through a sequence of surveys.[113,114] The results from these surveys are also important descriptively in health administration, planning and policy analysis as information on disease prevalence is often required to assess the need and demand for health services and to evaluate intervention programs in specific target populations. Thus DHS data are mainly applied in secondary or tertiary prevention.

Although the weaknesses of DHS are known, this thesis will investigate the association between indoor air pollution indicators and various health outcomes nevertheless. During the statistical analyses, various limitations regarding confounding and exposure assessment will be identified and the usefulness of secondary exposure and health data will be assessed. Recommendations will be made how to address these limitations in future SADHS in order to improve the link between indoor air pollution indicators and various health outcomes.

1.3 Research question

Does air pollution (using secondary exposure and health data) pose a significant potential risk on human health (specifically respiratory health of people >15 years and <5 years as well as 1-59 month mortality) in South Africa?

The following hypothesis will be tested:

Air pollution (using secondary exposure and health data) poses a significant potential risk on human health (specifically respiratory health of people >15 years and <5 years as well as 1-59 month mortality) in South Africa.

1.4 Aims

This thesis will attempt to investigate the usefulness of analysing secondary South African air pollution exposure and health data to project preliminary risk estimates for adult respiratory health, under five ARI and 1-59 month mortality due to exposure to indoor air pollution from using dirty fuels for cooking and heating purposes and ultimately improve public health in the country. These preliminary risk estimates will be used to determine preliminary attributable fractions and finally approximate estimates of the number of cases of disease or premature mortality that could be avoided if indoor air pollution due to combustion of dirty fuels for cooking and heating purposes could be completely eliminated in South Africa.

Furthermore, it will contribute to the current body of knowledge of strong evidence (ARI, chronic bronchitis) and moderate evidence (TB) on dirty fuel use. The findings of these analyses will be compared to the status quo of air pollution epidemiology in the country. The use of outdoor air pollution data from Cape Town in future time-series studies will be evaluated. Lastly, the study will investigated the current and future health implications due to outdoor PM_{10} mass exposure in the Khayelitsha sub-district in the City of Cape Town.

The specific aims are:

1. To determine the number of air pollution epidemiological studies conducted in South Africa and critically review them for study design and the strength of their results in linking air pollution exposure to human health.
2. To determine the temporal inter-site correlations of 24-hour averaged outdoor PM_{10} mass, NO_2, NO, SO_2, O_3 and CO concentrations in Cape Town whilst controlling for seasonal effects.
3. To investigate the current and future potential health implications due to outdoor PM_{10} mass exposure in the Khayelitsha sub-district in the City of Cape Town.
4. To determine the potential risk factors for adult respiratory diseases and symptoms in South Africa, whilst controlling for a number of confounders and effect modifiers.
5. To determine the whether the use of wood, animal dung, coal and paraffin for cooking and heating poses a potential risk for acute respiratory infections (ARI) in preschool children (0-59 months) living in South Africa, whilst controlling for a number of confounders and effect modifiers.
6. To determine whether the use of wood, animal dung, coal and paraffin for cooking and heating poses a potential risk for childhood mortality (1-59 months) in South Africa, whilst controlling for a number of confounders and effect modifiers.

1.5 Study design and structure of thesis

Chapter 2 will address the status quo of air pollution epidemiology in the country through a narrative review. In light of the lack of unique South African exposure-

response curves, the next chapter will address the question whether outdoor air pollution is homogenously distributed in Cape Town, South Africa (Chapter 3). This is useful to know when conducting relatively easy and inexpensive time-series studies, which outputs can be used to derive exposure-response curves. If outdoor concentrations of a particular air pollutant are homogenously distributed within a city area there will be inadequate exposure variation. Consequently health data cannot be linked to these outdoor air pollutant concentrations during time-series analyses. Chapter 4 reports on the current and future health implications due to outdoor PM_{10} mass exposure in the Khayelitsha sub-district in the City of Cape Town. Next the 1998 South African Demographic and Health Survey (SADHS) data were analysed in more detail than reported in the SADHS report. This endeavour resulted in the calculation of unique South African risk estimates for adult (15 years and older) respiratory health (Chapter 5), under five respiratory health (Chapter 6) and 1-59 month mortality (Chapter 7) due to exposure to indoor air pollution from using fossil and biomass fuels for cooking and heating purposes. Chapter 8 presents a general discussion on the main results of Chapters 2-7 and their bias and limitations. The final chapter will concentrate on research recommendations and will also look at the application of the results in policy and interventions.

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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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Chapter 3

SEASONAL INTER-SITE CORRELATION AMONG AIR POLLUTION MONITORING SITES IN CAPE TOWN, SOUTH AFRICA

Background: Many pollutants show complex spatio-temporal profiles, which complicates measuring or modelling exposure patterns and obscures subsequent estimation of human exposure. Nevertheless, the majority of air pollution epidemiological studies are based on exposure data from a single central outdoor monitor. This may introduce information bias, which impairs the validity of epidemiological studies. The temporal correlation among outdoor concentrations of a particular air pollutant measured at different sites (inter-site correlation) may be investigated to control information bias. The higher the inter-site correlation, the lower the anticipated information bias when using data from one site for another with similar air pollution sources and the more valid the exposure assessment of the epidemiological study. The majority of the air pollution epidemiological and exposure assessment studies are carried out in developed countries. Very few quantitative exposure assessment studies have been conducted in South Africa. The aim of this study is not source apportionment, but to consider the suitability of information derived from the air pollution monitoring network in Cape Town, South Africa in epidemiological studies. Consequently this study investigated the seasonal inter-site correlation between 24-h average outdoor PM_{10} mass, NO_2, NO, SO_2, O_3 and CO in Cape Town.

Methods: Daily averages (24-h) were obtained from hourly averages measured during 1 August 1998 – 31 July 2003. All 24-h averages are based on at least 20 1-h samples in accordance with the ISO 17025 guidelines. If this requirement was not met, a 24-h average was set as a missing value. All the 24-h average concentrations did not have a normal distribution. Nonparametric Spearman's Rho correlation analysis was conducted. Statistical significance was assessed at 99% confidence level for the correlations. Seasons were defined as winter, spring, summer and autumn for the following periods 1 June – 31 August, 1 September – 30 November, 1 December – 28(29) February and 1 March – 31 May, respectively.

Results: The descriptive statistics of the different air pollutants measured at the sites under investigation are in accordance with the major air pollution sources present in each area. It was found that in general pollutant inter-site correlation coefficients are

not strong during all seasons. On average, the most homogeneously distributed pollutant is NO_2, followed by PM_{10} (including Khayelitsha data), PM_{10} (excluding Khayelitsha data), NO, CO, O_3 and finally SO_2 in the Cape Town air shed. Inter-site correlation coefficients for NO_2, NO, SO_2, CO and O_3 vary from 0.456 to 0.832; 0.212 to 0.791; -0.100 to 0.662; 0.302 to 0.676 and 0.123 and 0.557, respectively. PM_{10} measured at Bothasig, City Centre, Goodwood and Tableview presents correlations from 0.261 to 0.859. The PM_{10} inter-site correlation coefficients between Khayelitsha and the other sites (Bothasig, City Centre, Goodwood and Tableview) vary from 0.396 to 0.769.

Conclusions: The results highlight the importance of properly characterising relations among different outdoor pollution monitoring sites. Studies conducted in locations with strong inter-site correlation coefficients among outdoor pollutant concentrations should not assume that they necessarily persist across seasons. Given the resource stricken research environment in South Africa there is still merit in using NO, NO_2, SO_2, O_3 and CO data from one site for another. This approach will make outdoor air pollution exposure assessment easier during hospital time-series studies. This is however not the case for SO_2 measured at Bothasig and Tableview and for PM_{10} at all sites. Stemming from the preliminary results and associated limitations some thoughts are suggested to the City of Cape Town regarding its air quality monitoring network.

3.1 Introduction

The World Health Organisation reports that 25% of all preventable diseases are due to a poor physical environment.[1] No case in support of environmental action is deeper than that of the need to eradicate health risks. Various epidemiological and toxicological studies have linked air pollution to mortality and various morbidity outcomes. For reviews, refer to Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society, Vedal and Lighty et al.[2-5] However, cohort studies focusing on long-term health effects are still scarce.[6,7]

Once emitted into the environment, air pollutants typically undergo a process of dispersion. Exposure occurs when humans encounter the air pollutants. During dispersion pollutants may undergo many kinds of transformation. Dilution occurs due to the mixture with the transporting medium (e.g. air). Physical processes such as sedimentation and coagulation are the principal factors for causing primary suspended particulate matter to be heterogeneously distributed.[8] Chemical reactions occur, breaking down the original pollutants or converting them into new compounds, which may pose more harm than the original compound. Inert pollutants (e.g. CO) simply disperse after emission, resulting in a concentration gradient with increasing distance to the source.[9] Chemically reactive pollutants (e.g. NO) display a sharper concentration gradient than inert pollutants.[9] The formation of secondary pollutants (e.g. ammonium sulphate and O_3) is a large-scale occurrence and these pollutants are quite homogenously distributed in space.[10] Exceptions to this occur for reactive species in the environs of other reactive species (e.g. depletion of O_3 by NO along busy roads). Diffusion and transport of pollutants are influenced by atmospheric conditions such as wind speed, vertical temperature gradient and solar radiation.[9] As a result many pollutants show complex spatio-temporal profiles. This complexity means that measuring or modelling exposure patterns and subsequently clarifying levels of human exposure can be complicated.

Nevertheless, the majority of air pollution epidemiological studies are based on exposure data from a single central outdoor monitor. This may introduce information bias, which involves misclassification of the study participants with respect to exposure status. The validity of epidemiological studies is impaired by information bias. In order to control information bias the inter-site correlation between outdoor air pollution concentrations within a particular area may be

investigated. The higher the inter-site correlation, the lower the anticipated information bias when using data from one site for another with similar air pollution sources and the more valid the epidemiological study.

Most air pollution epidemiological and exposure assessment studies are carried out in developed countries. It is important to conduct more air pollution epidemiological and exposure assessment studies in South Africa as it is faced by pollution caused by industry (First World situation) and by domestic burning of coal and biofuels (Third World situation). Thus study results (e.g. exposure-response curves) - obtained from developed countries – cannot merely be extrapolated with total conviction to developing countries. The three global factors that directly or indirectly impact on health - the community and social environment, the physical environment and the family and individual environment – are different for developed and developing countries.[11]

Very few quantitative exposure assessment studies have been conducted in South Africa.[12-16] Exposure is usually based on a proxy measure, such as smoking status[17,18] or use of polluting fuels (wood, coal, animal dung, crop residues or paraffin) for cooking and space heating.[19,20]

Air pollution monitoring sites in Cape Town are located in different types of areas. It is therefore anticipated that the inter-site correlation coefficients for the different pollutants will differ. However, it is still necessary to quantify the inter-site correlation coefficients instead of describing the situation qualitatively. Consequently this study investigated the seasonal inter-site correlation between 24-h average outdoor PM_{10} mass, NO_2, NO, SO_2, O_3 and CO in Cape Town, South Africa. The aim of this study is not source apportionment, but to consider the suitability of information derived from the air pollution monitoring network in Cape Town in epidemiological studies.

3.2 Meteorology and Topography

The City of Cape Town was established in December 2000 by the merging of the previous Cape Metropolitan Council and six Metropolitan Local Councils: Tygerberg, Oostenberg, Blaauwberg, South Peninsula, Helderberg and Cape Town. It is the southern most metropolitan area on the African continent and covers an

area of 2487 km^2. The area is encircled by mountains and the Atlantic Ocean. Table Mountain is 1 086 metres above sea level (ASL). It is flanked by Devil's Peak (1000 m ASL), Lion's Head (669 m ASL) and Signal Hill, which precedes the Twelve Apostles (Noordhoek Peak 756 m ASL and Chapman's Peak 592 m ASL) in the mountain chain.

The Peninsula has a Mediterranean climate. Summer is from December to February with temperatures averaging at around 28°C. The prevailing wind during October to March (spring to autumn) is from SSE to SSW. It brings very little rain with it. Occasionally during summer the Cape will experience northerly "berg winds" with associated increase in temperatures. The Southeaster subsides slightly during February and March. The prevailing wind between May and August (autumn and winter) blows from N to NW. This wind is not as strong as the South Easter and occurs less frequently. It precedes a cold front and is therefore followed by much needed rain. The rainy season peaks during June and July. During March to August the area have calm atmospheric conditions and low level inversions.

3.3 Air Pollution Monitoring Network

Monitoring of air quality in Cape Town commenced in 1958 with the introduction of the first monitoring stationing measuring SO_2 and smoke.[21]

Bailie et al pointed out the deficiencies in the Cape Town monitoring equipment and lack of information on trends in photochemical smog levels.[22] They called for an upgrading of monitoring of air pollution in the city and for appropriate steps to prevent its further increase. Progress was made in the mean time.

The City of Cape Town adopted an Integrated Metropolitan Environmental Policy (IMEP) and its implementation strategy on 31 October 2001. The City of Cape Town now, for the first time, has a bold and clear environmental policy. The IMEP addresses key environmental issues and sets out the City's commitment to improving Cape Town's environment. The IMEP has six priority strategies: air pollution, biodiversity, coastal zone, litter and illegal dumping, quality open spaces and noise pollution. During 2002 two additional strategies were initiated: environmental education and energy.

Currently the Cape Town ambient air quality monitoring network comprises 9 continuous monitoring stations operating across the 500km² city area (Figure 1). [21] Two mobile stations are located at Killarney and Plattekloof.

Figure 1 Location of air pollution monitoring sites in Cape Town, South Africa[21]

The two sites at the City Hall and the Drill Hall are close to each other and were grouped together as the City Centre site during this investigation. The City Centre site is located in an urban area next to busy roads (Figure 2). Tableview is located in a residential area, but still relatively close to a busy highway and an oil refinery (Figure 3). Bothasig is located in a residential area, but closer to the highway and oil refinery than Tableview (Figure 3). Goodwood is also located in a residential area, but further from the highways and closer to a light industrial area (Figure 4). Molteno is located in a residential area, quite far from busy roads (Figure 2). Khayelitsha is located the furthest away from

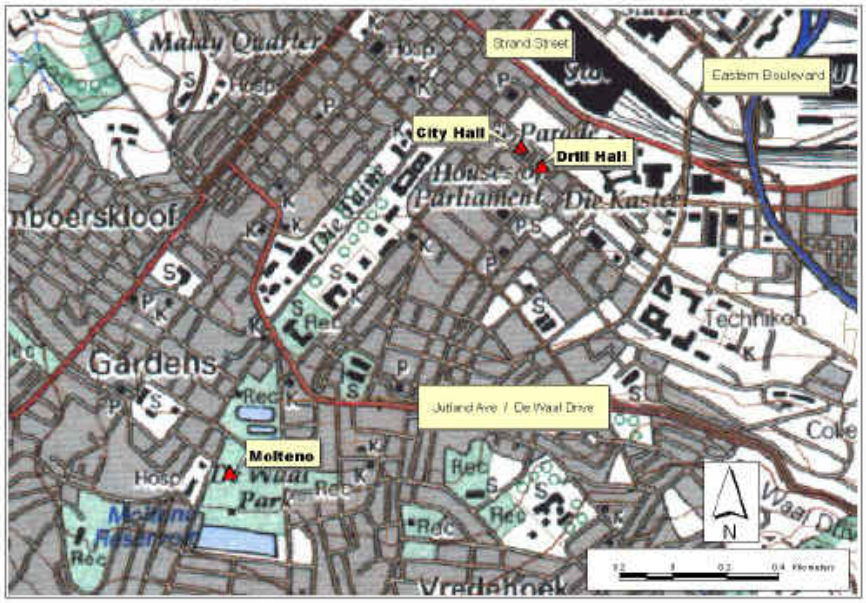


Figure 2 Location of air quality monitoring site in the City Centre and Molteno, City of Cape Town[21]

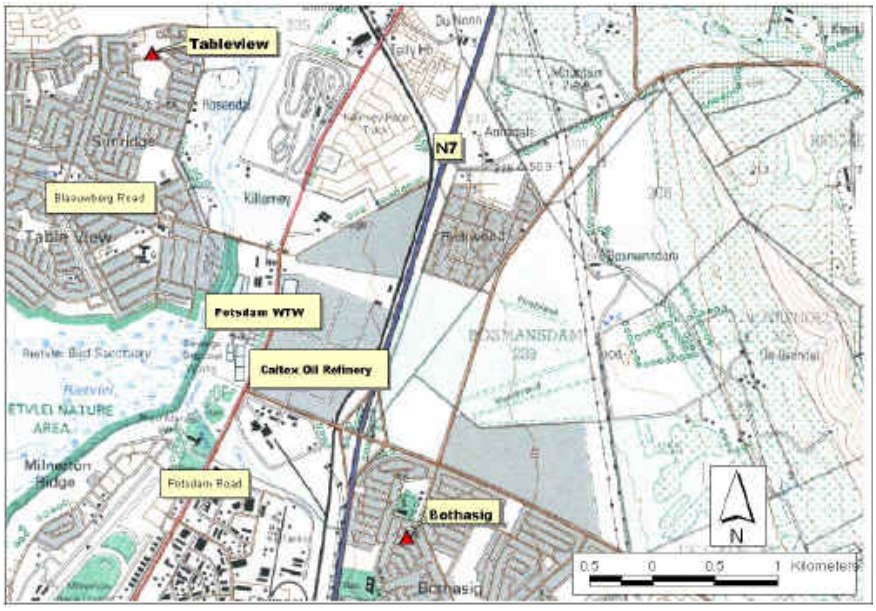


Figure 3 Location of air quality monitoring site in Tableview and Bothasig, City of Cape Town[21]

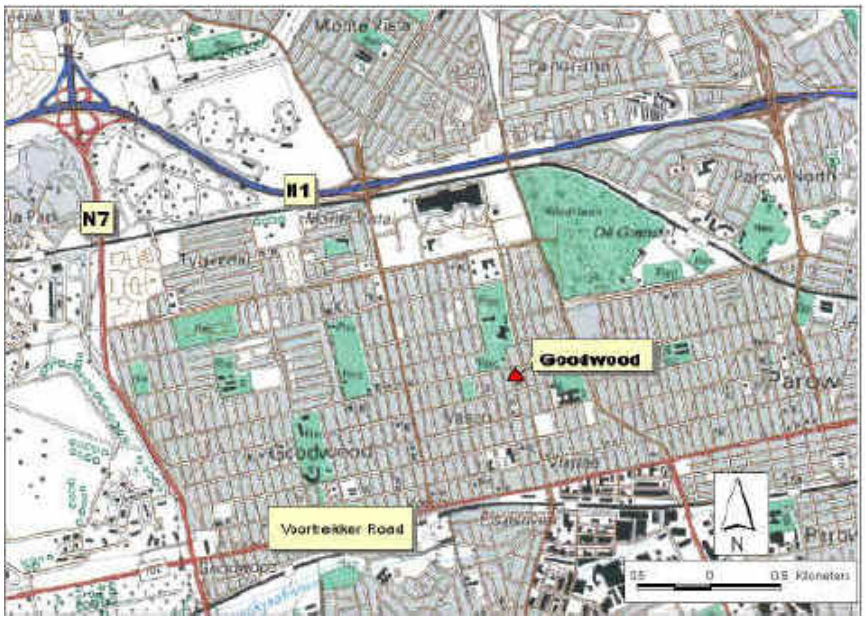


Figure 4 Location of air quality monitoring site in Goodwood, City of Cape Town[21]

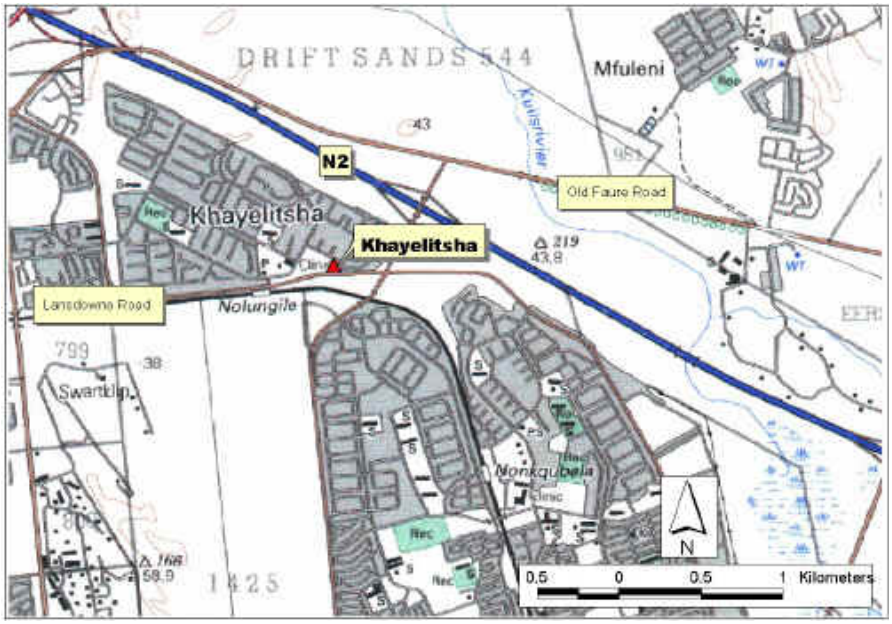


Figure 5 Location of air quality monitoring site in the Khayelitsha sub-district, City of Cape Town[21]

all the other sites (Figure 5) in a poor residential area, but closer to a highway and industries. The City Centre and Molteno sites are separated from the other sites as both are located in the city bowl, which is flanked on three sides by mountains. The location of City Centre and Molteno therefore provides favourable topographical conditions for the accumulation of pollutants over all seasons. All the sites are either downwind or upwind from the local sources as the wind direction changes during seasons (Section 3.2).

Thirty eight analysers, located at these 9 sites, continuously assess real-time concentrations of critical pollutants in the atmosphere using US-EPA equivalent methods in accordance with ISO 17025 guidelines. These instruments measure the concentration of ambient air pollutants in 20 second scans and values can be expressed in short term (10 min), one hour, twenty four hour, monthly or annual averages.

Minimum data completeness requirements for the calculation of period averages issued by the US-EPA in accordance with ISO 17025 guidelines for continuous monitoring are stipulated as follows: 24-h average - requires at least 20 1-h samples; monthly average requires at least 80% of data days of ≥ 20 h; quarterly average requires 80% data capture and no more than 10 consecutive incomplete days and annual average - requires 4 complete quarters.

Pollutants measured include PM_{10} (with TEOM 1400A), SO_2 (with fluorescence analysers), NO_x (with chemiluminescent analysers), O_3 (with UV analysers) and CO (with infrared analysers). The results are posted on the City's web site as soon as the data has been analysed statistically. The site can be visited at www.capetown.gov.za/airqual.

The ambient network is run by the city's Air Quality Monitoring Laboratory, located in Athlone. Here a central 'audit' monitoring station is used to certify transfer standards for calibration purposes. Both locally and internationally certified standards and a complete range of analysers are housed here. The City has two movable monitoring stations. One of these stations, operated by the city in agreement with the local refinery, is positioned downwind from the refinery to determine "hotspots". The stationing is determined in consultation with the local

community. This station is moved every 6 months to a year. A second movable monitor, owned by the council, is used to determine "hotspots" within the City of Cape Town as well as to assess future expansion and location of monitoring stations.

It is important to point out that this study was done independently from the City of Cape Town. The City of Cape Town is responsible for developing an air quality management plan which should be incorporated into its integrated development plan according to the National Environment Management: Air Quality Act (Act 39 of 2004).[23] However, human resources capacity to investigate fundamental issues pertaining to air pollution exposure assessment in detail is not available at local government level.

3.4 Air Pollution Sources

Various environmental challenges confront the area. These are primarily the consequence of the growing population of over 3.15 million people and their concurrent need for infrastructure, housing, employment and education.[21]

Rapid urbanisation and urban growth have resulted in a larger population in Cape Town and this in turn leads to a larger number of people making use of public and especially private transport to commute to work. Due to urban sprawl the distances between work and residences have also greatly increased. During peak periods the capacity of some road networks is exceeded and other road networks are reaching their capacity. One of the reasons for the lack of capacity in the road system is the lack of investment in both road and public transport systems. Currently there are 825 000 registered vehicles in all classes. Vehicle ownership has increased by 45% in the ten-year period 1990-2000. The number of cars has increased to approximately 570 000, doubling in the last 25 years. There are now approximately 178 cars per I 000 people in the City.

During Census 2001 it was established that 80%, 2% and 16% of the nearly 760 000 households in Cape Town use electricity, gas and paraffin for cooking.[24] The corresponding fuel use for heating is 75%, 1% and 19%, with 2% of households using wood. The majority of households use electricity for lighting (89%), followed by paraffin (9%) and candles (2%). However, it is noteworthy to highlight the

different fuel mix used in Khayelithsa. Paraffin, electricity and candles are used for lighting by 22%, 76% and 2% of the nearly 86 000 households in Khayelithsa, respectively.[25] Statistics on the energy use for cooking and heating are not available on a sub-district level. As nearly 100% of people living in Khayelithsa are Black, the energy use profiles for cooking and heating in Black households in the entire City of Cape Town will be quoted here.[25] Race is still presently very much linked to past access to resources, socio-economic status and educational status. The energy use profiles for cooking in Black households in the entire City of Cape Town are as follows: 45%, 4%, 49%, 1% and 1% for electricity, gas, paraffin, coal and animal dung, respectively.[24] The figures for heating are 33%, 1%, 57%, 4% and 4% respectively for electricity, gas, paraffin, wood and other unspecified fuels.[24] The households using wood, coal and animal dung for space heating and cooking contribute to PM_{10} emissions.

Outdoor air quality remains a key issue in Cape Town, largely because of the visible air pollution, particularly during March to August - known as the 'brown haze'. The brown haze is associated with calm atmospheric conditions and low level inversions. It occurs over most of the City and is typically most severe in the morning. Air pollutants (such as SO_2, NO_2, particulate matter and heavy metals) result from combustion processes in industry, services, agriculture, transport and homes.

A few years ago the National Association for Clean Air (NACA) commenced a pilot study executed by the Energy Research Institute (ERI) of the University of Cape Town.[26] The key objective of the study was the source apportionment of the brown haze. Conclusions were that small particles are the single largest cause of the visible brown haze; vehicular emissions are accountable for 65% of visible degradation, of which 49% is caused by diesel driven vehicle emissions; industry is a notable source, in particular low level emitting industries, the industrial contribution estimated to be 22%; wood burning and natural sources, such as wind-blown dust and sea salt, contribute very little towards the brown haze and assuming a laissez faire approach, air pollution is projected to escalate by 48% from 1997 to 2007.

3.5 Statistical Analyses

SAS version 8 was used in the statistical analyses. The data comprised of concentrations averaged at hourly intervals. Table 1 reflects the hourly data

availability during 1 August 1998 - 31 July 2003 for Bothasig, City Centre, Goodwood and Tableview. PM_{10} data availability at Khayelitsha was 26.4% (1998/1999), 73.4% (1999/2000), 56.7% (2000/2001), 16.6% (2001/2002) and 97.1% (2002/2003).

Table 1 Percentage of air quality data availability during 1 August 1998 - 31 July 2003

Station	SO_2	NO_x	PM_{10}	O_3	CO
Bothasig					
1998 – 1999	97.7	84.7	81.3	NR	NR
1999 – 2000	58.6	20.9	13.5	NR	NR
2000 – 2001	93.0	97.2	54.1	NR	NR
2001 – 2002	89.5	97.0	88.4	NR	NR
2002 – 2003	90.8	90.7	82.7	NR	NR
City Centre					
1998 – 1999	89.3	99.4	99.3	NR	10.0
1999 – 2000	97.1	96.8	99.7	NR	66.3
2000 – 2001	96.8	96.7[1]	99.4	NR	96.3
2001 – 2002	97.4	96.3[2]	99.5	NR	96.7
2002 – 2003	99.6	99.8[3]	100	NR	99.1
Goodwood					
1998 – 1999	69.5	70.8	96.2	44.0	10.3
1999 – 2000	82.2	92.7	99.3	98.6	86.1
2000 – 2001	98.9	98.9[4]	99.7	98.7	99.7
2001 – 2002	99.8	99.9[5]	98.0	99.9	99.9
2002 – 2003	98.0	97.7[6]	97.8	96.9	94.0
Tableview					
1998 – 1999	97.4	88.9	96.8	NR	NR
1999 – 2000	86.4	69.9[7]	88.4	NR	NR
2000 – 2001	93.3	74.5	93.6	NR	NR
2001 – 2002	98.3	98.0[8]	97.2	NR	NR
2002 – 2003	98.4	98.6[9]	99.1	NR	NR

Notes: NR: Not recorded, IDS: Incomplete data set, 1: NO_2: 95.9%, NO: 96.6%, 2: NO_2: 96.1%, NO: 96.0%, 3: NO: 99.7%, 4: NO: 99.0%, 5: NO: 99.6%, 6: NO: 98.0%, 7: NO_2: 67.9%, NO: 70.3%, 8: NO2: 95.3%, NO: 97.4%, 9: NO: 97.0%

The corresponding availability for O_3 measured at Molteno was 76.6%, 96.7%, 94.4%, 96.4% and 97.7%. Data from Killarney and Plattekloof were excluded in the analysis. These were mobile stations with responses below 50%. All 24-h averages are based on at least 20 1-h samples in accordance with the ISO 17025 guidelines. If this requirement was not met, a 24-h average was set as a missing value.

An exploratory data analysis was conducted in which the untransformed and log transformed data were tested for normality using the Kolmogorov-Smirnov test at the 95% confidence level. It was concluded that log transformations were ineffective in normalising the data. An alternative for normalising the data was attempted by the culling of extreme values. This is practical as the data remain in their original units. This procedure was also ineffective in normalising the data.

Hence a nonparametric Spearman's Rho correlation analysis was undertaken on the concentration levels of a particular pollutant measured at the same point in time at different stations. Statistical significance was assessed at 99% confidence level for the inter-site correlations due to the large sample sizes. The seasonal fluctuations of the inter-site correlation coefficients were investigated using seasons as a proxy for meteorological factors. Seasons were defined as winter, spring, summer and autumn for the following periods 1 June – 31 August, 1 September – 30 November, 1 December – 28(29) February and 1 March – 31 May, respectively.

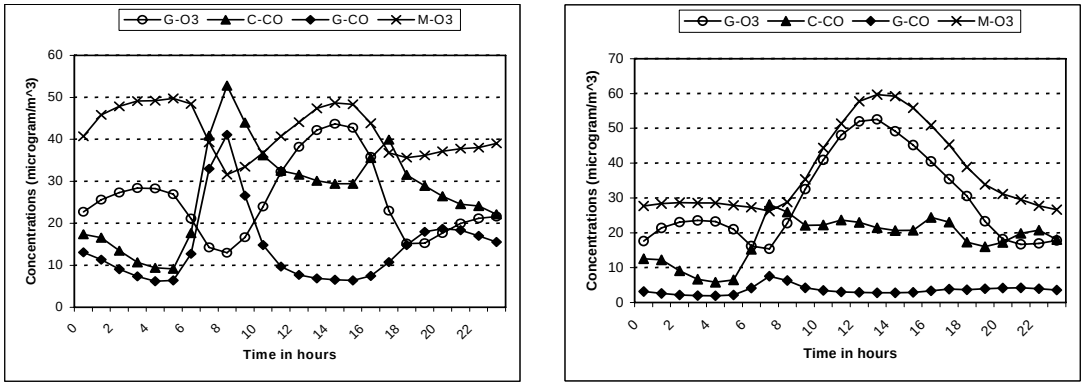
3.6 Results

3.6.1 General Seasonal and Diurnal Trends of Pollutant Concentrations

All the hourly pollutant concentrations were averaged over the five year period to derive Figure 6 and 7. These figures indicate the typical hourly variations of pollutant levels during winter and summer. The CO concentrations are reported as 1/100 of the original values in order to plot them on the same graph as those of O_3. In general the concentrations are highest in winter, decreasing in spring and summer and intensifying again in autumn, except for O_3 (diurnal trends not shown for spring and autumn). O_3 levels peak in spring and summer and were similar during winter and autumn.

Although the focus of this investigation is not source apportionment, it is still clear that the concentrations of NO, NO_2, SO_2, CO and PM_{10} closely follow the diurnal pattern of traffic with peak hour and off-peak hour differences, i.e. the higher levels are usually observed in the morning between 8:00 and 9:00 and the evening rush hours 16:00 to 19:00. However, the second peak for CO, SO_2 and PM_{10} vary between the sites during different seasons. NO_2 levels at City Centre appear to be constant between 8:00 and 18:00. Khayelitsha has a PM_{10} peak between 18:00 and 22:00 during winter, most probably due to the combustion of coal, animal dung and

paraffin for heating. The O_3 diurnal pattern is different from those of the other pollutants, it peaks during 13:00 to 14:00.



a) b)

Figure 6 Averaged 24-h O_3 and CO variations at City Centre (C), Goodwood (G) and Molteno (O) during a) winter and b) summer

Table 2 lists the descriptive statistics for valid 24-h averages of PM_{10}, SO_2, NO, NO_2, NO_x, CO and O_3 measured at Bothasig, City Centre, Goodwood, Khayelitsha and Tableview (24-h averages based on at least 20 1-hour values). PM_{10} concentrations appear to be higher at Khayelitsha compared to the other sites. SO_2 levels seem to be similar at all sites. NO, NO_2 and CO concentrations seem to be much higher at the City Centre site compared to the other sites. O_3 averages appear to be higher at Molteno compared to Goodwood.

3.6.2 Inter-site Correlations

Tables 3 to 5 list the inter-site correlation coefficients for pollutant concentrations between the sites. On average, the most homogeneously distributed pollutant is NO_2, followed by PM_{10} (including Khayelitsha data), PM_{10} (excluding Khayelitsha data), NO, CO, O_3 and finally SO_2 in the Cape Town air shed. A distinction is made between inter-site correlation coefficients for PM_{10} that include and exclude data from Khayelitsha due to poor data collection at Khayelitsha (Section 3.5 and Table 2). Inter-site correlation coefficients for NO_2, NO, SO_2, CO and O_3 vary from 0.456 to 0.832; 0.212 to 0.791; -0.100 to 0.662; 0.302 to 0.676 and 0.123 and 0.557, respectively. PM_{10} measured at Bothasig, City Centre, Goodwood and Tableview presents inter-site correlation coefficients from 0.261 to 0.859 (Table 3)

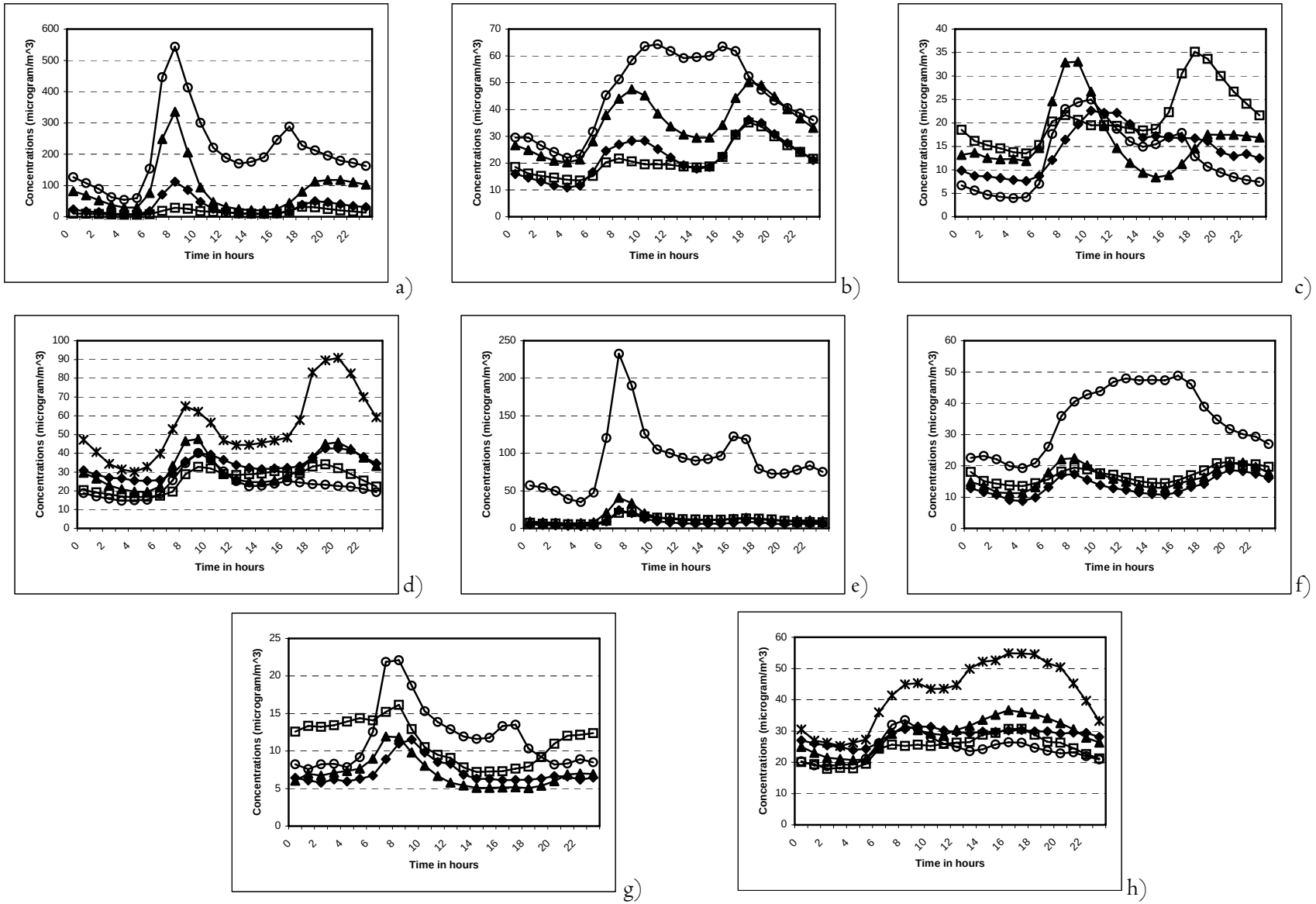


Figure 7 Averaged 24-h a) NO, b) NO_2, c) SO_2 and d) PM_{10} variations at Bothasig (◆), City Centre (○), Goodwood (▲), Khayelitsha (×) and Tableview (□) during winter and during summer e), f), g), h)

Table 2 Descriptive statistics for PM_{10}, SO_2, NO, NO_2, NO_x, CO and O_3 24-h average concentrations (in $\mu g.m^{-3}$) measured at Bothasig, City Centre, Goodwood, Khayelitsha and Tableview during 1 August 1998 – 31 July 2003

Pollutant	Site	N (valid obser-vations)	% valid obser-vations	Min	25th percent-tile	Median	Mean	75th percent-tile	Max	Std Dev
PM_{10}	Bothasig	1157	63.4	2.92	17.38	25.29	28.49	37.63	107.54	14.28
	City Centre	1811	99.2	3.79	15.08	20.92	23.29	28.75	95.58	11.49
	Goodwood	1782	97.6	3.29	19.58	26.69	29.48	35.42	110.42	13.90
	Khayelitsha	965	52.8	4.63	30.92	40.92	46.39	56.75	164.75	23.31
	Tableview	1720	94.2	4.67	16.60	22.35	25.19	30.94	108.13	12.69
SO_2	Bothasig	1559	85.4	0.17	4.79	8.04	10.35	13.00	87.63	8.80
	City Centre	1740	95.3	0.04	5.90	10.31	12.79	17.13	61.50	9.60
	Goodwood	1623	88.9	0.67	5.54	9.08	12.27	15.83	71.92	10.00
	Tableview	1718	94.1	0.46	4.96	8.75	12.47	16.17	94.42	11.34
NO	Bothasig	1415	77.5	1.17	4.88	8.63	17.61	18.13	203.08	24.96
	City Centre	1767	96.8	4.54	80.13	118.38	149.50	179.63	924.17	112.99
	Goodwood	1664	91.1	1.29	10.56	20.75	45.83	52.85	569.42	61.05
	Tableview	1547	84.7	0.38	4.75	8.71	12.79	15.88	125.88	13.10
NO_2	Bothasig	1416	77.5	1.38	10.13	16.27	18.04	23.10	104.96	10.77
	City Centre	1766	96.7	3.38	28.83	38.54	42.12	52.00	132.21	19.26
	Goodwood	1664	91.1	2.83	14.96	22.42	25.96	33.00	130.00	15.38
	Tableview	1539	84.3	0.29	11.04	18.83	20.82	27.71	78.67	12.74
CO	City Centre	1357	74.3	58.54	1528.08	2059.92	2254.33	2755.71	9273.71	1150.17
	Goodwood	1433	78.5	14.04	321.42	563.63	849.49	1080.25	5098.63	765.89
O_3	Goodwood	1613	88.3	0.04	20.00	27.54	28.48	35.71	86.63	11.76
	Molteno	1727	94.6	0.71	28.33	38.13	39.27	49.04	88.29	15.10

The PM_{10} correlation coefficients for the Bothasig, City Centre, Goodwood and Tableview sites with Khayelitsha are presented in Table 4. The inter-site correlation coefficients vary from 0.396 to 0.769. The majority of inter-site correlation coefficients for all the pollutants were positive, which means that if a particular air pollutant's levels are increasing at one site they are also increasing at another site. The inter-site correlation coefficient for SO_2 between Bothasig and Tableview was negative during spring, which means that if SO_2 levels were increasing at Bothasig they were decreasing at Tableview and vice versa. However, all SO_2 inter-site correlation coefficients between Bothasig and Tableview were not significant during all seasons.

The seasonal fluctuations of the inter-site correlation coefficients were investigated using seasons as a proxy for meteorological factors. The results suggest that the inter-site correlation coefficients of NO, CO and SO_2 display seasonal fluctuations: generally strongest in winter/autumn and weakest in summer (Tables 3 and 5). NO_2 appears to be homogenously distributed over all four seasons. This is also the case for the PM_{10} inter-site correlation coefficients between Bothasig-City Centre (BC), City Centre-Goodwood (CG). The inter-site correlation coefficients of PM_{10} between Bothasig-Goodwood (BG) and Bothasig-Tableview (BT) appear to be stronger during autumn and those between City Centre-Tableview (CT) and Goodwood-Tableview (GT) during spring. The inter-site correlation coefficients of PM_{10} between Khayelitsha and Bothasig, City Centre, Goodwood and Tableview seem to be independent of seasonal influences. O_3 presents the strongest inter-site correlation coefficients in summer and weakest during spring.

3.7 Discussion

As the air pollution monitoring sites in Cape Town are located in different types of areas it was anticipated that the inter-site correlation coefficients for the different pollutants would differ. However, it is still necessary to quantify the inter-site correlation coefficients instead of describing the situation qualitatively. This study therefore investigated the seasonal inter-site correlation between 24-h average outdoor PM_{10} mass, NO_2, NO, SO_2, O_3 and CO in Cape Town, South Africa. The aim of this study was not source apportionment, but to consider the suitability of information

Table 3 Sample sizes and p-values for NO_2, NO, SO_2 and PM_{10} inter-site correlation coefficients between Bothasig, City Centre, Goodwood and Tableview

Pollutant	Season	B-C			B-G			B-T			C-G			C-T			G-T		
		Corr	n	p	Corr	n	p	Corr	n	p	Corr	n	p	Corr	n	p	Corr	n	p
NO_2	Winter	0.504	355	<.0001	0.706	352	<.0001	0.786	289	<.0001	0.612	443	<.0001	0.613	388	<.0001	0.553	375	<.0001
	Spring	0.671	373	<.0001	0.733	306	<.0001	0.781	339	<.0001	0.591	357	<.0001	0.617	384	<.0001	0.606	305	<.0001
	Summer	0.456	338	<.0001	0.596	310	<.0001	0.832	281	<.0001	0.490	393	<.0001	0.597	335	<.0001	0.672	317	<.0001
	Autumn	0.607	310	<.0001	0.561	324	<.0001	0.703	312	<.0001	0.572	411	<.0001	0.681	385	<.0001	0.552	396	<.0001
NO	Winter	0.722	354	<.0001	0.791	352	<.0001	0.738	292	<.0001	0.656	442	<.0001	0.626	392	<.0001	0.487	380	<.0001
	Spring	0.613	372	<.0001	0.451	306	<.0001	0.363	339	<.0001	0.462	356	<.0001	0.350	383	<.0001	0.244	305	<.0001
	Summer	0.562	338	<.0001	0.360	310	<.0001	0.464	276	<.0001	0.392	393	<.0001	0.444	337	<.0001	0.212	317	<.0001
	Autumn	0.742	312	<.0001	0.713	323	<.0001	0.702	310	<.0001	0.628	414	<.0001	0.682	390	<.0001	0.532	398	<.0001
SO_2	Winter	0.148	373	0.004	0.395	393	<.0001	**0.048**	390	0.342	0.662	414	<.0001	0.388	411	<.0001	0.330	429	<.0001
	Spring	0.155	374	<0.010	0.206	302	0.000	**-0.100**	384	0.051	0.379	323	<.0001	0.387	434	<.0001	0.249	330	<.0001
	Summer	0.147	365	0.005	0.478	336	<.0001	**0.112**	356	0.034	0.294	385	<.0001	0.485	407	<.0001	0.170	373	0.001
	Autumn	0.360	380	<.0001	0.609	383	<.0001	**0.004**	357	0.938	0.536	424	<.0001	0.374	387	<.0001	0.287	398	<.0001
PM_{10}	Winter	0.564	290	<.0001	0.615	278	<.0001	0.272	277	<.0001	0.714	439	<.0001	0.564	443	<.0001	0.694	428	<.0001
	Spring	0.464	288	<.0001	0.490	272	<.0001	0.261	285	<.0001	0.678	436	<.0001	0.636	429	<.0001	0.750	415	<.0001
	Summer	0.398	281	<.0001	0.717	277	<.0001	0.414	276	<.0001	0.665	440	<.0001	0.609	430	<.0001	0.747	425	<.0001
	Autumn	0.601	290	<.0001	0.859	294	<.0001	0.487	275	<.0001	0.678	452	<.0001	0.544	405	<.0001	0.588	411	<.0001

Bold correlation coefficients are insignificant at 99% level

Table 4 Sample sizes and p-values for PM_{10} inter-site correlation coefficients between Khayalitsha and the other sites

Season	KH-B			KH-C			KH-G			KH-T		
	Corr	n	p	Corr	n	p	Corr	n	p	Corr	n	p
Winter	0.496	138	<.0001	0.529	270	<.0001	0.769	257	<.0001	0.676	267	<.0001
Spring	0.673	89	<.0001	0.396	225	<.0001	0.612	226	<.0001	0.678	205	<.0001
Summer	0.616	97	<.0001	0.406	217	<.0001	0.641	216	<.0001	0.756	206	<.0001
Autumn	0.548	116	<.0001	0.508	248	<.0001	0.655	249	<.0001	0.616	224	<.0001

Table 5 Sample sizes and p-values for O_3 and CO inter-site correlation coefficients between City Centre, Goodwood and Molteno.

Season	C-G (CO)			G-M (O_3)		
	Corr	n	p	Corr	n	p
Winter	0.676	404	<.0001	0.521	415	<.0001
Spring	0.517	297	<.0001	0.123	355	<.0001
Summer	0.302	268	<.0001	0.557	363	<.0001
Autumn	0.546	352	<.0001	0.237	440	<.0001

derived from the air pollution monitoring network in Cape Town in epidemiological studies.

The descriptive statistics are in accordance with the major air pollution sources present in each area. The majority of the inter-site correlation coefficients of all the pollutants under investigation are significantly different from zero at the 99% level, exceptions include those for SO_2 between Bothasig and Tableview. The majority of the coefficients for the pollutants are lower than 0.7. These differences in inter-site correlation coefficients may not only be because the sites are located in different types of areas with different local air pollution sources, but also due to topographical and meteorological factors (temperature, relative humidity, wind speed, wind direction, rainfall). These factors also influence the diurnal variation differences. The influence of meteorological factors on the inter-site correlation coefficients could not be investigated due to the lack of their measurements at these sites.

Seasonal inter-site correlation has exposure assessment implications. If outdoor inter-site correlations are found to be higher in winter it has little use as most people then spend their time indoors, whilst the opposite might be true in summer when correlations are low. The results highlight the importance of properly characterising relations among different outdoor pollution monitoring sites. Studies conducted in locations with strong inter-site correlation coefficients among outdoor pollutant concentrations should not assume that they necessarily persist across seasons.

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) and approximately 5% of the research budget is spent on health-related research, compared with 30% in developed nations.[27] This means that under the current research circumstances, there is still merit in using NO, NO_2, SO_2, O_3 and CO data from one site for another. This approach will make outdoor air pollution exposure assessment easier during hospital time-series studies when relating health outcomes of patients living and moving around in different areas of the city to outdoor air pollution. This is however not the case for SO_2 measured at Bothasig and Tableview. Furthermore, the composition of PM_{10} is anticipated to be heterogeneous at the different sites due to different local air pollution sources. The air quality data from a site should ideally be used in conjunction with knowledge of the local demographical factors when conducting human exposure assessments, as older and sicker people spend more time indoors and/or outdoors in the one area.

Using outdoor air pollution exclusively in epidemiological studies are still prone to information bias as people move around during the day from one area to the next, with a small proportion of their time spent outdoors. Great variability occurs in the different micro-environments, in particular indoors, where pollutants may be far in excess of national guidelines, especially in South Africa and other developing countries where people are still using polluting fuels for household purposes, living in poorly ventilated informal dwellings in close proximity to the pollution sources. The variability is particularly important in townships and informal settlements.

Limitations of this investigation include poor data collection of PM_{10} at Khayelithsa; poor data collection of NO, NO_2, SO_2, O_3 and CO at the other sites during some periods of the investigation; lack of meteorological data collection (temperature, relative humidity, wind speed, wind direction, rainfall) at each monitoring site; lack of emission source data and dispersion modelling.

The vast majority of studies published in reputable scientific journals on inter-site correlations are from developed nations. It is not feasible to compare the results from

this study with those derived in developed nations due to the mix of air pollution sources in South Africa. Pollution sources include traffic, industry and households using polluting fuels for lighting, cooking and heating. The only other study conducted in developing countries was identified as the Central European Study on Air Pollution and Respiratory Health (CESAR).[28] CESAR was conducted in Bulgaria, Czech Republic, Hungary, Poland, Romania and Slovak Republic during November 1995 and October 1996. The study found that the median of the correlation coefficients for PM_{10} between study areas within the same country was 0.76 and that between study areas from different countries was 0.54. The median of the correlation coefficients for PM_{10} for Cape Town is 0.614 (lumping correlation coefficients of all seasons in Tables 3 and 4 together). It thus appears that air pollution is much more heterogeneously distributed on a small scale in Cape Town than between different cities, as was the case for the CESAR study.

Given the lack of resources for outdoor air pollution measurement and the dearth of air pollution epidemiological studies in South Africa, local cities should thus attempt to optimise and update their air quality monitoring networks in such a manner as to serve both compliance monitoring and epidemiological exposure assessment. Inter-site correlation analysis is only one of the evaluation criteria in the design of an air quality monitoring network. The local authorities are encouraged to consult the latest scientific literature with the assistance of research and academic institutions when updating or designing their air quality monitoring networks.[29,30]

Chang and Tseng discussed the optimal evaluation of expansion alternatives for existing air quality monitoring networks in a growing metropolitan region, which poses many uncertainties such as the changing population density and the changing emission sources in the urban environment.[29] They discussed the principles for siting air quality monitoring stations through a multi-objective analysis, which may include the following: monitoring stations should be located in areas of high population density; where pollution concentrations are expected to be highest; where the highest frequency of violation can be detected; where significant growth is expected to occur and near major downwind sources. The optimisation modelling addressed by Chang and Tseng

considered three objectives: the maximisation of protection capability of the highest population density; detection capability of the highest pollution concentrations and detection capability of the highest frequency of violation of health guidelines/standards, along with cost, effectiveness and efficiency factors.[29]

Kanaroglou et al discussed the establishment of an air pollution monitoring network for intra-urban population exposure assessment, which is quite relevant in epidemiological studies.[30] The impetus for their study was to address the limitation of locating monitors in an ad hoc fashion, which favours the placement of monitors in source "hot spots" or in areas deemed subjectively to be of interest. Their study addressed the development of a formal method of optimally locating a dense network of air pollution monitoring stations and the subsequent development of an exposure assessment model based on these monitoring data and related land use, population and biophysical information.

Stemming from the preliminary results of this investigation along with its associated limitations, the following thoughts are suggested to the City of Cape Town:

(1) not to reduce the number of sites in the city;
(2) add NO_x, SO_2, CO and O_3 monitors to the Khayelitsha site due to the proximity to a busy highway, the dense population of the area and relatively high use of polluting fuels for cooking and space heating;
(3) increase data response at the Khayelitsha site;
(4) collect meteorological data (temperature, relative humidity, wind speed, wind direction, rainfall) at each monitoring site;
(5) increase the number of O_3 monitors in background areas and
(6) place O_3 monitors downwind from precursor sources.

Although O_3 is not a primary pollutant, it is at the moment unclear whether it is generated in the Khayelitsha area. Furthermore, it is suggested not to reduce the number of measurements per day as hourly measurements are needed to indicate peaks and for acute health effects assessment. These suggested recommendations aim at increasing the accuracy and reliability of using the air quality network data in epidemiological studies. Future studies should investigate the current air pollution trends against health

guidelines and establish the surrogate or confounder relationship between air pollutants in a time-series analysis (that is inter-pollutant correlations at each monitoring site).

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Chapter 4

POTENTIAL HEALTH IMPLICATIONS OF OUTDOOR PM_{10} MASS EXPOSURE IN THE KHAYELITSHA SUB-DISTRICT, CAPE TOWN, SOUTH AFRICA

Objectives: This study investigated the current and future potential health implications due to outdoor PM_{10} mass exposure in the Khayelitsha sub-district in the City of Cape Town in a preliminary analysis.

Methods: The PM_{10} mass data comprised of concentrations averaged at hourly and daily intervals from 1 March 1999 – 31 July 2003. Daily PM_{10} mass concentrations were compared to the United Kingdom (UK) Daily Limit value (DLV) (50 $\mu g.m^{-3}$) to assess current potential health implications. Preliminary trend analysis was conducted to investigate future potential health implications.

Results: The results indicate that elevated daily PM_{10} mass concentrations are frequently present. During autumn 1999 and winter 2003 the UK Daily Limit value (50 $\mu g.m^{-3}$) was exceeded 44 times. The number of times the UK DLV may be exceeded should not be more than 35 during a year. Although this requirement was only surpassed twice, the preliminary trend analysis indicates an increase in monthly average and maximum daily average PM_{10} concentrations.

Conclusions: Currently PM_{10} mass concentrations frequently exceed the UK DLV. Health of sensitive sub-populations might therefore be more at risk. Within a few years the UK Annual Limit value (40 $\mu g.m^{-3}$) might be exceeded based on the preliminary trend analysis. Consequently a higher proportion of the community might be at risk from suffering various detrimental health effects in the future.

4.1 Introduction

Airborne particulate matter represents a complex mixture of organic and inorganic substances. Mass and composition tend to divide into two principal groups: coarse particles (between 2.5 and 10 μm in aerodynamic diameter)(PM_{10}) and fine particles mostly smaller than 2.5 μm in aerodynamic diameter ($PM_{2.5}$). The smaller particles contain the secondarily formed aerosols (gas-to-particle conversion), combustion particles and recondensed organic and metal vapours. The larger particles usually contain earth crust materials and fugitive dust from roads and industries. Particles less than 10 μm in diameter are considered respirable and capable of gaining access to the lower respiratory tract.

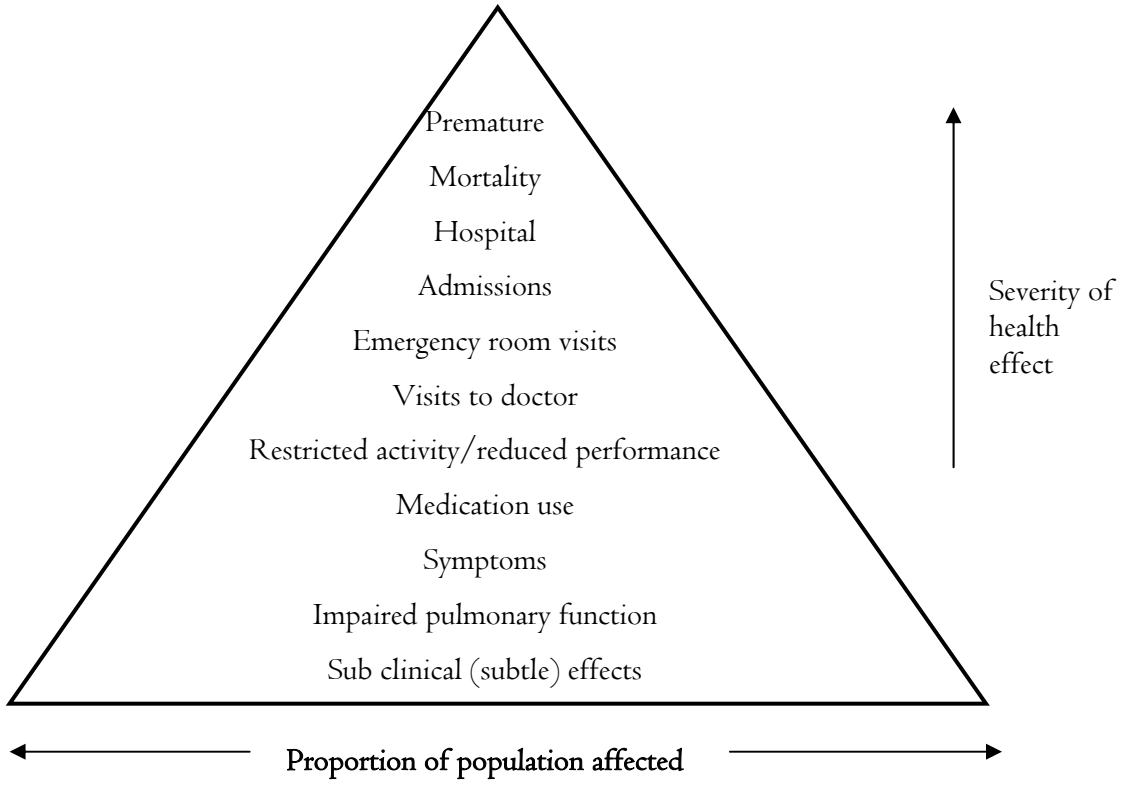


Figure 1 Air pollution health effects pyramid

Numerous epidemiological and toxicological studies have related PM exposure to a range of adverse health outcomes, ranging from mortality to subclinical respiratory symptoms (Figure 1).[1-4] Epidemiological studies also indicate associations between the size of inhaled particles and the development of pulmonary diseases.[5,6]

The majority of the air pollution epidemiological and exposure assessment studies are carried out in developed countries. It is important to conduct more air pollution

epidemiological and exposure assessment studies in South Africa as it is faced by pollution caused by industry (First World situation) and by domestic burning of coal and dirty fuels (Third World situation). Study results (such as exposure-response curves) acquired in developed countries cannot be extrapolated with total conviction to developing countries.[7] The three global factors that directly or indirectly impact on health - the community and social environment, the physical environment and the family and individual environment – are different for developed and developing countries.

Very few quantitative exposure assessment or analytical epidemiological studies have been conducted in South Africa. Exposure is usually based on a proxy measure, such as smoking status[8,9] or use of dirty fuels (such as wood, coal, paraffin, animal dung, crops) for space heating.[10,11] In a review by Wichmann and Voyi four studies were identified that measured outdoor air pollution directly.[12-19] Wichmann and Voyi concluded that the local studies do provide some evidence of associations with a range of serious and common health problems.[12] Three of the studies established a strong significant relationship with air pollution indicators and deteriorated human health.[16-21] Seven studies established a significant link with air pollution indicators and disease.[8-10,22-24] Four studies did not report any significant link between air pollution indicators and disease.[13,15,25,26] Two studies mention a correlation between air pollution indicators and disease, but did not quote the p-value or the confidence interval of the association measure.[14,27] The lower limit of many of the association measures of some of the reviewed studies is more or less one.[22-24] Most of the studies to date have had an observational rather than analytical or intervention design. The latter two study designs result in more robust evidence on the nature of the relationship between air pollution and health. None of the studies established exposure-response curves for any of the criteria air pollutants (lead, PM_{10}, SO_2, NO_2, O_3, CO) due to the lack of quantitative air pollution measurements. The repercussion of these limitations is that, although results are fairly confident that air pollution indicators do boost the risk of adverse health outcomes, it is not yet clear by how many people. It is therefore problematical to use the results of the studies in risk assessment studies.

For the purpose of this paper, Khayelitsha sub-district in Cape Town comprises of electoral wards 88 to 99.[28] The electoral wards do not always cover the same area as

the sub-districts. The nearly 330 000 residents of the Khayelitsha sub-district are particularly vulnerable to the health implications of exposure to air pollution. The severity of health outcomes to air pollution increases with vulnerability. The predominant age group in the Khayelitsha sub-district is 15-34 years old (46%), followed by the 35-64 year olds (24%), 5-14 year olds (19%), 0-4 year olds (10%) and 65+ year olds (1%).[28] Seventy percent of entire population does not have any individual monthly income, whilst 25% earn less than ZAR1600 per month. The remainder earn between ZAR1600-ZAR6400 per month. In terms of the internationally comparable poverty line of $1 per capita per day, the level of poverty in Khayelitsha is about 75% ($1 = ZAR8). The unemployment rate is currently 50%. The majority of the homes fall within the informal category (65%), with the remainder under the formal type. Seventy percent of the households have 1-4 members. Most of the homes have 1-4 rooms (86%). HIV prevalence increased from 22.0% in 2000 to 24.9% in 2002.[29] The rate of tuberculosis in Khayelitsha is the second highest (977 per 1000 in 2002)(after the Nyanga sub-district) in the City of Cape Town (638 per 1000). It is well known that permanent lung damage is a side-effect of TB infection, even for person who successfully complete treatments.[30,31] The infant mortality rate during 2000 was 47 per 1000 compared the City of Cape Town (26 per 1000) and the national level (45 per 1000). Main causes of infant mortality are HIV/AIDS, diarrhoeal disease, short gestation/low birth weight and pneumonia. IMF decreased from 63 per 1000 in 1999 to the current rate. Under five mortality and low birth weight are the highest compared to the other sub-districts in the City of Cape Town.

This study investigated the current and future potential health implications due to outdoor PM_{10} mass exposure in the Khayelitsha sub-district in the City of Cape Town in a preliminary analysis.

It is important to point out that this study was done independently from the City of Cape Town. The City of Cape Town is responsible for developing an air quality management plan which should be incorporated into its integrated development plan according to the National Environment Management: Air Quality Act (Act 39 of 2004).[32] However, human resources capacity to investigate fundamental issues pertaining to air pollution exposure assessment in detail are *currently* not available at local government level.

4.2 Meteorology and topography

The City of Cape Town was established in December 2000 by the merging of the previous Cape Metropolitan Council and six Metropolitan Local Councils: Tygerberg, Oostenberg, Blaauwberg, South Peninsula, Helderberg and Cape Town. It is the southern most metropolitan area on the African continent and covers an area of 2 487 km^2. The Khayelitsha sub-district is a large, predominantly informal settlement located about 30km from the centre of Cape Town. Khayelitsha was established in 1983 and grew rapidly.

The City of Cape Town has a Mediterranean climate. Summer is from December to February with temperatures averaging at around 28°C. The prevailing wind during October to March (spring to autumn) is from SSE to SSW. It brings very little rain with it. Occasionally during summer the area will experience northerly "berg winds" with associated increase in temperatures. The Southeaster subsides slightly during February and March. The prevailing wind between May and August (autumn and winter) blows from N to NW. This wind is not as strong as the South Easter and occurs less frequently. It precedes a cold front and is therefore followed by much needed rain. The rainy season peaks during June and July. During March to August the area have calm atmospheric conditions and low level inversions.

4.3 Air pollution monitoring network

Figure 2 indicates the air quality monitoring sites in the City of Cape Town. PM_{10} mass monitoring commenced during March 1999 in the Khayelitsha sub-district using a TEOM 1400A analyser (Figures 3).[33] The analyser continuously assess real-time concentrations of PM_{10} mass using US-EPA equivalent methods in accordance with ISO 17025 guidelines. These instruments measure the concentration of ambient air pollutants in 20 second scans and values can be expressed in short term (10 min), one hour, twenty four hour, monthly or annual averages.

Minimum data completeness requirements for the calculation of period averages issued by the US-EPA in accordance with ISO 17025 guidelines for continuous monitoring are stipulated as follows:

- 24-hour average - requires at least 20 one-hour samples;
- Monthly average requires at least 80% of data days of >20 hours or more;

- Quarterly average requires 80% data capture and no more than 10 consecutive incomplete days;
- Annual average - requires 4 complete quarters.

The PM_{10} mass data are posted on the City's web site as soon as the data has been analysed statistically. The site can be visited at the City's Air Quality Department website.[34]

Figure 2 Air quality monitoring sites in the City of Cape Town[33]

4.4. Air pollution sources

Various environmental challenges confront the Khayelitsha sub-district. These are primarily the consequence of the growing population and their concurrent need for infrastructure, housing, employment and education.

Rapid urbanisation and urban growth have resulted in a larger population of the City of Cape Town and this in turn leads to a larger number of people making use of public and especially private transport to commute to work. During peak periods the capacity of some road networks is exceeded and other road networks are

reaching their capacity. A large proportion of the residents of Khayelithsa live close to a busy highway (N2) and other busy roads (such as the R310) (Figure 3).

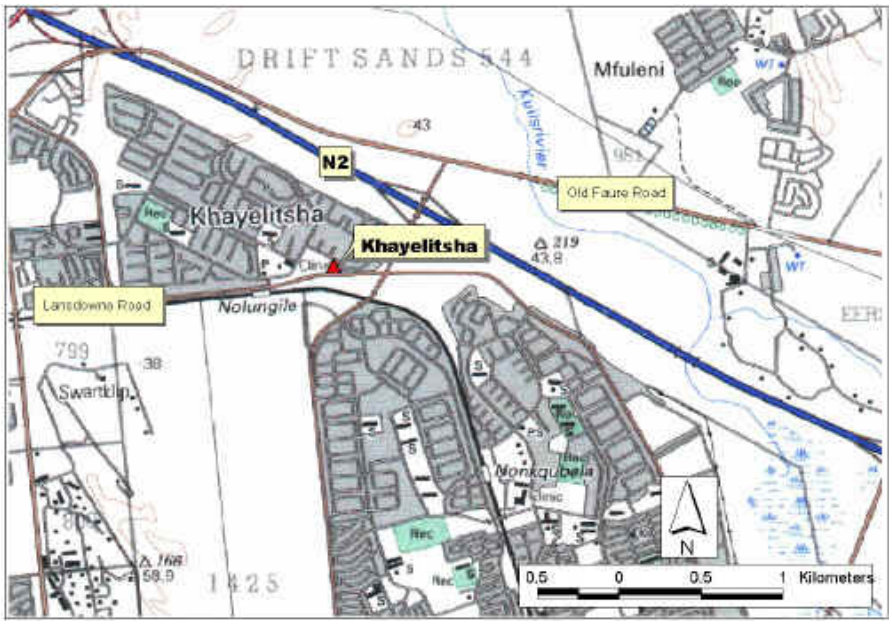


Figure 3 Location of air quality monitoring site in the Khayelitsha sub-district, City of Cape Town[33]

Paraffin, electricity and candles are used for lighting by 22%, 76% and 2% of the nearly 86 000 households in Khayelithsa, respectively.[28] Statistics on the energy use for cooking and heating are not available on a sub-district level. As nearly 100% of people living in Khayelithsa are Black, the energy use profiles for cooking and heating in Black households in the entire City of Cape Town will be quoted here.[28] 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, socio-economic status and educational status. The energy use profiles for cooking in Black households in the entire City of Cape Town are as follows: 45%, 4%, 49%, 1% and 1% for electricity, gas, paraffin, coal and animal dung, respectively.[35] The figures for heating are 33%, 1%, 57%, 4% and 4% respectively for electricity, gas, paraffin, wood and other unspecified fuels.[35] The Khayelitsha sub-district is also located close to an industrial area.

Outdoor air quality remains a key issue in the City of Cape Town as a whole, largely because of the visible air pollution, particularly during March to August - known as the 'brown haze'.[33] The brown haze is associated with calm atmospheric conditions and low level inversions. It occurs over most of the City and is typically most severe in the morning. Air pollutants (such as SO_2, NO_2, particulate matter and heavy metals) result from combustion processes in industry, services, agriculture, transport and homes.

4.5 Methods

SAS version 8 was used in the statistical analyses for data collected from 1 March 1999 – 31 July 2003. MS Excel was applied in the preliminary trend analysis. The data comprised of concentrations averaged at hourly and daily intervals. During the statistical analysis the ISO 17025 guidelines were adhered to, namely all 24-h averages and monthly averages are based on at least 20 1-h and at least 24 24-h averages, respectively. If this requirement was not met, a 24-h average or monthly average was set as a missing value. Percentages of hourly data available measured at Khayalithsa are: 26.4% (1 March 1999-29 February 2000), 73.4% (1 March 2000-28 February 2001), 56.7% (1 March 2001-28 February 2002), 16.6% (1 March 2002-28 February 2003) and 97.1% (1 March 2003-31 July 2003).

An exploratory data analysis was conducted in which the untransformed and log transformed data were tested for normality using the Kolmogorov-Smirnov test at the 95% confidence level. It was concluded that log transformations were ineffective in normalising the data. An alternative for normalising the data was attempted by the culling of extreme values above 61 $\mu g.m^{-3}$ (hereafter refer to as high values). This is practical as the data remain in their original units. This procedure was ineffective in normalising the data.

On average 92% of the hourly values of all the pollutants measured over the five year period were less than the threshold value of 61 $\mu g.m^{-3}$. As no hourly South African and UK limit values exist, no consideration was given to setting a threshold value based on these values. The daily and annual South African guideline values are 180 $\mu g.m^{-3}$ and 60 $\mu g.m^{-3}$, respectively. Those of the UK are 50 $\mu g.m^{-3}$ and 40 $\mu g.m^{-3}$, respectively. In its State of the Environment report of 1998, the City of

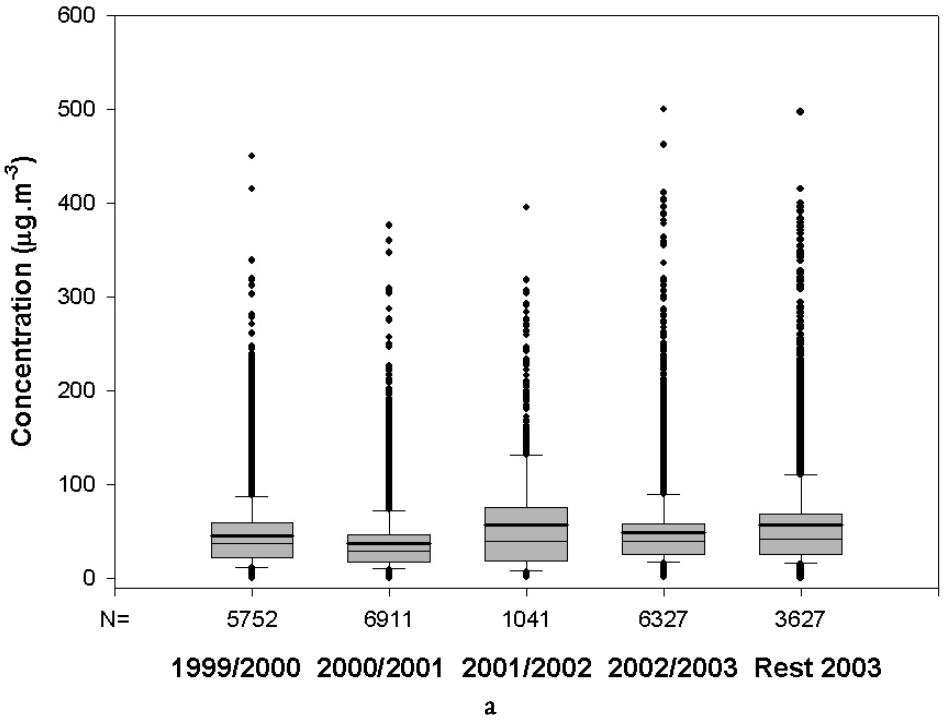
Cape Town implemented the guidelines and air quality banding system as adopted in the UK.[36] This was as the result of extensive research by the UK's Department of the Environment's Expert Panel on Air Quality Standards (EPAQS), which is linked with health advice from the UK's department of Health's Committee on the Medical Effects of Air Pollutants (COMEAP). The South African guideline system has been under review when writing this article.

4.6 Results

Figure 4 presents the descriptive statistics of the hourly and daily PM_{10} concentrations. The annual PM_{10} means cannot be computed as data are not composed of 4 complete quarters according to the ISO 17025 quality assurance guidelines. The hourly and daily PM_{10} concentrations both have a positively skewed distribution (Figure 4).

Figure 5 indicates the typical hourly variations of PM_{10} during the four seasons when all the hourly pollutant concentrations were averaged over the monitoring period. In general the concentrations were highest in winter, decreasing in spring and summer and increase again in autumn. The profiles shown in the figure show that the concentrations of PM_{10} closely follow the diurnal pattern of traffic with peak hour and off-peak hour differences as well as lighting, cooking and space heating times, i.e. the higher levels are usually observed in the morning between 8:00 and 9:00 and the evening rush hours 16:00 to 19:00. The low values (i.e. concentrations $\leq$ 61 $\mu g.m^{-3}$) are similar during all seasons (Figure 5b). The high values (i.e. concentrations >61 $\mu g.m^{-3}$) indicate a peak of 160 $\mu g.m^{-3}$ between 20:00 and 22:00 during winter (Figure 5c).

Figure 6 illustrates the maximum and minimum daily concentrations for the period under investigation. All the maximum and minimum daily concentrations are larger and smaller than the UK Daily Limit value (DLV) of 50 $\mu g.m^{-3}$, respectively. In general the minima concentrations appear to vary less over time compared to the maxima. The maxima initially indicate a declining trend followed by a sudden increase.



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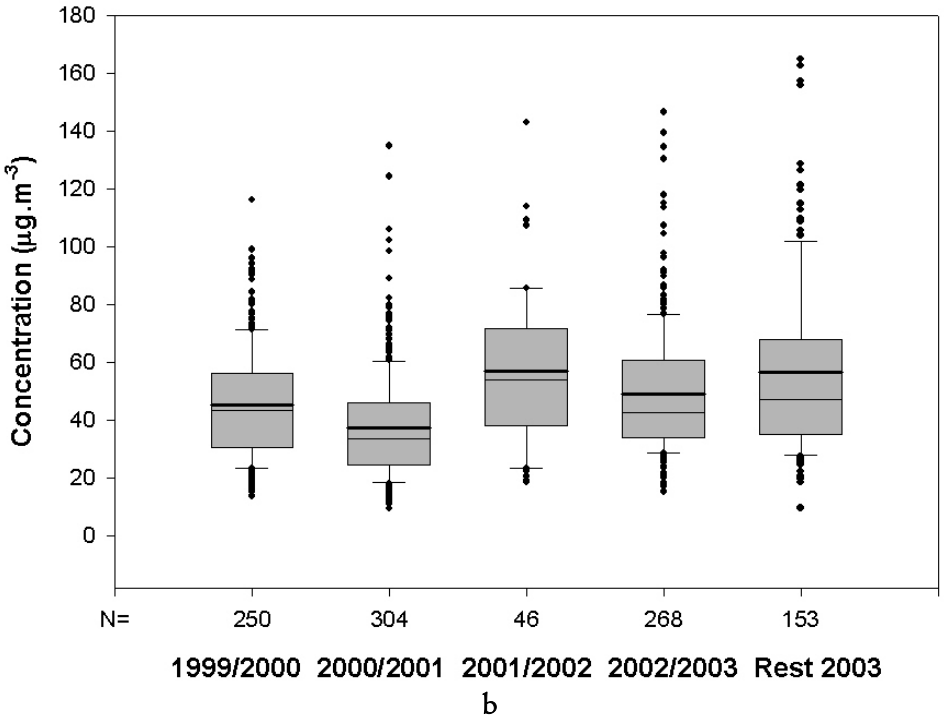

b

Figure 4 Descriptive statistics of a) hourly and b) daily PM_{10} concentrations ($\mu g.m^{-3}$)

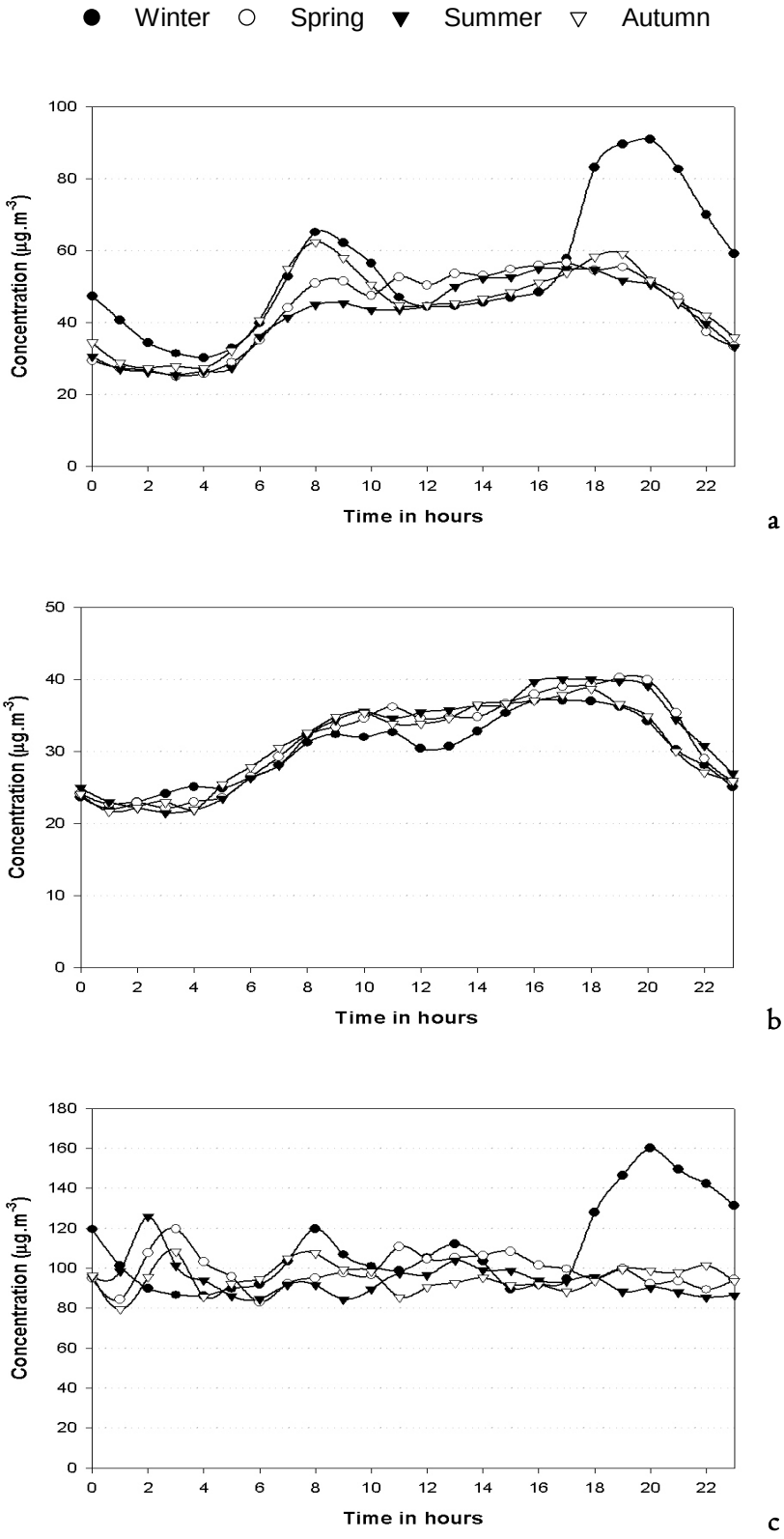

Figure 5 Seasonal diurnal trends of PM_{10} for a) all, b) low and c) high concentrations ($\mu g.m^{-3}$)

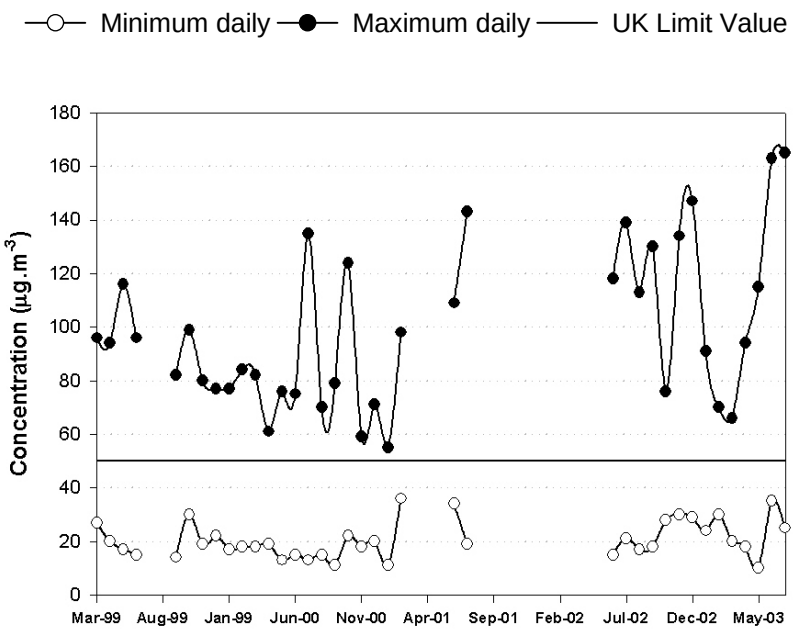

Figure 6 Daily minimum and maximum values (µg.m^{-3}) for PM_{10} during 1 March 1999 – 31 July 2003

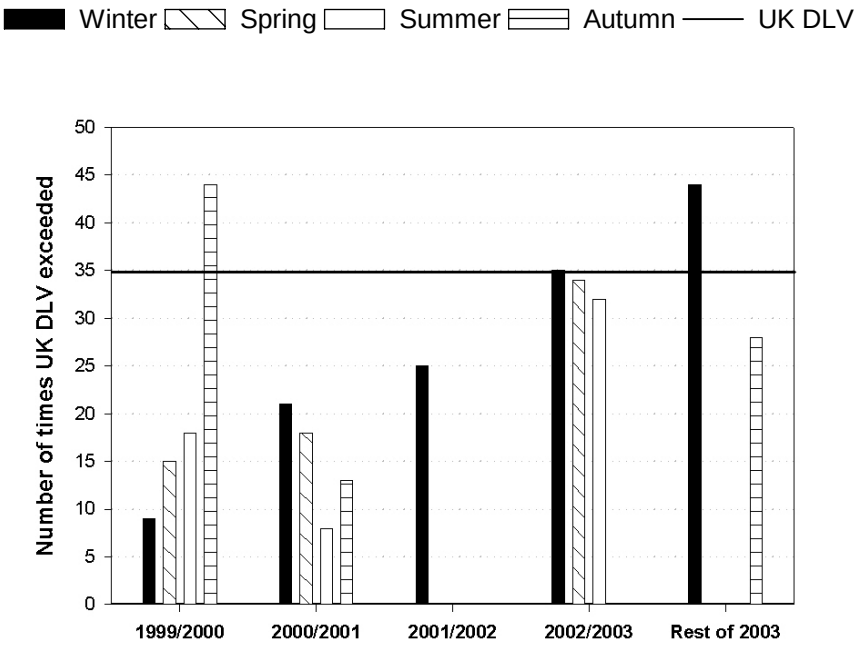

Figure 7 Number of times the UK Daily Limit value are exceeded for PM_{10} during 1 March 1999 – 31 July 2003

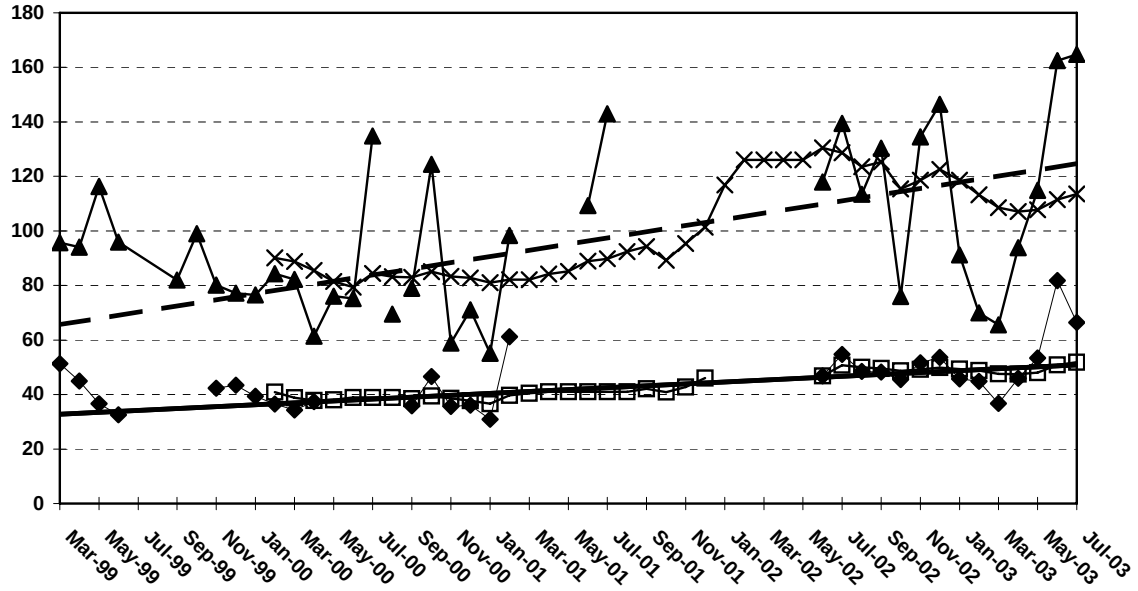

Figure 8 PM_{10} concentrations ($\mu g.m^{-3}$) during 1 March 1999-31 July 2003 for monthly (solid trend line) mean and maximum daily per month (broken trend line)
12 MMA 12 month moving average, ◆ monthly average, □ 12 MMA, ▲ maximum daily, × 12 MMA of max daily

Figure 7 presents the number of times PM_{10} concentrations exceeded the UK DLV. The number of times the DLV are exceeded increases for winter during the period of examination. This trend is not clear for other seasons due to the lack of data. The UK DLV may be exceeded 35 times during a year. During autumn 1999 and winter 2003 alone the UK DLV was exceeded 44 times on each occasion. Winter 2002 and Spring 2002 were close to exceeding the UK DLV more than 35 times.

A moving average provides trend information that a simple average of all historical data would mask. The trend can be used to forecast PM_{10} concentrations. From March 1999 to July 2003 the monthly and maximum daily PM_{10} concentrations increased from 33 $\mu g.m^{-3}$ to 49 $\mu g.m^{-3}$ and from 65 $\mu g.m^{-3}$ to 125 $\mu g.m^{-3}$, respectively according to the preliminary trend lines of the relevant 12 month moving averages (Figure 8). This translates into a rate of 1.22 $\mu g.m^{-3}$/month and 0.33 $\mu g.m^{-3}$/month, respectively. The slope of the trend line of the 12 month moving averages based on the maximum daily PM_{10} concentrations is steeper than the one based on the monthly PM_{10} concentrations.

4.7 Discussion

This study investigated the current and future potential health implications of exposure to outdoor PM_{10} mass in the Khayelitsha sub-district in the City of Cape Town.

During autumn 1999 and winter 2003 the UK DLV was exceeded 44 times (Figure 7). The number of times the UK DLV may be exceeded should not be more than 35 during a year. Although this requirement was only surpassed twice, the preliminary trend analysis indicates an increase in monthly average and maximum daily average PM_{10} concentrations (Figure 8). The maxima initially indicate a declining trend followed by a sudden increase – maxima during July 2001, December 2002 and July 2003. Possible reasons for the increased maxima trend are not clear as the maxima occur both in winter and summer. Figure 8 suggests that within a few years the UK Annual Limit value of 40 $\mu g.m^{-3}$ will be exceeded. The trend line of the 12 MMA based - on the maximum daily PM_{10} concentrations - has a steeper slope than the one based on the monthly PM_{10} concentrations. As there is a UK DLV the increase in the former trend line is important. This indicates that the UK DLV will be exceeded more in the future.

The preliminary results indicate that a higher proportion of the community is at risk from suffering reduced lung function, aggravation and developing of respiratory diseases and symptoms (e.g. asthma, emphysema, acute lower and upper respiratory tract infections, chronic bronchitis, chronic obstructive pulmonary disease, lung cancer) and cardiovascular diseases (Figure 1). Health effects from inhaled particles are influenced by the depth of penetration of the particles into the respiratory system, the amount of particles deposited in the respiratory system, and by the biological reaction to the deposited particles. The risks of adverse health effects are greater when particles enter the tracheobronchial and alveolar portions of the respiratory system. Small particles can penetrate into these deeper regions of the respiratory system. Mouth breathing becomes more prevalent during upper respiratory tract infections (e.g. blocked nose). This results in the functional loss of nasal filtering of PM with greater deposition of particulates and irritants to the lower respiratory tract .[37]

However, it is given that the trend lines might be misrepresentative of the true trend due to the lack of PM_{10} and meteorological data (temperature, relative humidity, rainfall, wind speed, wind direction). Nevertheless, this in turn points towards the requirement to improve data collection at Khayelitsha. Although no guideline or limit value exists for hourly PM_{10}, it is still important to take notice of the extent of exposure to maximum hourly PM_{10} concentrations (Figure 4a). The maximum hourly PM_{10} concentrations ranged between 376-500 $\mu g.m^{-3}$.

Figure 5 suggests traffic and dirty fuels as pressures to the state of the atmospheric environment in Khayelitsha. Consequently the City of Cape Town will need to address these two pressures in order to reduce PM_{10} emissions. It is acknowledged that meteorological conditions such as low inversions may also result in higher observed PM_{10} concentrations.

4.8 Conclusions and recommendations

This study investigated the current and future potential health implications due to outdoor PM_{10} mass exposure in the Khayelitsha sub-district in the City of Cape Town in a preliminary analysis.

Elevated PM_{10} mass concentrations are frequently experienced in Khayelitsha. The temporal variations in PM_{10} mass have repercussions for air monitoring strategies and epidemiological studies concerned with the relationship of exposure to PM_{10} mass and the health impact on populations. Micro variations in exposure need to be considered when assessing exposure to PM_{10} mass in epidemiological studies and improved awareness raised to collect accurate measures of environmental exposure in the light of considerable variations which may occur temporally.

In the South African context economical, social and cultural factors may render the population more vulnerable to increased exposure to PM_{10} mass, due to factors such as poor hygiene, overcrowding, dusty environments, poor nutrition, open dwellings, outdoor lifestyles and continued use of dirty fuels for lighting, heating and cooking. Recommendations are to increase data collection and extend monitoring to NO_x, SO_2, CO, O_3 and $PM_{2.5}$ in Khayelitsha. Future studies need to scrutinise these preliminary trend results in more robust autoregressive integrated moving average (ARIMA) analyses once data collection has been improved and a larger database

established at the Khayelitsha sub-district site. ARIMA analysis removes any last traces of autocorrelation between the 24-hour average data that can lead to errors, which ordinary trend analysis (as applied in this investigation) is not capable of. These robust ARIMA analyses should ideally also include meteorological parameters. Future source apportionment studies may also clarify the contribution of traffic and dirty fuel use to PM_{10} levels. Epidemiological studies in South Africa need to link improved air pollution data to health effects. Health promotion interventions to reduce air pollution exposure can then be developed from these results.

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Chapter 5

POTENTIAL RISK FACTORS OF RESPIRATORY DISEASES AND SYMPTOMS AMONGST ADULTS IN SOUTH AFRICA

Objective: To determine the prevalence and potential risk factors (occupational and environmental, socio-demographic, BMI, TB) of various respiratory symptoms and diseases in a representative adult population (15 years and older) of South Africa.

Methods: During 1998, a probabilistic national survey was performed in 12 763 households. 13 826 individuals from 6 457 households were interviewed for the health survey. Univariate and multivariate logistic regression analyses were used to generate crude and adjusted odds ratios (OR) and 95% confidence intervals (CI) in order to assess the influence of possible risk factors on respiratory diseases and symptoms.

Results: The survey also revealed relatively low crude prevalence rates for doctor diagnosed asthma (3.7%), chronic bronchitis (2.4%), doctor diagnosed emphysema/bronchitis (4.2%) and TB (2.7%), but higher respiratory symptoms: wheeze and shortness of breath (11.1%), cough with phlegm (6.8%); nocturnal coughing (13.1%), nocturnal wheezing/tight chest (10.8%). Nearly 1.7% of the respondents reported using asthma medication, whilst 0.5% were using TB medication. In general most of the potential risk factors were significantly related to the respiratory diseases and symptoms in the unadjusted models. The multivariate logistic regression analyses suggested that the prevalence of respiratory symptoms and diseases could be diminished in South Africa by health promotion predictors (increasing connection to electricity, having a medical aid and improved education). This preliminary analysis suggests that the following potential risk factors should be lessened in order to have a beneficial influence on the prevalence rates of respiratory symptoms and diseases: households going hungry, years smoked, households with smokers, exposure at work to fumes, smoke, dust or strong smells and period worked in such a job as well as BMI increase for the underweight and decrease of the obese. Other potential risk factors included age and race.

Conclusions: Although there is potential for residual confounding despite adjustment in this preliminary analysis, the documented international evidence on most of the potential risk factors suggests that these associations may be real. It is trusted that more detailed South African analytical intervention studies will

scrutinise these results in order to develop integrated intervention programmes to improve adult respiratory health in the country.

5.1 Introduction

The ultimate endeavour of epidemiology is to identify modifiable risk factors of disease occurrence and progression and to contribute in testing the efficacy and effectiveness of interventions on these risk factors including the health services.

Chronic diseases were the main cause of death amongst South Africans in 2000.[1] Much of what must be done to prevent respiratory symptoms and diseases lies outside of the sphere of health care. Therefore interventions should be targeted at risk factors, rather than only providing medical treatment for those already affected.

However, without a clear understanding of the complex interaction between the personal, educational, political, social, economic, cultural, occupational and environmental risk factors, the prevention of chronic diseases at a population level will be hampered. South Africa, a middle income country, is faced by health risk factors from a First World situation (e.g. industry, traffic, aging population) along those from a Third World situation (e.g. domestic burning of coal/biomass fuels, poor sanitation, overcrowding). Thus intervention strategies deduced from studies conducted in developed countries are not merely applicable in this country.

Globally the prevalence of chronic obstructive pulmonary disease (COPD)(such as chronic bronchitis and emphysema) has not been studied to the same extent as asthma. Although there are a number of studies of chronic bronchitis in selected populations in middle- and low income countries, the overall burden and risk factors of COPD in these countries are not well documented.[2-6]

Most environmental epidemiological studies in South Africa focused on children health.[7-18] The 1998 South African Demographic and Health Survey (SADHS) is the first national health survey conducted across the entire country.[19] Data from this survey provided the opportunity to examine the prevalence and potential risk factors of various respiratory symptoms and diseases in a representative national population (both adults and children) rather than a selected high risk population, as has been the case in most previous studies in developed countries.

It is trusted that the results of this preliminary analysis will draw attention to the socio-demographic, environmental and occupational risk factors and lead to debate on potential integrated intervention programs.

5.2 Methods

5.2.1 Survey method

The 1998 SADHS had a cross-sectional design and was a national household survey of the population living in private households in the country. Detailed information on the survey design is outlined elsewhere.[19] The sampling frame for the SADHS was the list of approximately 86 000 enumeration areas (EAs) created by Central Statistics (now Statistics South Africa, SSA) for the Census conducted in October 1996. The EAs, ranged from about 100 to 250 households and were stratified by 9 provinces, urban and non-urban residence and by EA type. The number of households in the EA served as a measure of size of the EA.

The first stage (proportional stratified sampling) of the two-stage sampling led to a total of 972 EAs being selected for the SADHS (690 in urban areas and 282 in non-urban areas). The second stage involved a systematic random sample of 10 and 20 houses in selected urban and rural EAs, respectively. Oversampling was conducted in some areas to enable inference to be made about differences across provinces and race – and in the Eastern Cape province, across health districts.

In addition to the main survey of households an adult health questionnaire was administered individually to a sample of adults aged 15 and over in half of the households selected for the main survey. The SADHS questionnaires were translated into 9 of the 11 official languages of South Africa and checked by backtranslation (Refer to Appendices 1 and 2).[19] The questionnaires were pretested in November/December 1996 as part of a pilot study.

The household questionnaire characterised all household members, including their age, sex, race and education, household characteristics such as fuels use for cooking and heating. The adult health questionnaire elicited information about medical history, symptoms of disease, utilisation of health services, occupational history and smoking habits of the respondents. The questionnaire was accompanied by measurements of height and weight.

Interviewers were trained over several weeks. Interviews were conducted after working hours. Interviewers were constructed to return twice if a suitable respondent was not found at home. Fieldwork commenced late January 1998 and was completed in September 1998. The response rate at the household level was 97% of 12 860 households in 966 EAs. Of the 6 457 households selected for the adult survey, 95.3% were completed. At the individual level, 92.6% of eligible adults were included in the survey, although not all of them had all the measurements taken. The overall response rate for the adult survey was 89.7%. It was substantially lower in Gauteng (67.5%) where a large proportion of adults were not at home (13%). The response rate was higher in the non-urban than urban area.

Ethical approval was granted by the Ethics Committee of the South African Medical Research Council. Informed consent was obtained from each respondent.

5.2.2 Variable definitions

Chronic phlegm was defined as usual cough with phlegm every day for at least 3 months a year for at least 2 successive years. Participants were considered having asthma, emphysema/bronchitis and TB if they answered affirmatively the questions, "Has a doctor or nurse or staff member at a clinic or at a hospital told you that you had or have any of the following conditions: asthma or emphysema/bronchitis or TB". The four respiratory symptoms were prompted by the following questions: "During the last year have you had wheezing or tightness of your chest? If "yes" were you also short of breath?; Is your sleep ever interrupted by you coughing?; Is your sleep ever interrupted by wheezing or a tight chest?; Do you usually cough?; When you cough, do you usually bring up phlegm from your chest?" [19]

Socio-demographic variables included residence in urban/rural area, more than two persons per room, household going hungry, covered by medical aid/medical benefit scheme, payment of medication, age distribution (categorised in quartiles) and ethnic identity (African/Black, Coloured, White, Asian/Indian). 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, socio-economic status and educational status. Educational status was classified as less or equal to primary school, secondary and tertiary education.

Environmental exposure variables included home connection to electricity, type of cooking and heating fuels used (classified as electricity only, electricity and other fuels - such as gas, paraffin, coal, wood and animal dung) and other fuels only - and living in household with smokers.

Occupational exposure variables included, having a job with smokers, ever worked in a job where regularly exposed to smoke, dust, fumes or strong smells and period worked in a job where regularly exposed to smoke, dust, fumes or strong smells (categorised in quartiles).

Variables related to active smoking included ever smoked tobacco, used snuff or chewed tobacco, ever smoked at least 100 cigarettes (5 packets) in lifetime, years smoked on a daily basis (categorised in quartiles) and frequency smoking.

BMI was included in the analysis because of a renewed interest in the association with various respiratory conditions.[20,21] Weight and height were used to calculate the BMI (the weight in kilograms divided by the square of the height in meters, $kg.m^{-2}$) which was used as a measure of adiposity. Categories of BMI were created (<22, 22–24.9, 25–27.4, 27.5–29.9, ≥ 30 $kg.m^{-2}$). The decision to select 22–24.9 $kg.m^{-2}$ as the reference category is based on a large prospective study where the lowest rates of death from all causes were found at a BMI between 22-23.4 $kg.m^{-2}$ in women and between 23.5-24.9 $kg.m^{-2}$ in men.[22] The cut-off points as proposed by the World Health Organisation were used where a BMI of 25–29.9 $kg.m^{-2}$ is termed overweight or pre-obese and a BMI of 30 $kg.m^{-2}$ or higher is considered obese.[23]

5.2.3 Data analysis

All subsequent statistical analyses of results were done using SAS version 8. The 1998 SADHS report pointed out that the potential risk factors might be correlated with each other.[19] Independence among the potential risk factors was investigated with a χ^2 analysis. It was observed that most of the potential risk factors were significantly correlated at the 95% confidence level, although very poorly with correlations coefficients varying from 0.01 to 0.40. Table I lists the variables found

to be significantly related ($p \leq 0.001$) with Phi coefficients larger than 0.5. Consequently conventional logistic regression analysis was conducted, instead of a conditional analysis.

Table 1 Correlations among potential risk factors with Phi coefficients > 0.50 (averaged over strata)

	Connected to electricity	Residence location in urban area	Covered by medical aid/medical benefit scheme	Payment of medicine	Age distribution in years	Educational status	Years smoked on a daily basis
Fuel type used for cooking and heating	✓	✓					
Ethnic identity			✓	✓		✓	
Covered by medical aid/medical benefit scheme				✓			
Age distribution in years				✓			✓
Period worked in job exposed to smoke, dust, fumes or strong smells					✓		✓
Educational status			✓	✓			

The survey population reflected the ethnic make-up of the South African population: Africans (77.2%), Whites (10.5%), Coloureds (8.8%) and Indians (2.5%).[24] Thus no weighting was conducted during the analysis. Simple descriptive statistics were used to examine the potential risk factors and in calculating the prevalence of respiratory symptoms and conditions. The crude odds ratio (OR) and 95% confidence intervals (CI) were derived from conventional univariate logistic regressions performed for binary (coded as 1 for an affirmative response and 0 for a negative response) dependent variables, specifying Mantel-Haenszel tests. The

adjusted odds ratio (OR) and 95% confidence intervals (CI) were derived from a conventional multivariate logistic regression analysis.

The PROC LOGISTIC statement was applied. By specifying the FAST option, PROC LOGISTIC eliminates insignificant variables without refitting the model repeatedly. This analysis uses a significance level of 0.2 (SLSTAY=0.2) to retain variables in the model. Owing to the small number of observations in the dependent variables categories, the following procedure was implemented during the multivariate logistic regression analysis: the analysis was conducted using all variables that were significantly associated with the particular dependent variable in the univariate logistic regression analysis. The first variable selected by the stepwise procedure was then excluded, and the procedure recalculated with the remaining variables. Finally, only those variables selected by each iteration were used and a stepwise multivariate logistic regression model was fitted to these variables. The analyses were not computed separately for men and women as only nocturnal coughing, nocturnal wheezing/tight chest and medically diagnosed TB were significantly influenced by the sex of the participants.

5.3 Results

The data presented here represent a more detailed analysis of the first national survey of the symptoms and prevalence of chronic lung disease in South Africa. Table 2 lists the characteristics of the 13 826 individuals from 6 457 households.

Table 3 summarises the crude prevalence of respiratory symptoms and conditions among South African adults. About 3.7% were reported as having doctor diagnosed asthma. The survey also reveals high incidence of other respiratory symptoms and diseases including wheeze and shortness of breath (11.1%), cough with phlegm (6.8%); chronic bronchitis (2.4%), nocturnal coughing (13.1%), nocturnal wheezing/tight chest (10.8%) and doctor diagnosed bronchitis (4.2%) and TB (2.7%). Nearly 1.7% of the respondents reported using asthma medication, whilst 0.5% was using TB medication.

Table 2 Characteristics of study population in terms of socio-demographic, active smoking, BMI, environmental and occupational exposure variables

Characteristics/feature	Percentage
Residence	
Urban	56
Rural	44
Connected to electricity	65
Cooking and heating fuels	
Electricity only	35
Electricity and biomass or fossil fuels	15
Biomass or fossil fuels	50
People per room	
< 2	40
≥ 2	60
Household going hungry	
Often	13
Sometimes	34
Seldom	5
Never	48
Covered by medical aid/medical benefit scheme	15
Payment of medicine	
Respondent	30
Family	4
Medical aid	23
Provided at clinic/public hospital	42
Employer	1
Age distribution in years	
15-23	48 (M*), 52 (F*)
24-35	40 (M), 60 (F)
36-51	41 (M), 59 (F)
52-95	37 (M), 63 (F)
Ethnic identity	
Black/African	76
Coloured	13
White	8
Asian/Indian	3
Ever smoked tobacco, used snuff or chewed tobacco	37
Ever smoked at least 100 cigarettes in lifetime	74
Years smoked on a daily basis – distribution	
1-7	
8-16	26
17-28	25
29-78	24
	25

** Male; Female*

Table 2 *(continues)*
Characteristics of study population in terms of socio-demographic, active smoking, BMI, environmental and occupational exposure variables

Characteristics/feature	Percentage
Frequency smoking	
Daily	18
Occasionally	72
Not at all	10
Household with smokers	36
Job with smokers	31
Ever worked in job where regularly exposed to smoke, dust, fumes or strong smells	21
Period worked in job exposed to smoke, dust, fumes or strong smells – distribution in years	
0-2	21
3-5	30
5-13	25
14-50	24
Gender	
Male	42
Female	58
Educational status	
≤ Primary school	43
Secondary school	51
Tertiary education (partly or completed)	6
Province of residence	
Western Cape	8
Eastern Cape	24
Northern Cape	9
Free State	9
KwaZulu-Natal	15
North West	9
Gauteng	8
Mpumalanga	9
Limpopo	9
Body Mass Index (BMI) $kg.m^{-2}$	
<22	37
22-24.9	21
25-27.4	13
27.5-29.9	9
30+	20

The effects (expressed in crude odds ratios) of various socio-demographic, environmental and occupational potential risk factors on prevalence of respiratory symptoms and conditions are summarised in Table 4.

The adjusted odds ratios are presented in Table 5. Race was the only potential predictor that remained significant for wheezing and shortness of breath. The risk of Asians/Indians increased now nearly 1.7 fold from the crude model, whilst Blacks/Africans were also significantly decreased compared to Whites. The results suggested that risk of nocturnal coughing was now significantly influenced by connection to electricity, covered by medical aid, educational status and household with smokers. The first three predictors were somewhat less beneficial, whilst the detrimental impact of the latter predictor increased somewhat compared to the crude model. Two potential risk factors remained significantly associated with nocturnal wheezing/thigh chest: Exposed at work to fumes, smoke, dust or strong smells along with educational status. The effect of these potential risk factors decreased somewhat from the univariate analysis.

Table 3 Crude prevalence (%) of respiratory symptoms and conditions in the survey population

Condition	Overall prevalence (%)
Wheezing and shortness of breath	11.1
Nocturnal coughing	13.1
Nocturnal wheezing/tight chest	10.8
Cough with phlegm	6.8
Chronic bronchitis	2.4
Medically diagnosed asthma	3.7
Medically diagnosed emphysema/bronchitis	4.2
Medically diagnosed TB	2.7
Currently using asthma medication	1.7
Currently using TB medication	0.5

The results hinted that cough with phlegm was significantly influenced in a protective manner by connection to electricity and detrimentally by years smoked on a daily basis along and BMI. The odds ratios decreased and increased somewhat for the protective and harmful potential predictors, respectively compared to the crude

Table 4 Potential risk factors for respiratory symptoms and conditions: Crude OR (95% CI)

Characteristics/feature	Wheezing and shortness of breath	Nocturnal coughing	Nocturnal wheezing/tight chest	Cough with phlegm	Chronic bronchitis	Medically diagnosed asthma	Medically diagnosed emphysema or bronchitis	Medically diagnosed TB
Residence in urban area	1.02 (0.84-1.23)	0.83 (0.75-0.92)*	0.74 (0.66-0.82)*	0.68 (0.56-0.83)*	0.64 (0.47-0.88)*	1.29 (1.07-1.54)*	1.42 (1.19-1.69)*	0.65 (0.53-0.80)*
Connected to electricity	1.11 (0.92-1.35)	0.78 (0.71-0.87)*	0.68 (0.61-0.76)*	0.63 (0.51-0.77)*	0.57 (0.41-0.78)*	1.10 (0.92-1.33)	1.34 (1.11-1.61)*	0.47 (0.38-0.58)*
Cooking and heating fuels								
Electricity only†								
Electricity and other fuels	1.17 (0.85-1.61)	0.83 (0.70-0.97)*	1.07 (0.89-1.27)	1.19 (0.87-1.62)	0.75 (0.43-1.30)	0.59 (0.44-0.78)*	0.39 (0.29-0.53)*	1.42 (1.00-2.03)
Other fuels only	0.92 (0.75-1.13)	1.15 (1.03-1.28)*	1.45 (1.29-1.64)*	1.75 (1.40-2.18)*	1.60 (1.11-2.32)*	0.69 (0.58-0.84)*	0.46 (0.39-0.56)*	2.18 (1.69-2.80)*
≥ 2 people per room	0.83 (0.69-1.01)	1.09 (0.99-1.21)	1.07 (0.96-1.19)	1.09 (0.89-1.34)	0.97 (0.70-1.34)	0.83 (0.69-0.99)*	1.13 (0.92-1.40)	0.67 (0.56-0.79)
Household going hungry								
Never†								
Seldom	1.70 (1.00-2.90)	0.82 (0.63-1.07)	0.98 (0.74-1.30)	1.28 (0.76-2.14)	0.98 (0.44-2.21)	1.10 (0.74-1.64)	0.22 (0.11-0.43)*	1.07 (0.60-1.91)
Sometimes	1.16 (0.94-1.44)	1.25 (1.11-1.39)*	1.52 (1.34-1.71)*	0.89 (0.72-1.11)	1.81 (1.26-2.62)*	0.99 (0.81-1.21)	0.46 (0.37-0.57)*	2.18 (1.72-2.77)*
Often	1.09 (0.82-1.45)	1.40 (1.20-1.63)*	1.83 (1.56-2.15)*	1.75 (1.28-2.41)*	2.61 (1.64-4.16)*	1.05 (0.80-1.39)	0.80 (0.62-1.03)	2.50 (1.85-3.37)*
Covered by medical aid/medical benefit scheme	0.93 (0.71-1.21)	0.76 (0.65-0.88)*	0.62 (0.52-0.74)*	0.85 (0.62-1.16)	0.85 (0.51-1.42)	1.75 (1.42-2.17)*	3.11 (2.60-3.72)*	0.33 (0.21-0.51)*
Payment of medicine								
Provided at public clinic/hospital†								
Respondent	0.87 (0.53-1.42)	1.04 (0.81-1.32)	0.97 (0.75-1.25)	1.38 (0.85-2.22)	1.32 (0.65-2.67)	1.19 (0.86-1.63)	1.04 (0.72-1.50)	0.52 (0.31-0.86)*
Family	1.40 (0.40-4.92)	0.75 (0.44-1.30)	1.19 (0.72-1.99)	0.88 (0.31-2.53)	0.86 (0.15-5.04)	0.95 (0.48-1.91)	0.73 (0.31-1.73)	0.47 (0.14-1.53)
Medical aid	0.48 (0.29-0.80)*	0.55 (0.40-0.74)*	0.43 (0.31-0.60)*	1.30 (0.68-2.46)	0.86 (0.31-2.42)	1.08 (0.76-1.53)	2.01 (1.43-2.84)*	0.18 (0.08-0.42)*
Employer	0.63 (0.12-3.23)	1.13 (0.39-3.25)	0.94 (0.30-2.93)	1.54 (0.27-8.63)	- (-)	0.93 (0.21-4.12)	0.58 (0.08-4.47)	2.68 (0.75-9.60)

*† Reference category * p<0.05 for stratum OR*

Table 4 *(continues)*
Potential risk factors for respiratory symptoms and conditions: Crude OR (95% CI)

Characteristics/feature	Wheezing and shortness of breath	Nocturnal coughing	Nocturnal wheezing/tight chest	Cough with phlegm	Chronic bronchitis	Medically diagnosed asthma	Medically diagnosed emphysema or bronchitis	Medically diagnosed TB
Age distribution in years								
15-23†								
24-35	1.41 (1.04-1.92)*	1.31 (1.11-1.54)*	1.26 (1.05-1.51)*	1.05 (0.76-1.44)	1.14 (0.62-2.09)	1.20 (0.89-1.63)	1.24 (0.91-1.67)	2.35 (1.56-3.55)*
36-51	1.71 (1.27-2.29)*	1.92 (1.64-2.25)*	1.78 (1.50-2.12)*	1.80 (1.32-2.46)*	1.59 (0.91-2.80)	1.90 (1.43-2.52)*	1.91 (1.43-2.53)*	3.87 (2.62-5.73)*
52-95	1.71 (1.30-2.26)*	2.73 (2.35-3.17)*	2.76 (2.35-3.25)*	1.85 (1.38-2.47)*	2.71 (1.59-4.60)*	2.70 (2.06-3.54)*	3.45 (2.65-4.48)*	4.29 (2.91-6.33)*
Ethnic identity								
White†								
Black/African	1.29 (0.96-1.73)	1.18 (0.96-1.44)	1.47 (1.17-1.85)*	1.07 (0.72-1.59)	0.68 (0.37-1.25)	0.39 (0.31-0.50)*	0.12 (0.10-0.15)*	3.56 (1.83-6.93)*
Coloured	1.29 (0.89-1.88)	1.94 (1.55-2.43)*	1.66 (1.27-2.15)*	1.01 (0.66-1.55)	0.34 (0.17-0.68)*	0.52 (0.38-0.72)*	0.36 (0.28-0.45)*	5.07 (2.52-10.18)*
Asian/Indian	4.31 (2.05-9.05)*	1.63 (1.19-2.23)*	1.63 (1.14-2.33)*	0.61 (0.31-1.22)	0.32 (0.09-1.14)	0.77 (0.49-1.19)	0.23 (0.15-0.37)*	1.06 (0.32-3.45)*
Educational status								
≤Primary school†								
Secondary school	0.74 (0.60-0.89)*	0.47 (0.42-0.52)*	0.48 (0.43-0.53)*	0.63 (0.51-0.77)*	0.64 (0.45-0.90)*	0.60 (0.50-0.72)*	0.91 (0.76-1.09)	0.41 (0.33-0.50)*
Tertiary education (partly or completed)	0.57 (0.37-0.88)*	0.29 (0.22-0.38)*	0.26 (0.19-0.37)*	0.48 (0.26-0.87)*	0.82 (0.30-2.27)	1.07 (0.77-1.49)	1.93 (1.46-2.55)*	0.10 (0.04-0.28)*
Male	0.90 (0.74-1.10)	0.71 (0.64-0.79)*	0.75 (0.67-0.84)*	1.01 (0.82-1.23)	1.02 (0.74-1.42)	0.90 (0.75-1.08)	0.92 (0.78-1.09)	1.37 (1.12-1.68)*
BMI								
<22	1.08 (0.83-1.42)	1.22 (1.06-1.40)*	1.30 (1.11-1.51)*	1.38 (1.05-1.83)*	1.13 (0.72-1.79)	1.10 (0.86-1.42)	0.98 (0.78-1.24)	2.30 (1.71-3.12)*
22-24.9†								
25-27.4	0.87 (0.62-1.23)	0.96 (0.80-1.15)	1.01(0.82-1.24)	1.24 (0.86-1.78)	0.60 (0.33-1.11)	0.95 (0.68-1.33)	0.98 (0.72-1.32)	0.87 (0.55-1.37)
27.5-29.9	1.06 (0.73-1.54)	0.92 (0.75-1.13)	0.96 (0.76-1.20)	1.18 (0.79-1.76)	1.15 (0.60-2.20)	1.09 (0.77-1.55)	1.04 (0.75-1.44)	1.16 (0.74-1.82)
30+	1.08 (0.81-1.46)	1.33 (1.14-1.56)*	1.40 (1.18-1.67)*	1.18 (0.86-1.62)	0.99 (0.59-1.66)	1.42 (1.08-1.86)*	1.23 (0.95-1.59)	0.85 (0.57-1.26)
Household with smokers	1.08 (0.89-1.31)	1.34 (1.21-1.48)*	1.19 (1.06-1.33)*	0.98 (0.81-1.20)	0.66 (0.47-0.91)*	1.18 (0.99-1.41)	1.16 (0.98-1.38)	1.04 (0.84-1.29)
Ever smoked tobacco, used snuff or chewed tobacco	1.13 (0.94-1.37)	1.73 (1.56-1.91)*	1.61 (1.45-1.80)*	1.40 (1.15-1.71)*	1.19 (0.86-1.63)	1.54 (1.29-1.84)*	2.26 (1.91-2.67)*	2.45 (1.99-3.01)*

*† Reference category * p<0.05 for stratum OR*

Table 4 *(continues)*
Potential risk factors for respiratory symptoms and conditions: Crude OR (95% CI)

Characteristics/feature	Wheezing and shortness of breath	Nocturnal coughing	Nocturnal wheezing/tight chest	Cough with phlegm	Chronic bronchitis	Medically diagnosed asthma	Medically diagnosed emphysema or bronchitis	Medically diagnosed TB
Ever smoked at least 100 cigarettes in lifetime	0.83 (0.60-1.15)	1.02 (0.86-1.21)	0.83 (0.70-0.99)*	1.05 (0.76-1.44)	1.05(0.64-1.72)	1.27 (0.93-1.73)	1.73 (1.29-2.32)*	1.06 (0.78-1.45)
Years smoked on a daily basis – distribution								
1-7†								
8-16	1.43 (0.89-2.30)	1.10 (0.85-1.43)	1.22 (0.91-1.63)	1.15 (0.73-1.84)	1.01 (0.44-2.34)	1.07 (0.68-1.70)	0.77 (0.52-1.13)	1.63 (0.99-2.69)
17-28	1.21 (0.78-1.90)	1.84 (1.44-2.35)*	1.78 (1.35-2.34)*	1.72 (1.08-2.75)*	1.20 (0.55-2.64)	1.35 (0.86-2.10)	1.31 (0.93-1.85)	2.44 (1.52-3.92)*
29-78	1.88 (1.22-2.91)*	2.32 (1.83-2.93)*	2.21 (1.70-2.88)*	1.99 (1.29-3.07)*	1.85(0.90-3.81)	2.22 (1.49-3.31)*	1.58 (1.13-2.19)*	2.50 (1.57-3.98)*
Frequency smoking								
Daily†								
Occasionally	1.08 (0.73-1.59)	1.14 (0.92-1.43)	0.97 (0.77-1.24)	0.98 (0.61-1.57)	0.96 (0.45-2.07)	0.65 (0.46-0.92)*	0.47 (0.36-0.62)*	0.73 (0.51-1.04)
Not at all	1.30 (0.72-2.33)	0.99 (0.71-1.38)	1.23 (0.88-1.73)	0.94 (0.49-1.80)	1.44 (0.51-4.05)	0.75 (0.44-1.28)	0.44 (0.27-0.71)*	0.51 (0.27-0.96)*
Job with smokers	0.86 (0.66-1.13)	1.17 (1.01-1.35)*	1.01 (0.86-1.18)	0.96 (0.72-1.28)	0.77 (0.47-1.26)	0.79 (0.61-1.03)	1.82 (1.45-2.30)*	0.70 (0.50-0.98)*
Ever worked in job where regularly exposed to smoke, dust, fumes or strong smells	1.96 (1.57-2.44)*	2.03 (1.82-2.26)*	1.97 (1.75-2.22)*	1.26 (1.02-1.57)*	1.16 (0.83-1.63)	2.56 (2.13-3.07)*	3.04 (2.57-3.61)*	1.99 (1.60-2.48)*
Period worked in job exposed to smoke, dust, fumes or strong smells – distribution in years								
0-2†								
3-5	1.94 (1.10-3.43)*	1.14(0.87-1.49)	1.23 (0.92-1.63)	1.34 (0.79-2.27)	1.32 (0.52-3.35)	0.87 (0.55-1.38)	1.22 (0.81-1.83)	0.97 (0.59-1.61)
5-13	1.30 (0.75-2.25)*	1.11 (0.84-1.47)	1.10 (0.81-1.48)	1.28 (0.73-2.26)	1.92 (0.74-4.96)	1.57 (1.02-2.42)*	1.12 (0.73-1.71)	0.97 (0.57-1.64)
14-50	1.22 (0.72-2.08)	1.13 (0.85-1.49)	1.06 (0.78-1.44)	1.40 (0.81-2.42)	1.38 (0.54-3.55)	1.49 (0.96-2.31)	1.90 (1.28-2.82)*	0.97 (0.57-1.65)

*† Reference category * p<0.05 for stratum OR*

Table 5 Potential risk factors for respiratory symptoms and conditions in adult population of South Africa: Adjusted OR (95% CI)

Characteristics/feature	Wheezing and shortness of breath (n=1535)	Nocturnal coughing (n=1816)	Nocturnal wheezing/tight chest (n=1491)	Cough with phlegm (n=937)	Chronic bronchitis (n=325)	Medically diagnosed asthma (n=514)	Medically diagnosed emphysema or bronchitis (n=578)	Medically diagnosed TB (n=378)
Connected to electricity	-	0.58 (0.37-0.92)*	-	0.61 (0.41-0.89)*	-	-	-	-
Household going hungry								
Never†								
Seldom	-	-	-	1.48 (0.58-3.59)	0.95 (0.42-2.16)	-	-	0.59 (0.14-2.51)
Sometimes				0.66 (0.45-0.96)*	1.72 (1.18-2.50)*			2.12 (1.30-3.46)*
Often				1.30 (0.75-2.27)	2.42 (1.50-3.90)*			2.21 (1.15-4.25)*
Covered by medical aid/medical benefit scheme	-	0.37 (0.22-0.62)*	-	-	-	-	-	-
Age distribution in years								
15-23†								
24-35	-	-	-	-	1.23 (0.66-2.29)	0.56 (0.31-1.02)	-	-
36-51					1.56 (0.88-2.76)	0.92 (0.54-1.57)		
52-95					2.53 (1.48-4.34)*	1.28 (0.75-2.17)		
Ethnic identity								
White†								
Black/African	1.89 (1.17-3.07)*	-	-	-	-	0.37 (0.23-0.58)*	0.23 (0.15-0.35)*	-
Coloured	1.68 (0.91-3.10)					0.44 (0.26-0.73)*	0.54 (0.35-0.83)*	
Asian/Indian	7.18 (1.63-31.64)*					0.63 (0.29-1.34)	0.21 (0.08-0.54)*	
Years smoked on a daily basis – distribution								
1-7†	-	-	-		-	-	-	-
8-16				1.19 (0.73-1.93)				
17-28				2.00 (1.23-3.27)*				
29-78				2.24 (1.42-3.55)*				
Household with smokers	-	1.85 (1.27-2.71)*	-	-	-	-	-	-

*† Reference category * p<0.05 for stratum OR*

Table 5 *(continues)*
Potential risk factors for respiratory symptoms and conditions in adult population of South Africa: Adjusted OR (95% CI)

Characteristics/feature	Wheezing and shortness of breath (n=1535)	Nocturnal coughing (n=1816)	Nocturnal wheezing/tight chest (n=1491)	Cough with phlegm (n=937)	Chronic bronchitis (n=325)	Medically diagnosed asthma (n=514)	Medically diagnosed emphysema or bronchitis (n=578)	Medically diagnosed TB (n=378)
Ever worked in job where regularly exposed to smoke, dust, fumes or strong smells	-	-	1.66 (1.17-2.36)*	-	-	2.34 (1.74-3.17*)	-	-
Period worked in job exposed to smoke, dust, fumes or strong smells – distribution in years	-	-	-	-	-	-		-
0-2†								
3-5							1.45 (0.83-2.54)	
5-13							0.92 (0.50-1.68)	
14-50							2.08 (1.21-3.58)*	
Educational status								
≤Primary school†								
Secondary school	-	0.59 (0.39-0.90)*	0.38 (0.26-0.55)*	-	-	0.57 (0.39-0.83)*	-	0.49 (0.30-0.78)*
Tertiary education (partly or completed)		0.20 (0.06-0.70)*	0.24 (0.11-0.52)*			0.47 (0.23-0.95)*		0.14 (0.02-1.02)
BMI								
<22				2.12 (1.36-3.29)*				3.58 (1.76-7.27)*
22-24.9†	-	-	-		-	-	-	
25-27.4				1.45 (0.80-2.64)				0.88 (0.27-2.90)
27.5-29.9				0.71 (0.35-1.42)				1.97 (0.65-6.00)
30+				1.42 (0.80-2.51)				1.96 (0.72-5.36)

*† Reference category * p<0.05 for stratum OR*

model. The significant level of household going hungry with cough with phlegm changed now from often (detrimental) to sometimes (protective) in the adjusted model. Chronic bronchitis was negatively, yet a bit less, influenced by household going hungry (sometimes and often stratum) and age (>51 years) compared to the crude model.

The results implied that medically diagnosed asthma was beneficially (yet weaker) influenced by race and educational status. Having some tertiary education was beneficial to lower medically diagnosed asthma prevalence. Exposed at work to fumes, smoke, dust or strong smells remained potentially detrimental (yet weaker) to medically diagnosed asthma. Two potential risk factors remained significantly associated with medically diagnosed emphysema/bronchitis: race (beneficial) and period exposed at work to fumes, smoke, dust or strong smells. (detrimental). The effect of these potential risk factors generally increased somewhat from the univariate analysis. The results inferred that medically diagnosed TB was potentially negatively influenced by household going hungry (sometimes and often) and BMI (22 $kg.m^{-2}$). Increased educational status may potentially reduce medically diagnosed TB, however the tertiary stratum was now insignificant compared to the crude model. The influence of educational status and BMI strengthened somewhat, whilst that of household going hungry weakened.

5.4 Discussion

Airway obstructive diseases and respiratory symptoms have increased world-wide.[25-27] The crude prevalence rate for chronic bronchitis (2.4%) from the 1998 SADHS was much lower compared to those from other developing countries (13-27%), but comparable to developed countries (3-17%).[28] Chronic bronchitis rates in men are considerably lower than those reported in working populations and in some general populations in Africa including South Africa, which range from 10-45%.[3]

The prevalence of asthma varies between countries but also between different areas within the same country.[25-27,29] Asthma is not necessarily more prevalent in industrialised than non-industrialised countries (Global Initiative for Asthma, 2004). Nriagu et al reported a rate of 12% for doctor diagnosed asthma amongst adults living in the highly industrialsed area of Durban South, South Africa.[13] The report issued by the Global Initiative for Asthma (GINA) estimated the mean

prevalence of clinical asthma in Southern Africa as 8.1%, compared to the crude rate of 3.7% found in the SADHS.[19,27] The prevalence of asthma is higher in Southern Africa than in many other regions in Africa. South Africa is 25th on the list of 84 countries in terms of asthma prevalence and 5th out of 49 countries in terms of asthma mortality rates. Asthma prevalence rates for other developing countries are: Brazil (11.4%), Mexico (3.3%), Nigeria (5.4%), India (3.0%), China (2.1%).[27]

In Southern Africa, mining-related diseases such as pneumoconiosis remain the leading occupational respiratory diseases, but occupational asthma is becoming increasingly prevalent as non-mining industrialisation expands. Occupational asthma now represents the second most frequently reported occupational respiratory disease.[19]

Globally only six countries have more cases of TB than South Africa (243 000 cases, 0.55% compared to population of 44 million). These are India (1 820 000 cases), China (1 448 000 cases), Indonesia (582 000 cases), Bangladesh (328 000 cases), Nigeria (275 000 cases) and Pakistan (247 000 cases).[30]

Nriagu et al reported self-reported prevalence rates for wheezing and chronic phlegm as 37±40% and 31±32% amongst adults from Durban South, South Africa, respectively.[13] The SADHS rate for wheeze and shortness of breath (11.1%) was comparable to those from the European Community Respiratory Health Survey (ECRHS), which ranged from 3.0% (Mumbai, India) to 16.1% (Melbourne, Australia).[31] Nocturnal cough (13.1%) compared well to the rate from Mumbai, India (11.2%) and Athens, Greece (17.8%), but was much lower to those reported at Portland, USA (32.5%) and much higher than rural Beijing, China (2.6%).[31,32] The SADHS rate for nocturnal tight chest (10.8%) was comparable to those from Wellington, New Zealand (10.4%) and Melbourne, Australia (11.4%), but higher than Algiers, Algeria (4.4%) and rural Beijing, China (1.4%).[31,32] The SADHS rate for coughing with phlegm (6.8%) was lower compared to that reported by Langhammer et al (8.3% for 20-44 year olds from Norway), but higher than those from rural Beijing, China (1.9%).[32,33]

The univariate logistic regression analyses suggested that the prevalence of the various respiratory symptoms and conditions were influenced differently by the

range of socio-demographic, environmental and occupational potential risk factors. The multivariate logistic regression analyses suggested that the prevalence of respiratory symptoms and diseases may potentially be diminished in South Africa by health promotion predictors (increasing connection to electricity, having a medical aid and improved education). The following potential risk factors should be lessened in order to have a beneficial influence on the prevalence rates of respiratory symptoms and diseases: households going hungry, years smoked, households with smokers, exposure at work to fumes, smoke, dust or strong smells and period worked in such a job as well as BMI increase for the underweight and decrease of the obese. Other potential risk factors included age and race. One aspect of the chronic disease prevention that has been particularly successful in South Africa has been the introduction of strong tobacco control legislation At the time of the survey, South Africa did not have any comprehensive strong anti-tobacco legislation that was enforced in public places and the working environment. The legislation only came into force on 1 January 2001.[34] Tobacco control initiatives have increased dramatically in South Africa, especially since 1994. In 1993, the first Tobacco Products Control Act was passed and in 1999 the Tobacco Products Control Amendment Act.

The risk of acquiring chronic bronchitis increased with increasing age. This is in agreement with studies from Nepal and England.[35,36] Studies from the United States and Canada, however, have failed to show an increase with age.[37,38] Campello et al also did not observe an increase of risk for asthma and asthma-like symptoms amongst Italian adults (as observed in this analyses), whilst Zhang et al did amongst adults (≥15 years) in rural Beijing, China.[32,39]

The risk for nocturnal cough, nocturnal wheezing/tight chest, asthma and TB diminished with increasing educational status. The results are consistent with those from a cross-sectional analysis in Hordaland County in Sweden after adjustment for sex, age, smoking, and occupational exposure.[40]

In general, White people had a lower risk for wheezing and shortness of breath compared to the other ethnic groups, except for asthma and emphysema/bronchitis. In contrast, the Global Burden of Asthma report reported that asthma mortality rates are disproportionately higher among Africans and Coloureds.[27] It further

reported that the majority of asthma deaths in the region occur outside hospitals. Poor availability of health care, poor transport and emergency services and inadequate home management of acute asthma are therefore recognised as important contributing factors.

The SADHS results revealed that households going hungry often and sometimes were more at risk for chronic bronchitis and TB compared to households never going hungry. A recent review by Brug et al documented that there are some indications from epidemiological studies on the potential protective role of some nutrients, high intake of fish and fresh fruits in the development of COPD related diseases and symptoms.[41-46] The evidence is not extensively sufficient to justify dietary recommendations for primary prevention of COPD. Most of the evidence refers to omega-3 fatty acids, vitamins C and E, which have an antioxidant action that may supposedly counteract the oxidative damage produced by exposures like smoking and air pollution.[47]

Being connected to electricity significantly reduced the risk for nocturnal coughing, and cough with phlegm. It is estimated that two-thirds of the households in the developing world are still primarily dependent on biomass and fossil fuels in conditions of inadequate ventilation. These household conditions have the potential to produce high concentrations of indoor air pollution, which are many times higher than outdoor concentrations.[48,49] Very few quantitative environmental exposure assessment or analytical epidemiological studies have been conducted in South Africa. Most of the studies also focused on children as study population. Exposure is usually based on a proxy measures, such as smoking status or use of biomass fuels for space heating.[13,14,17,18]

Gas use was included in the "other fuels" category during this investigation, as there is evidence that people who use gas for cooking have reduced lung function than those who use electricity for cooking.[50,51] NO_2 is the main pollutant produced during unvented gas cooking.

The prevalence of nocturnal coughing was significantly elevated in households with smokers. ETS is a common indoor exposure in many countries and it is a major contributor to indoor RSP concentrations.[52] It was estimated that 37% of the South

African respondents had ever smoked, used snuff or chewed tobacco, 36% were living in a home where someone smoked and 31% had a job with smokers (Table 2). The ECRHS study indicated that between 8.6% (Umeå, Sweden) and 50.6% (Galdakao, Spain) of respondents are exposed to ETS at home.[53]

The research on health effects of ETS has expanded since the 1980's. Among children there is relatively strong evidence showing that parental smoking is associated with respiratory symptoms.[54-57] In contrast, the studies carried out in adult populations have provided more inconsistent results. Some studies did not observe significant associations.[58,59] However, far more cross-sectional studies have shown increased occurrence of chronic respiratory symptoms and deficits in ventilatory lung function in relation to ETS exposure at home and/or at work.[60-63]

It was found that the prevalence of wheeze/tightness of chest and asthma was significantly elevated when respondents were exposed to smoke, dust, fumes or strong smells at work. This was also observed for emphysema/bronchitis and period worked in a job where regularly exposed to smoke, dust, fumes or strong smells. While there are a number of specific work exposures that have been shown to cause fibrosis of the lung[64-67] and chronic bronchitis[68,69], there is more general evidence associating work in dusty occupations with COPD.[70] Work-related asthma is one of the most common occupational lung diseases worldwide.[67,71] Menezes et al reported a significant increase in risk of chronic bronchitis due to occupational exposure to dust (OR = 2.48, 95% CI 1.56 to 3.94).[72]

The key to prevention of occupational respiratory disease is the control of occupational air pollution. This requires the enforcement of engineering and other workplace control solutions as required by legislation. However, a large proportion of South Africans are employed in the informal sector, where this legislation does not apply. A revamp of the administration system for occupational diseases, whose flaws currently signify a main blockage to the coverage and management of occupational diseases, is required.[73] The division of compensation and preventions systems further enhances to incompetence.

The adjusted risk models indicated no significant relationship between BMI and TB and cough with phlegm. The SADHS study differs from others in that it describes

data from a population of diverse ethnic and socioeconomic background. However, it is difficult to compare the risks pose by being obese (BMI $\geq$ 30 $kg.m^{-2}$) to those reported by other studies as different reference groups are applied. The role of diet and sedentary indoor lifestyle have been speculated in asthma development.[74,75] Obesity can directly affect the airway caliber through the chest wall restriction. Narrowing of airway and the reduction of lung volume have been associated with bronchial hyperreactivity.[76] Pooled data from three large epidemiologic studies in Australia found that underweight individuals had an increased risk for asthma, respiratory symptoms and airway hyperresponsiveness, whereas obese subjects had increased risk for asthma and respiratory symptoms, but not airway hyperresponsiveness. Gender differences were not reported.[77] Celedon et al found among adult men and women living in rural China that both underweight and overweight were associated with an increased risk of asthma.[78]

There are some important limitations in this study, which should be taken into account when interpreting the results. The SADHS had a cross-sectional design. Cross-sectional studies are weak to prove causation as they are subject to difficulties interpreting the temporal sequence of events since health status and risk factors are measured simultaneously.

Reliance on self-reported data does, however, carry a risk of differential or nondifferential misclassification of disease and exposure status resulting in statistical significance arising by chance. However, the biological plausibility of the potential risk factors has been addressed. Consequently the direction of bias on the association is not easy to predict. Respondents with current symptoms and diseases may be more likely to report exposures and remember past TB infections than asymptomatic respondents.

Self-reporting of emphysema and bronchitis can be used only as a very rough guide to the prevalence of chronic lung diseases for a variety of reasons. First, use of diagnostic terms reflects health service access, which in South Africa varies considerably by socio-economic status and geography. A term such as emphysema is likely to be used inconsistently by medical practitioners based on varying clinical criteria. Lung function testing, which contributes important information to diagnosis, is uncommon at primary care level. Bronchitis also is a non-specific term

that would elicit reports of acute bronchitis as well as chronic bronchitis. Acute bronchitis is a common ailment, often a mild and self-limiting viral infection, which may occur without underlying chronic disease. A literature review of asthma symptoms assessed by questionnaire found that "physician-diagnosed asthma" had a mean specificity of 99% and a mean sensitivity of 68% for asthma defined by symptoms, suggesting that underdiagnosis is more likely than overdiagnosis.[79] Finally, asthma in adults is probably frequently misdiagnosed as bronchitis. On the one hand, self-reporting of asthma is likely to reflect some degree of under-diagnosis. On the other hand, asthma rates may be inflated by confusion with emphysema and chronic bronchitis, particularly in older age groups. Reporting of symptoms is less likely to be influenced by contact with health services than is reporting of diagnoses. The chronic bronchitis symptom complex is defined by chronic bronchitis every day for at least 3 months a year, for at least 2 successive years. It was one of the earliest symptom complexes to be defined by standard respiratory questionnaires, and has entered into common usage as both a clinical and epidemiological definition.

Furthermore, other factors that might contribute to adult respiratory health, such as outdoor and indoor air pollution (e.g. location of household close to industry, transportation sources or waste fill sites, insecticide or fertiliser use, allergens such as pollen, dust, fungal spores from mildew and moulds), meteorological variables (precipitation, temperature, humidity), the current HIV/AIDS epidemic and respiratory infections, were not recorded. Excluding these risk factors from the analysis might introduce substantial bias (differential or nondifferential). Thus the direction of bias on the calculated association measures is not easy to predict. The definition of a confounder is important to remember: it must be associated with both the exposure variable of interest and the health effect. As the association among these different risk factors and the investigated potential risk factors is not readily available from the literature, it is impossible to predict the direction of the potential bias on the association measure.

Differential and/or nondifferential misclassification may have influenced the risk estimates for cooking and heating fuel use. Many households in South Africa in general use a combination of cooking and heating fuels. The calculated effects may be underestimated if only considering using high polluting fuels (wood and dung)

exclusively and not in combination with paraffin, coal, LPG/natural gas and/or electricity. However, none of the households under investigation used wood or dung exclusively or paraffin or coal exclusively. No quantitative exposure assessment (including duration of exposure, as reflected by frequency and duration of fuel use for heating and cooking per day) was conducted during the SADHS. It is recommended that future SADHS should separate the type of fuels use for cooking and heating in two separate questions. Exposure to smoke from polluting fuels during heating is much longer than exposure during cooking.

The most common indicators used for measurement of socio-economic status are income level, occupation and educational level.[80] Demographic and Health Surveys traditionally do not include questions on income and expenditure. Educational level measures one aspect of socioeconomic status and we cannot rule out that the results would have been different with another measure. However, the relation between socioeconomic status (SES) and socioeconomic factors with respiratory health in adults is not well understood. Existing studies are heterogeneous regarding the definition of the socioeconomic indicators used. Possibly, a more ideal measure would be one that took into account several aspects, e.g. educational level and household assets.

Another bias is that employed low-income men are underrepresented, as they work overtime, shifts or away from home. Male worker hostels, a common form of housing for African migrant workers in mining and certain urban areas, were not surveyed. Nevertheless the results presented here are the first national survey of the symptoms and prevalence of chronic lung disease in South Africa. Previous morbidity information was derived from surveys of selected adult populations only.[3,81]

During the analysis it was assumed that confounding is additive and not multiplicative. If confounding is additive, then the confounding variable would produce the same additional risk of a health outcome in the exposed and unexposed; but if the health outcome is rare in unexposed, it would follow that the confounder might account for a much larger proportion of health outcome in that group. Conversely, if two exposures act multiplicatively, the proportional increase in health outcome rates due to confounding would be the same in exposed and unexposed;

but if the health outcome is more prevalent in the exposed group, the absolute increase would be larger in the exposed. This issue thus has important risk assessment and public health policy implications.

There is a deficiency of local studies investigating risk factors for adult respiratory health. In order to improve respiratory health of the unique South African adult population through epidemiological studies, it is imperative that future studies should attempt to minimise systematic and random errors and subsequently strengthen their validity and accuracy. Yach et al addressed the methodological difficulties in undertaking epidemiological studies in developing countries.[82] They pointed out the use of ecological and cross-sectional studies in determining the relationship between risk factors and disease and consequently applying detailed analytical studies to determine the reasons for these relationships. In South Africa, detailed analytic epidemiology studies will have to compete with the demands on the public and research purse for work on common diseases of pressing current importance (e.g. HIV/AIDS). Therefore analytical studies should not merely redocument the impact of known risk factors, but should provide a basis for designing interventions.

In conclusion, although there is potential for residual confounding despite adjustment in this preliminary analysis, the documented international evidence on most of the potential risk factors suggests that these associations may be real. It is trusted that more detailed South African analytical intervention studies will scrutinise these results in order to develop integrated intervention programmes to improve adult respiratory health in the country.

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Chapter 6

POTENTIAL IMPACT OF COOKING AND HEATING FUEL USE ON ACUTE RESPIRATORY HEALTH OF PRESCHOOL CHILDREN IN SOUTH AFRICA

Background: Dependence on polluting fuels (wood, coal, crop residues, animal dung, paraffin) for cooking and heating exposes countless women and young children in developing countries to elevated air pollution concentration indoors. This study explored the connection between polluting fuel use for cooking and heating with childhood (<5 years) acute respiratory infections (ARI) in South Africa.

Methods: Analysis is based on data from 4 679 children living in 2 651 households collected during the 1998 South African Demographic and Health Survey. Cases were defined as those who experienced cough accompanied by short, rapid breathing during the 2 weeks prior to the survey. Logistic regression was applied to estimate the odds of suffering from ARI among children from households using high (wood or dung in combination with other fuels) and medium polluting fuels (coal or paraffin in combination with electricity or liquid petroleum gas/natural gas) relative to those from households using electricity or liquid petroleum gas/natural gas exclusively, after controlling for potentially confounding factors.

Results: Two-thirds of children lived in households using high and medium polluting fuels. Nineteen percent suffered from ARI. After adjustment, children in households using high and medium polluting fuels for cooking and heating were 26-29% more likely to have an ARI event than children from households using cleaner fuels (OR 1.26; 95% CI : 1.00-1.58 and OR 1.29; 95% CI : 1.02-1.62, respectively).

Conclusions: Although there is potential for residual confounding despite adjustment, the better documented international evidence on indoor air pollution and ARI suggests that this association may be real. As nearly half of households in South Africa still rely on polluting fuels, the attributable risk arising from this association, if confirmed, could be substantial. It is trusted that more detailed analytical intervention studies will scrutinise these results in order to develop integrated intervention programmes to reduce children's exposure to air pollution emanating from cooking and heating fuels.

6.1 Introduction

The ultimate endeavour of epidemiology is to identify modifiable determinants of disease occurrence and progression and to contribute in testing the efficacy and effectiveness of interventions on these determinants.

A study conducted in 1990 reported that acute respiratory infections (ARI)(such as pneumonia) were then the principal cause of death amongst young children in large parts of South Africa.[1] This is supported by international findings that ARI are a leading cause of childhood illness and death worldwide, accounting for an estimated 6.5% of the entire global burden of disease.[2] In addition there is no simple and rapid treatment for ARI as is the case with diarrhoeal disease and oral rehydration therapy.

Respiratory ill health is the main reason for use of the health services in the country. However, much of what must be done to prevent respiratory symptoms and diseases lies outside of the sphere of health care. Therefore interventions should be targeted at risk factors and determinants, rather than only providing medical treatment for those already affected. South Africa, a middle income country, is faced by health risk factors from a First World situation (e.g. industry, traffic, aging population) along those from a Third World situation (e.g. domestic burning of wood, animal dung, crop residues, coal and/or paraffin, poor sanitation, overcrowding). Thus intervention strategies deduced from studies conducted in developed countries are not merely applicable in this country.[3]

Wood, animal dung, coal, crop residues and paraffin (hereafter "polluting fuels") are at the bottom of the energy ladder regarding combustion efficiency and cleanliness, yet 41%, 48% and 23% of 11 million South African households used these polluting fuels for cooking, heating and lighting, respectively during 2001, even when access to electricity was available.[4,5] Smoke from fossil and biomass combustion produces numerous air pollutants that are detrimental to health, including respirable particulate matter (RSP, PM_{10}), carbon monoxide (CO), nitrogen oxides (NO_x), formaldehyde, benzene, 1,3 butadiene, polycyclic aromatic hydrocarbons (PAHs)(such as the carcinogen benzo[a]pyrene, B[a]P), and many other toxic volatile organic compounds

(VOCs). The fuels are typically burned in simple, inefficient and mostly unvented household cookstoves, which, combined with poor ventilation, generate large volumes of smoke indoors. A local study by Thomas et al reported that on average 14% of households had children (<6 years) usually or always present when their mothers were cooking.[6] This percentage increased to 18.3% of households in the lowest wealth quintile. Paraffin stoves were burning in the lower wealth quintile homes on average 4 hours per day, with the evening meal taking over half of this time to prepare. Muller et al established in a local study that people spend on average 2 hours cooking indoors during both winter and summer.[7] Consequently children and women are exposed to much higher air pollution levels indoors than from outdoor sources.

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, including paraffin, was used.[5] The mean maximum hourly average was 28 $\mu g.m^{-3}$ (range 0–451 $\mu g.m^{-3}$) for NO_2, 1 414 $\mu g.m^{-3}$ (range 0-17 723 $\mu g.m^{-3}$) for SO_2 and 34 $mg.m^{-3}$ (range 0-388 $mg.m^{-3}$) for CO. The number of households where standards were exceeded by the maximum hourly averages by NO_2 and CO was six (9%) and 20 (30%) respectively (Hourly WHO standard of 200 $\mu g.m^{-3}$ and 30 $mg.m^{-3}$, respectively) and for SO_2 the number was 28 (42%) (Hourly Californian standard of 655 $\mu g.m^{-3}$)(no WHO or US EPA standard for maximum *hourly* average for SO_2 exists).[8,9] Total suspended particulates (TSP) concentrations ranged from a minimum of 7 $\mu g.m^{-3}$ to a maximum of 433 $\mu g.m^{-3}$. Comparisons of TSP concentrations with international standards have not been made, as these are now focused on PM_{10} and $PM_{2.5}$ particles. No *hourly* standards or guidelines also exist for PM_{10} and $PM_{2.5}$. Röllin et al provided scientific evidence that even in partially electrified homes in South Africa levels of RSP were significantly lower (mean 77 $\mu g.m^{-3}$, median 37.5 $\mu g.m^{-3}$) relative to their non-electrified counterparts (mean 162 $\mu g.m^{-3}$, median 107 $\mu g.m^{-3}$)($p=0.012$) during summer.[10] Stationary (kitchen CO) levels in un-electrified and electrified dwellings ranged from 0.36-20.95 ppm and 0-11.8 ppm, respectively. The mean level of log (CO) in the kitchen was significantly higher in the un-electrified areas (1.25 vs. 0.69) ($p=0.0004$). The mean level of log (CO) for personal measurements conducted on

children (<18 months) was higher in the un-electrified areas (0.83 vs. 0.34) ($p<0.0001$).

The mechanisms by which polluting fuel smoke can boost the risk of ARI is not entirely known, however exposure has been associated with diminished pulmonary immune defence mechanisms.[11-14] Tobacco smoke also has been indicated to cause poor immune system responses.[15-19] Exposure to one of the numerous pollutants in biomass smoke, PM_{10}, has been reported to provoke a systemic inflammatory response that includes stimulation of the bone marrow, which can play a role in the pathogenesis of the cardiorespiratory morbidity. [11-14] Exposure to PAH, especially B[a]P, can cause immune suppression and can raise the risk of infection and disease.[15-17] Acute and chronic exposures to NO_x can increase bronchial reactivity and susceptibility to bacterial and viral infections.[18,19] It is thus probable that persistent exposure to high levels of polluting fuel smoke can weaken the pulmonary defence mechanisms, reduce lung function and intensify children's susceptibility to ARI.

International studies have reported strong associations between exposure to polluting fuel smoke and ARI in preschool children.[20-23] However, a few studies of preschool children have failed to find a relationship between polluting fuel smoke and ARI.[24,25] Notwithstanding warnings of high levels of indoor air pollution in informal and traditional housing in South Africa, barely any comprehensive epidemiological studies have been embarked on in the country. Two local studies established a significant link with indoor air pollution indicators and respiratory health of preschool children[26,27], whilst two other local studies did not.[28,29] Wichmanna and Voyi concluded in a review that most of the local studies have dealt inadequately with confounding factors.[30] It is thus advisable to apply caution when interpreting results from local studies.

The current study will attempt to add to the current body of knowledge regarding indoor air pollution exposure and respiratory health of preschool children. The study is based on the 1998 South African Demographic and Health Survey (SADHS).[31] The 1998 SADHS is the first national health survey conducted across the entire country. Data from this survey provided the opportunity to examine the prevalence and

determinants of various respiratory symptoms and diseases in a representative national population rather than a selected high risk population, as has been the case in most previous studies in developed countries.

This study examines the association between household use of fuels for cooking and heating and ARI prevalence in children (< 5 years), whilst considering a number of confounders or effect modifiers. It is trusted that more detailed analytical intervention studies will scrutinise these results in order to develop integrated intervention programmes to reduce children's exposure to air pollution emanating from cooking and heating fuels.

6.2 Materials and methods

6.2.1 Survey method

The 1998 SADHS had a cross-sectional design and was a national household survey of the population living in private households in all 9 provinces of South Africa. Detailed information on the survey design is outlined elsewhere.[31] The sampling frame for the SADHS was the list of approximately 86 000 enumeration areas (EAs) created by Central Statistics (now Statistics South Africa, SSA) for the Census conducted in October 1996. The EAs, ranged from about 100 to 250 households and were stratified by 9 provinces, urban and non-urban residence and by EA type. The number of households in the EA served as a measure of size of the EA. The first stage (proportional stratified sampling) of the two-stage sampling led to a total of 972 enumerator areas (EAs) being selected for the SADHS (690 in urban areas and 282 in non-urban areas). The second stage involved a systematic random sample of 10 and 20 houses in selected urban and rural EAs, respectively. Oversampling was conducted in some areas to enable inference to be made about differences across provinces and race – and in the Eastern Cape province, across health districts.

In addition to the main survey of households a women's health questionnaire was administered individually to women (15-49 years) in all the households selected for the main survey (Refer to Appendices 1 and 3). The SADHS questionnaires were translated into 9 of the 11 official languages of South Africa and checked by backtranslation.[31]

The questionnaires were pretested in November/December 1996 as part of a pilot study.

Interviewers were trained over several weeks. Interviews were conducted after working hours. Interviewers were instructed to return twice if a suitable respondent was not found at home. Fieldwork commenced late January 1998 and was completed in September 1998. The response rate at the household level was 97% of 12 860 households in 966 EAs. For the women's health survey, the overall response rate was 92.3%.

Ethical approval was granted by the Ethics Committee of the South African Medical Research Council to conduct the 1998 SADHS. The survey obtained informed consent from each respondent (in this case, mothers of the children included in the women's health questionnaire) before asking questions. The analysis presented in this paper is based on existing survey data with all identifying information removed.

6.2.2 Health outcome, exposure and confounder variables

For each child (<5 years), the mother was asked if the child had been ill with coughing in the 2-week period preceding the survey interview. For children who had been ill with coughing in the last 2 weeks, the mother was additionally asked if the child, when ill with coughing, breathed faster than usual with short, rapid breaths. Children who suffered from coughing accompanied by short and rapid breathing at any time during the last 2 weeks are defined as having suffered from an acute respiratory infection. This reported prevalence of ARI is the response variable in our analysis.

Exposure to cooking and heating smoke was ascertained indirectly by type of fuels used. This was the main independent variable. The survey question was, 'What does your household use for cooking and heating?' Respondents indicated all the different fuel types that were used. The households were grouped into three categories representing the extent of exposure to cooking and heating smoke—high pollution fuels (if either wood or dung was used in combination with paraffin, coal, liquid petroleum gas (LPG)/natural gas or electricity), medium pollution fuels (if either paraffin or charcoal

was used with LPG/natural gas or electricity) and low pollution fuels (if LPG/natural gas or electricity was used exclusively).

Confounders pertaining to a child included: age (in months, categorised in 5 groups), sex, birth order (categorised in 4 groups) and number of children in the household. Confounders related to the biological mother of a child included: age at birth (in years, categorised in 3 groups), education (in years, categorised in 3 groups) and ethnic identity (Africa/Black, White, Coloured, Asian/Indian). 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 ethnic 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, socio-economic status and educational status. Household standard of living index (SLI) is calculated by using the approach of Mishra by adding the following scores: 3 for a car or tractor; 2 each for a scooter/motorcycle, TV, telephone, refrigerator, piped/public tap water, flush toilet, electricity, wood/vinyl/asphalt/ceramic/cement/carpet of main floor material; 1 each for a bicycle, radio.[32] Index scores range from 0–2 for low SLI, 3–8 for medium SLI, 9–21 for high SLI.

The location of the household was categorised on an urban/rural level.

6.2.3 Data analysis

Data from the household and women's health questionnaires were merged in this analysis. The merged data file had 5 093 observations. Statistical analyses were conducted using STATA version 8. A small residual category of other fuels used for cooking and heating (n=6, 0.13% of the sample, N=5 093) was excluded from the analysis due to unknown nature of fuels in that category. All children from multiple births (n=133, 2.61% of the sample, N=5 093) were excluded from the analysis as well as all children who had passed away (n=269, 5.28% of the sample, N=5 093). Eventually 4 679 children from 2 651 households were included in the analyses. The 1998 SADHS report pointed out that the risk factors might be correlated with each

other.[31] Independence among risk factors was investigated with χ^2 tests. It was observed that most of the risk factors were significantly correlated at the 95% confidence level, although very poorly with correlations coefficients varying from 0.01 to 0.40. Consequently conventional logistic regression analysis was conducted, instead of a conditional analysis. Simple descriptive statistics were used to describe the characteristics (TAB command) of the sample and in calculating the prevalence of ARI for each characteristic (SVYTAB command).

The crude odds ratio (OR) and adjusted OR along with 95% confidence intervals (CI) were derived using the SVYLOGIT procedure. A weighting factor was applied to all observations to compensate for over-sampling of certain categories of respondents in the study design. The estimation of CI accounts for design effects due to clustering at the EA, provincial and household level. The adjustments for clustering at the EA and provincial levels were done using the SVYSET command.

6.3 Results

The data presented here represent a more detailed analysis of the first national survey of the symptoms and prevalence of ARI amongst preschool children in South Africa. Table 1 lists the characteristics of the 4 679 children from 2 651 households.

Nearly 40% of children live in households that use high polluting fuels compared to 27% and 34% who live in households using medium and low pollution fuels, respectively (Table 1). Children are relatively equally distributed by sex. Children aged 0-11 months are somewhat less represented compared to the other age groups. The percentage of children in the sample decreases from birth order 1 to 3. A quarter of children are born at birth order >3. Less than half of the children live in homes with other children (43%). Approximately the same number of children is born to mothers aged 15–24 and 25-34 years. More than half (57%) of them have mothers with 3-6 years of education at the time of the survey. The majority of the children (48%) live in high standard of living households, followed by medium (36%) and low (16%) SLI homes.

Table 1 Sample distribution of South African children (<5 years) by selected characteristics, reported prevalence of acute respiratory infections (ARI) during the 2 weeks preceding the survey and crude odds ratios

Characteristic	Sample distribution (%)	ARI prevalence (%)	OR (95% CI)
South Africa	-	19.26±3*0.76	-
Cooking and heating fuel type			
Low polluting*	33.71	16.24	-
Medium polluting	26.86	19.32	1.28 (1.02-1.61)
High polluting	39.43	18.68	1.25 (1.00-1.57)
Age of child (in months)			
0-5*	10.86	18.90	-
6-11	10.84	23.08	1.24 (0.90-1.71)
12-23	20.39	22.54	1.15 (0.85-1.56)
24-35	19.81	18.02	0.89 (0.65-1.23)
36-59	38.11	13.91	0.65 (0.48-0.88)
Sex of child			
Boy*	50.37	17.82	-
Girl	49.63	18.22	1.08 (0.92-1.28)
Birth order			
1*	33.70	17.88	-
2	24.56	17.75	1.06 (0.84-1.33)
3	15.26	19.61	1.16 (0.89-1.50)
>3	25.48	17.51	0.97 (0.75-1.24)
Number of children per household	56.66	19.95	-
1*	43.34	15.48	0.70 (0.57-0.85)
>1			
Mother's age at childbirth			
15-24*	39.90	18.05	-
25-34	42.62	18.30	1.01 (0.83-1.22)
35-49	17.48	17.24	1.01 (0.76-1.33)
Mother's education (in years)			
<3*	24.11	20.21	-
3-6	57.47	17.78	0.96 (0.77-1.20)
>6	18.42	15.89	0.86 (0.64-1.14)
Ethnic identity			
Black/African*	80.81	17.77	-
Coloured	11.97	18.39	1.02 (0.78-1.33)
White	4.38	21.46	1.36 (0.86-2.16)
Asian/Indian	2.27	16.98	0.95 (0.49-1.84)
Household standard of living			
Low*	16.03	17.73	-
Medium	36.25	17.87	0.97 (0.70-1.34)
High	47.72	18.23	0.99 (0.73-1.34)
Residence			
Urban*	45.18	18.07	-
Rural	54.82	17.97	1.06 (0.88-1.29)
Number of children	4 679	4 679	4 679

** Reference category*

Roughly the same number of children lives in urban and rural areas. The majority of the children are classified as African/Black, followed by 12% as Coloured, 4% as White and 2% as Asian/Indian. The survey population does not quite reflect the ethnic make-up of the South African population (all ages) for Whites and Coloureds according to the 2000 Census data (Africans (77.2%), Whites (10.5%), Coloureds (8.8%) and Indians (2.5%).[4]

Nineteen per cent of children (<5 years) had an ARI event during the 2 weeks preceding the survey. The reported prevalence of ARI is somewhat higher among children living in dwellings using medium and high polluting fuels (19%) than among those living in households using low polluting fuels (16%) (Table 1). Children aged 6–23 months are somewhat more probable to have experience an ARI event than children under 6 months of age or older children.

The reported prevalence of ARI is higher for White children compared to the other ethnic groups. Children living in households with other children are less prone to have an ARI event (15% compared to 20%). Children with birth order 3 have a higher prevalence rate of ARI compared to the other groups (20% compared to 18%). Children whose mothers have less than 3 years of education at the time of the survey have more ARI events compared to the other groups. Prevalence of ARI does not fluctuate much by sex of child, mother's age at childbirth, household SLI or urban/rural setting.

Of all the variables, only cooking and heating fuel type, age of child and number of children in households are significantly associated with ARI in the unadjusted analyses (Table 1). Children living in households using medium and high polluting fuels are 25-28% more likely to have an ARI event compared to those living in households using low polluting fuels for cooking and space heating (OR 1.28; 95% CI : 1.02-1.61 and OR 1.25; 95% CI : 1.00-1.57, respectively). Children aged 36-59 months are 35% less likely to have an ARI event compared to the other age groups (OR 0.65; 95% CI : 0.48-0.88). Children living in households with other children are 30% less likely to have an ARI event (OR 0.70; 95% CI : 0.57-0.85).

In the adjusted analysis, high polluting fuel use (OR 1.29; 95% CI : 1.02-1.62), the oldest child age category (OR 0.66; 95% CI : 0.49-0.89) and having more than one child living in a household (OR 0.69; 95% CI : 0.56-0.83) are statistically associated with ARI (Table 2).

Table 2 Adjusted odds ratio estimates of cooking and heating fuel type and other risk factors on acute respiratory infection (ARI) among South African children (< 5 years)

Characteristic	OR (95% CI)
Cooking and heating fuel type	
Low polluting*	-
Medium polluting	1.26 (1.00-1.58)
High polluting	1.29 (1.02-1.62)
Age of child (in months)	
0-5*	-
6-11	1.27 (0.92-1.76)
12-23	1.15 (0.84-1.56)
24-35	0.87 (0.63-1.20)
36-59	0.66 (0.49-0.89)
Number of children in household	
1*	-
>1	0.69 (0.56-0.83)
Number of children	4 679

* *Reference category*

6.4 Discussion and conclusions

ARI has been identified as a severe problem in South Africa, particularly for children under 5 years of age.[1] There is also a strong case for acknowledging the large public health risk arising from indoor air pollution exposure due to continued reliance on polluting fuels for cooking and heating in South Africa. Results of this study suggest that exposure to cooking and heating smoke from high polluting fuels is significantly associated with ARI prevalence in young children, independent of a child's age or the number of children in a household.

The adjusted risk estimates for the type of fuel use are consistent with other South African studies that investigated the risk of indoor fuel use and ARI in children

(<5 years).[26,27,33] This study therefore provides further evidence that cooking and heating homes with especially high polluting fuels can amplify the risk of ARI in young children.

Dudley et al investigated the impact of indirect indicators of air pollution on human health.[26] Dudley et al also focused on low vitamin A levels as a risk factor for respiratory infection.[26] The results revealed differences in risk between sever and mild cases of acute respiratory infection in respect of housing conditions (OR=4.2; 95% CI=1.3-14.5) and possession of clinic report card (OR=3.4; 95% CI=1.0-11.5). Mild cases were more likely to have had a previous ARI than the 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 the sever cases were more likely to have had a hospital admission the previous 6 months (OR=5.5; 95% CI=1.2-33.4), poorer housing conditions (OR=7.9; 95% CI=2.2-29.9) and not to have electricity (OR=4.9; 95% CI=1.6-16.2).

The study performed by Von Schirnding et al was based in the major urban and peri-urban areas of the country.[27] The main aim of the project was to examine the impact of environmental risk factors associated with housing on diarrhoeal disease and acute respiratory infections (ARI). The study results were reported on a national level. The presence of more than one adult smoking in a household significantly increased the likelihood of coughing and breathing problems (OR=2.0; 95% CI=1.3-3.3). Other potential risk factors of coughing and breathing problems included: not using electricity (OR=1.7; 95% CI=1.1-2.5); using gas, paraffin, coal or wood as cooking fuels (OR=1.7; 95% CI=1.3-2.5); using gas, paraffin, coal or wood as heating fuels (OR=2.0; 95% CI=1.7-3.3); not owning a refuse bin (OR=2.4; 95% CI=1.1-5.0); not having a chimney in the home (OR=1.8; 95% CI=1.3-2.5); child younger than 2 years (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).

Sanyal and Maduna determined the levels of indoor gaseous pollutants and their impact on the respiratory health of children.[33] The study established that high levels of

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

There are some important limitations in this study, which should be considered when interpreting the results. The SADHS had a cross-sectional design. Cross-sectional studies are weak to prove causation as they are subject to difficulties interpreting the temporal sequence of events since health status and determinants are measured simultaneously. However, the biological plausibility of exposure to smoke from medium and high polluting fuels has been addressed.[11-19]

Reliance on self-reported data does include a risk of misclassification of disease and exposure status resulting in statistical significance arising by chance. Information on ARI is based on mothers' reports and no clinical measurements were undertaken and smoke exposure was ascertained from type of fuel used for cooking and heating. Although the symptomatic definition used here is aimed to assess acute lower respiratory infections (ALRI) in children, some acute upper respiratory illness may have been integrated in the conveyed prevalence. As it is impossible to separate ALRI from these data, the term ARI is used in this study, not ALRI. In developing countries such as South Africa, where clinical data on ARI are frequently unattainable or very weak, the symptomatic definition of illness used here is assumed to present a reasonably accurate estimation of ARI in the population.

Notwithstanding the lack of measurement of air pollution exposure and ARI, the uniformity in the significance of crude and adjusted effects of polluting fuel use on childhood ARI implies a probable 'exposure–response' relationship as the adjusted odds of experiencing ARI increased from 1.26 (95% CI : 1.00-1.58) to 1.29 (95% CI : 1.02-1.62) (Table 2) when using medium polluting fuels and high pollution fuels compared to low polluting fuels, respectively.

Differential and/or nondifferential misclassification may have influenced the risk estimates. Many households in South Africa in general use a combination of cooking and heating fuels. The calculated effects may be underestimated if only considering

using high polluting fuels (wood and dung) exclusively and not in combination with paraffin, coal, LPG/natural gas and/or electricity. This is also expected using medium polluting fuels (paraffin, coal) exclusively and not in combination with LPG/natural gas and/or electricity. However, none of the households under investigation used wood or dung exclusively or paraffin or coal exclusively. No quantitative exposure assessment (including duration of exposure, as reflected by frequency and duration of fuel use for heating and cooking per day) was conducted during the SADHS. It is recommended that future SADHS should separate the type of fuels use for cooking and heating in two separate questions. Exposure to smoke from polluting fuels during heating is much longer than exposure during cooking.

Given the high prevalence of ARI and relatively small number of deaths in the sample (n=269, 5.28% of the sample, N=5 093), the impact of selection bias in the sample due to ARI-related mortality on the estimated effect is likely to be little. If such bias is prominent, the risk estimates of effect of cooking and heating fuel smoke on ARI will be downwardly biased as children living in households using medium and high polluting fuels are more likely to die from ARI.

It was found that children who have received treatment for ARI are more likely to have suffered from ARI than not (64% compared to 5%). So there is little bias due from underreporting of ARI due to lack of awareness that the child had an ARI event during the 2-week reference period. This is supported by Kauchali et al who conducted a local rural study on maternal perceptions of childhood ARI (<5 years).[34] They reported that maternal recognition of respiratory distress was good (sensitivity 91.3%, 95% CI : 86.8-95.8%; specificity 95%, 95% CI : 89.5–100%), with little variation between mothers (kappa = 0.704).

Other factors that might contribute to childhood ARI, such as outdoor and indoor air pollution sources (e.g. mother's smoking status, location of household close to industry, transportation sources or waste fill sites, insecticide or fertiliser use, allergens such as pollen, dust, fungal spores from mildew and moulds) along with meteorological variables (precipitation, temperature, humidity), mother's pre-pregnancy weight, child's

birthweight, mother's exposure to other pollution sources and risk factors during pregnancy as well as the current HIV/AIDS epidemic were not recorded. Excluding these risk factors from the analysis might introduce substantial bias (differential or nondifferential). Thus the direction of bias on the calculated association measures is not easy to predict. The definition of a confounder is important to remember: it must be associated with both the exposure variable of interest and the health effect. As the association between these unaccounted potential factors and polluting fuel use is not available from the literature, it is impossible to predict the direction of the potential bias on the association measure.

The most common indicators used for measurement of socioeconomic status are income level, occupation and educational level.[35] We cannot rule out that the results would have been different with another measure. However, Demographic and Health Surveys traditionally do not include questions on income and expenditure. Educational level measures one aspect of socio-economic status and we cannot rule out that the results would have been different with another measure. More than a third (35%) of the women never married, so we could not assume that the current partner's job or education remained the same during the 5 years preceding the survey. Mishra analysed the Zimbabwean DHS data and calculated a household standard of living index as a possible confounder in the association between household cooking fuel use and ARI in preschool age children (<5 years).[32] In this study a similar approach was followed.

The current HIV/AIDS epidemic along with escalating number of TB infections could also influence the association between ARI and exposure to smoke from polluting fuels. The TB prevalence rate was low amongst household members clinically diagnosed with the disease (2%) and did not indicate an association with ARI amongst preschool children (results not shown).

During the analysis it was assumed that confounding is additive and not multiplicative. If confounding is additive, then the confounding variable would produce the same additional risk of a health outcome in the exposed and unexposed; but if the health outcome is rare in unexposed, it would follow that the confounder might account for a

much larger proportion of health outcome in that group. Conversely, if two exposures act multiplicatively, the proportional increase in health outcome rates due to confounding would be the same in exposed and unexposed; but if the health outcome is more prevalent in the exposed group, the absolute increase would be larger in the exposed. This issue thus has important risk assessment and public health policy implications.

The generalisibility of data is determined by the non-response rate. The response rate was larger than 90% for both the household and women's health surveys. Thus the bias that might be introduced by non-response is relatively low for the SADHS data.

Wichmann and Voyi concluded that most of the local studies on this topic are fraught with systematic and random errors, with limitations similar to this study.[30] However, this study had the advantage of controlling for more possible confounders than previous local studies. In order to improve health for the unique South African population through epidemiological studies it is imperative that these studies should attempt to minimise systematic and random errors and subsequently strengthen their validity and accuracy. It is hoped that future analytic studies will validate and improve the understanding how smoke from paraffin, coal, wood and dung detrimentally impacts on children's respiratory health in South Africa. Such research is important because a large proportion of households in South Africa and other developing countries rely on biomass fuels for household energy and ARI are a leading cause of ill health and death in young children.

However, given the fact that only 5% of the research budget is spent on health related research in South Africa, compared to 30% in developed countries, it is important that analytical studies should not merely redocument the impact of known risk factors.[36] Instead, such studies should provide a basis for designing technical or socio-behavioural interventions to minimise exposure to air pollution from cooking and heating fuels, such as the study by Smith-Sivertsen et al.[37] They conducted the very first ever published randomised control intervention trail in a poor rural community in Guatemala. South African intervention studies should include a comprehensive exposure assessment with

indoor and personal measurements for SO_2, NO_2, O_3 and $PM_{2.5}$ and a detailed health assessment.

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Chapter 7

POTENTIAL INFLUENCE OF COOKING AND HEATING FUEL USE ON 1-59 MONTH OLD MORTALITY IN SOUTH AFRICA

Objectives: To determine the association between dirty cooking and heating fuel use and 1-59 month old mortality in South Africa whilst allowing for a number of confounders or effect modifiers.

Methods: Data from the 1998 South African Demographic and Health Survey (SADHS) were analysed. The SADHS was the first national health survey conducted across the entire country and provided the opportunity to examine the prevalence and determinants of various morbidity and mortality outcomes in a representative national population rather than a selected high risk population, as has been the case in most previous studies in developed countries.

Results: The results from 3 556 children (142 deaths) from 2 828 households suggested that exposure to cooking and heating smoke from dirty fuels is significantly associated with 1-59 month mortality, after controlling for mother's age at birth, water source, asset index and household crowdedness (OR = 1.99, CI: 1.04-3.68).

Conclusions: Although there is potential for residual confounding despite adjustment, the better documented evidence on outdoor air pollution and mortality suggest this association may be real. As nearly half of households in South Africa still rely on polluting fuels and women of childbearing age perform most cooking tasks, the attributable risk arising from this association, if confirmed, could be substantial. It is trusted that more detailed analytical intervention studies will scrutinise these results in order to develop integrated intervention programmes to reduce children's exposure to air pollution emanating from cooking and heating fuels.

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7.1 Introduction

The aim of epidemiology is to identify factors that cause disease, with the broader goal of identifying opportunities for prevention in order to reduce and eventually eliminate the burden of disease in human populations. Despite all the shortcomings of epidemiological studies, they are important in linking exposure to human health directly.[1-8] Determining the burden of disease due to preventable risk factors is important for identifying and prioritising environmental and public health interventions.

Notwithstanding technical advances that have improved survival of children (12-59 months) in developing countries, infant (< 12 months) mortality rates are still at least 10 times higher in developing countries than in developed countries.[9] Infant deaths mainly happen in the perinatal (< 7 days old) and neonatal periods (< 1 month old), mostly due to maternal characteristics, delivery factors, prematurity, intrauterine growth retardation and congenital causes, whilst later deaths are more likely to be the result of infection and environmental factors.[10-23] It is therefore important to identify risk factors that predict early infant and child mortality. This article will concentrate on indoor air pollution exposure due to the combustion of animal dung, wood, paraffin and coal (henceforth dirty fuels) as the source of energy for cooking and heating as a potential risk factor for 1-59 month old mortality. Over 40% of the global burden of disease attributed to environmental factors falls on children below five years of age, who account for only about 10% of the world's population.[24] Furthermore, air pollution is the largest single environment-related cause of ill health among children in most countries.[25] In other countries it is the second, after the scarcity of safe water. Globally, 2.6% of all ill-health is attributable to indoor smoke from dirty fuels, nearly all in poor regions.[26]

A recent report by the World Health Organisation accounted that there is currently substantial evidence concerning the adverse effects of air pollution - especially for outdoor respirable particulate matter (PM_{10}, particles with aerodynamic diameters below 10 μm) - on different pregnancy outcomes and infant health.[27] The evidence is sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the postneonatal period (28 days to 1 year of life)(WHO Monograph, 2004).

Thus this concrete evidence can be used to strengthen outdoor air pollution mitigation in South Africa. However, as about 47% of 11 million households in the country still rely on dirty fuels as the source of energy for cooking and heating and with 70% using electricity for lighting[28], it is obvious that indoor air pollution may be magnitudes higher than outdoor air pollution exposure. Although outdoor sources often dominate air pollution emissions, indoor sources frequently dominate air pollution exposures. Exposure is a function of both the pollutant concentration in an environment, and the person-time spent in the environment. Since most people spend the majority of their time in homes, schools and workplaces, human exposure to air pollution is largely a function of pollutant levels in indoor settings (which can arise from outdoor sources, and vice-versa).

As the exposure-response relationships and risk estimates for under five mortality have been derived for outdoor air pollution in developed country urban situations, it raises a number of issues about their suitability for application indoor air pollution due to dirty fuel combustion in developing countries.[29] Over the past 20 years, the hazards of indoor air pollution have been documented by a growing body of literature[30], but very few studies focused on its impact on infant and child mortality. South Africa, a middle income country, is faced by health risk factors from a First World situation (e.g. industry, traffic, aging population) along those from a Third World situation (e.g. domestic burning of dirty fuels, poor sanitation, overcrowding). The three global factors that directly or indirectly impact on health - the community and social environment, the physical environment and the family and individual environment – are different for developed and developing countries. In the South African context economical, social and cultural factors may render the population more vulnerable to increased air pollution exposure, due to factors such as poor hygiene, overcrowding, dusty environments, poor nutrition, open dwellings, outdoor lifestyles and the escalating HIV/AIDS epidemic. Thus there is a need to derive local risk estimates from local data in order to calculate the burden of disease due to indoor air pollution exposure.

Dirty fuels are at the bottom of the energy ladder regarding combustion efficiency and cleanliness.[31] Smoke from dirty fuel combustion produces numerous air pollutants that

are detrimental to health, including respirable particulate matter (RSP, PM_{10}), carbon monoxide (CO), nitrogen oxides (NO_x), formaldehyde, benzene, 1,3 butadiene, polycyclic aromatic hydrocarbons (PAHs)(such as the carcinogen benzo[a]pyrene, B[a]P), and many other toxic volatile organic compounds (VOCs). The fuels are typically burned in simple, inefficient and mostly unvented household stoves, combined with poor ventilation, generate large volumes of smoke indoors. Even when the stoves are vented to the outside, combustion of unprocessed solid fuels produces enough pollution to significantly affect local neighbourhood pollution levels, with implications for total exposures.

The poorest and most vulnerable populations in developing countries are most exposed to indoor air pollution from fossil and biomass fuel combustion for cooking and heating. A local study by Thomas et al reported that on average 14% of households had children (<6 years) usually or always present when their mothers were cooking.[32] This percentage increased to 18.3% of households in the lowest wealth quintile. Paraffin stoves were burning in the lower wealth quintile homes on average 4 hours per day, with the evening meal taking over half of this time to prepare. Muller et al established in another local study that people spend on average 2 hours cooking indoors during both winter and summer.[33]

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, including paraffin, was used.[34] The mean maximum hourly average was 28 $\mu g.m^{-3}$ (range 0–451 $\mu g.m^{-3}$) for NO_2, 1 414 $\mu g.m^{-3}$ (range 0–17 723 $\mu g.m^{-3}$) for SO_2 and 34 $mg.m^{-3}$ (range 0-388 $mg.m^{-3}$) for CO. The number of households where standards were exceeded by the maximum hourly averages by NO_2 and CO was six (9%) and twenty (30%) respectively (Hourly WHO standard of 200 $\mu g.m^{-3}$ and 30 $mg.m^{-3}$, respectively) and for (sulphur dioxide) SO_2 the number was 28 (42%) (Hourly Californian standard of 655 $\mu g.m^{-3}$)(no hourly WHO or USEPA standard for SO_2 exists).[35,36] Total suspended particulates (TSP) concentrations ranged from 7-433 $\mu g.m^{-3}$. Comparisons of TSP concentrations with international standards have not been made, as these are now focused on PM_{10} and $PM_{2.5}$ particles. No hourly standards or guidelines also exist for

PM_{10} and $PM_{2.5}$. Röllin et al provided scientific evidence that even in partially electrified homes in South Africa levels of RSP were significantly lower (mean 77 $\mu g.m^{-3}$, median 37.5 $\mu g.m^{-3}$) relative to their non-electrified counterparts (mean 162 $\mu g.m^{-3}$, median 107 $\mu g.m^{-3}$)($p=0.012$) during summer.[37] Stationary (kitchen CO) levels in un-electrified and electrified dwellings ranged from 0.36-20.95 ppm and 0-11.8 ppm, respectively. The mean level of log (CO) in the kitchen was significantly higher in the un-electrified areas (1.25 vs. 0.69) ($p=0.0004$). The mean level of log (CO) for personal measurements conducted on children (<18 months) was higher in the un-electrified areas (0.83 vs. 0.34) ($p<0.0001$).

The mechanism by which dirty fuel smoke can increase the risk of acute and chronic respiratory diseases, which may lead to premature death is not fully understood. Exposure has been associated with compromised pulmonary immune defence mechanisms in both animals and humans.[38-41] The special vulnerability and susceptibility of children in respect to air pollution exposure are related to several differences between children and adults: the ongoing process of lung growth and development, incomplete metabolic systems, immature host defences and high rates of infection by respiratory pathogens. Furthermore, activity patterns specific to children can lead to higher exposure to air pollution and higher doses of pollutants reaching the lungs. The efficiency of detoxification systems exhibits a time dependent pattern during pre- and post-natal lung development that in part accounts for increased susceptibility of young children to pollutants at critical time points. Tobacco smoke also has been shown to cause depressed immune system responses.[42-46] Of the specific pollutants in dirty fuel smoke, exposure to PM_{10} has been shown to induce a systemic inflammatory response that includes stimulation of the bone marrow, which can contribute to the pathogenesis of the cardiorespiratory morbidity.[38-41] Other evidence indicates that exposure to PAH (especially B[a]P, which is found in large quantities in dirty fuel smoke) can cause immune suppression and can increase the risk of infection and disease.[47-49] Moreover, acute and long-term exposures to NO_x, commonly found in dirty fuel smoke, can increase bronchial reactivity and susceptibility to bacterial and viral infections.[50,51] It is, therefore, possible that extended exposure to high levels of dirty fuel smoke can impair the pulmonary defence mechanisms, compromise the lung function, and render people

more susceptible to acute respiratory infections (ARI) and developing asthma or increase the frequency and severity of attacks in asthmatic people.

The current study will add to the current body of knowledge regarding cooking and heating fuel use and 1-59 month old mortality, whilst considering a number of confounders or effect modifiers. The 1998 South African Demographic and Health Survey (SADHS) is the first national health survey conducted across the entire country.[52] Data from this survey provided the opportunity to examine the prevalence and determinants of various morbidity and mortality outcomes in a representative national population rather than a selected high risk population, as has been the case in most previous studies in developed countries. It is trusted that the results of this study will draw attention to risk factors and lead to debate on potential integrated intervention programs.

7.2 Methods

7.2.1 Survey method

The 1998 SADHS had a cross-sectional design and was a national household survey of the population living in private households in the country. Detailed information on the survey design is outline elsewhere.[52] The sampling frame for the SADHS was the list of approximately 86 000 enumeration areas (EAs) created by Central Statistics (now Statistics South Africa, SSA) for the Census conducted in October 1996. The EAs, ranged from about 100 to 250 households and were stratified by 9 provinces, urban and non-urban residence and by EA type. The number of households in the EA served as a measure of size of the EA.

The first stage (proportional stratified sampling) of the two-stage sampling led to a total of 972 EAs being selected for the SADHS (690 in urban areas and 282 in non-urban areas). The second stage involved a systematic random sample of 10 and 20 houses in selected urban and rural EAs, respectively. Oversampling was conducted in some areas to enable inference to be made about differences across provinces and race – and in the Eastern Cape province, across health districts.

In addition to the main survey of households a women's health questionnaire was administered individually to women (15-49 years) in all the households selected for the main survey. The SADHS questionnaires were translated into 9 of the 11 official languages of South Africa and checked by backtranslation.[52] The questionnaires were pretested in November/December 1996 as part of a pilot study.

The household questionnaire characterised all household members, including their age, sex, race and education, household characteristics such as fuels use for cooking and heating (Refer to Appendix 1). The women's health questionnaire was designed principally to produce reliable estimates of demographic rates (particularly fertility and childhood mortality rates), of maternal and child health indicators and of contraceptive knowledge and use for the country as a whole, the urban and the non-urban areas separately and for the nine provinces (Refer to Appendix 3).

Interviewers were trained over several weeks. Interviews were conducted after working hours. Interviewers were instructed to return twice if a suitable respondent was not found at home. Fieldwork commenced late January 1998 and was completed in September 1998. The response rate at the household level was 97% of 12 860 households in 966 EAs. For the women's health survey, the overall response rate was 92.3%.

The analysis presented in this paper is based on secondary analysis of existing survey data with all identifying information removed.

7.2.2 Definitions

For each child under age 5, the mother was asked if the child was still alive and if not, at what age the child passed away (Refer to Appendix 3).[52]

Variables related to the household included exposure to cooking and space heating smoke, access to flush toilet facilities (coded 0 if own/shared; coded 1 if bucket/pit larine or no facility/bush/field), access to clean water (coded 0 if using piped water in dwelling/site/yard, tap water/water carrier/tanker, borehole/well or bottled water;

coded 1 if water from dam/river/stream/spring, rain water tank), number of rooms per people living in household (code 0 if ≤ 2; coded 1 if >2), nutritional status (coded 0 if household going hungry never/seldom; coded 1 if often/seldom) and asset index as an indicator of socio-economic status.

Exposure to cooking and space heating smoke was ascertained indirectly by type of fuels used. This was the main independent variable. The question was, 'What does your household use for cooking and heating?' Respondents indicated all the different fuel types that were used. The households were grouped into two categories representing the extent of exposure to cooking and heating smoke: dirty fuels (if either wood, dung, coal or paraffin was used in the fuel combination without using LPG/natural gas or electricity) and clean fuels (if LPG/natural gas or electricity was used exclusively in the fuel combination). The small residual category of other fuels (n=6, 0.12%, N=5001, 33 missing values) was excluded from the analysis due to unknown nature of fuels in that category.

A modified version of the method used by Mishra to calculate a household standard of living index (SLI) was applied to calculate an asset index.[53] This modified approach still used the scores as applied by Mishra, however the variables related to water supply and type of toilet facility were omitted from the modified approach.[53] This was done to assess the potential influence of water supply and type of toilet facility on 1-59 month mortality otherwise these variables would have been embedded in a SLI. The asset is calculated by adding the following scores: 3 for a car or tractor; 2 each for a scooter/motorcycle, TV, telephone, refrigerator, electricity, wood/vinyl/asphalt/ceramic/cement/carpet of main floor material; 1 each for a bicycle, radio. In this modified approach the asset index was coded as 0 if ≥ 35 and coded as 1 if <35.

Variables pertaining to a child included age (coded 0 if <12 months; coded 1 if ≥ 12 months), sex (coded 0 for a boy; coded 1 for a girl), birth order (coded 0 if 1; coded 1 if > 1), birth interval (coded 0 if <24 months; coded 1 if ≥ 24 months) and whether the child was breastfed (coded 0 if yes; coded 1 if no).

Variables related to the biological mother of a child included age at birth (coded 0, 1, 2 and 3 if <19, 19-24, 25-34 and ≥ 35 respectively). Education was not considered as the educational status at the birth of the child was not known and was not constant over the observation period.

The location of the household was categorised on an urban/rural level and provincial level. The child's year of birth is included to capture a time trend in child mortality.

7.2.3 Data analysis

All subsequent statistical analyses of results were done using STATA version 8. The household (N=12 209) and women's questionnaire (N=5 066) data were linked with a unique identification variable, containing the cluster number and household number. The merge data set had 5 060 observations. This analysis included only children whose mothers had indicated their ethnic identity as Africa/Black. This ethnic group comprised 81.9% (n=4 114, N=5 060 with 16 missing values) of the data with 237 deaths compared to 25 from the other 3 groups. 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 still very much linked to past access to resources, socio-economic status and educational status.

All children from multiple births (n=113, 2.75%, N=4 114 with 237 missing values) were excluded from the analysis as well as all children who did not live with their mothers (n=331, 8.54%, N=4 114 with no missing values), women who only visited the household during the survey (n=77, 1.87%, N=4 114 with 1 missing value) and deaths before 1 month since the cause of death for newborns is difficult to determine and may be due to quite different risk factors (n=27, 0.66%, N=4 114 with no missing values). Eventually 3 556 children, of whom 142 were deaths, were included in the analysis.

Simple descriptive statistics were used to describe the characteristics of the sample (TAB command) and in calculating the mortality incidence rate for each characteristic (STSUM command).

The 1998 SADHS report pointed out that the risk factors might be correlated with each other.[52] Correlations among risk factors were investigated with χ^2 analysis (PWCORR command). It was observed that most of the risk factors were significantly correlated at the 95% confidence level, although very poorly with correlations coefficients varying from 0.01 to 0.40.

To estimate the effects of the independent variables on 1-59 month old mortality, Cox proportional hazards analysis was performed (STCOX command), stratified by urban or rural. The time variable was either set as the child's current age (months since birth until interview date) or age of death (in months), respectively for those who were still alive and those who were deceased at the interview date. Time was fitted to estimate the hazard rate ratio, hereafter denoted relative risk, of mortality. The results were presented as the relative risk of mortality with 95% confidence intervals (CI).

The proportional hazards assumption was confirmed for each independent variable (STPHTEST command). First univariate analyses were run and subsequently a multivariate analysis with all the significant independent variables identified from the former procedure.

7.3 Results

The data presented here represent a more detailed analysis of the first national survey of under-five mortality in South Africa. Table 1 lists the characteristics of the 3 556 children from 2 828 households. The overall mortality incidence rate in the 5 years within the study was 1.473/1000.

Table 1 Number of observed person-months, incidence rates and deaths for risk factors under investigation

Risk factor	No.	%	No. of deaths	%	No. of person-months at risk	Incidence rates per 1000 person months
Age (months)						
1-11	2 740	77	120	85	91 710	1.309
12-59	816	23	22	15	4 666	4.715
Sex						
Boy	1 804	51	78	55	48 159	1.620
Girl	1 752	49	64	45	48 217	1.327
Birth order						
1	1 144	32	48	34	30 060	1.597
>1	2 412	68	94	66	66 316	1.418
Mother's age at birth (years)						
<19	375	11	26	18	10 331	2.517
19-24	1 116	31	44	31	29 782	1.477
25-34	1 443	41	42	30	39 360	1.067
≥35	622	17	30	21	16 903	1.775
Preceding birth interval (months)						
<24	3 222	91	123	87	86 437	1.423
≥24	334	9	19	13	9 939	1.912
Breastfed						
Yes	3 151	89	387	273	84 207	1.473
No	405	11	18	13	12 169	1.479
Area of residence						
Urban	1 262	35	36	25	34 963	1.030
Rural	2 294	65	106	75	61 413	1.726
Fuel use						
Clean	714	20	13	9	19 721	0.659
Dirty	2 819	79	129	91	76 112	1.695
Toilet						
No Flush	2 683	75	120	85	71 800	1.671
Flush	850	24	21	15	23 924	0.878
Water source						
Clean	2 487	70	74	52	27 670	2.458
Dirty	1 045	29	68	48	67 995	1.088
Asset index						
<35	1 956	55	97	68	52 411	1.851
≥35	1 600	45	45	32	43 965	1.024
Persons per room						
≤ 2	1 041	29	52	37	28 138	1.848
>2	2 515	71	90	63	68 238	1.319
Household going hungry						
Never/seldom	1 277	36	44	31	35 098	1.254
Often/sometimes	2 220	62	98	69	59 734	1.641

Table 1 (continues)
Number of observed person-months, incidence rates and deaths for risk factors under investigation

Risk factor	No.	%	No. of deaths	%	No. of person-months at risk	Incidence rates per 1000 person months
Year of birth						
1993	456	13	26	18	24 031	1.082
1994	662	19	30	21	28 718	1.045
1995	663	19	30	21	21 033	1.426
1996	736	21	25	18	14 994	1.667
1997	851	24	31	22	7 180	4.318
1998	188	5	0	0	420	0.000
Province				0		
Western Cape	57	2	2	1	1 439	1.390
Eastern Cape	1 070	30	55	39	2 8432	1.934
Northern Cape	107	3	4	3	3 030	1.320
Free State	236	7	9	6	6 378	1.411
KwaZulu-Natal	520	15	25	18	13 917	1.796
North West	302	8	7	5	8 512	0.822
Gauteng	271	8	8	6	7 531	1.062
Mpumalanga	442	12	15	11	11 867	1.264
Limpopo	551	15	17	12	15 270	1.113
Total	3 556	100	142	100	96 376	1.473

Children aged 12-59 months were somewhat less represented compared to the <12 months age group. Children were relatively equally distributed by sex. The minority of the children were first borns (32%). Most of the children were born to mothers aged 19-24 and 25-34 years and spaced within 24 months of their siblings. The majority of the children were breastfed (89%) at some stage.

Most of the children lived in rural areas (65%). The majority of the children lived in households that use dirty fuels for cooking and heating either exclusively or in combination with clean fuels (79%), with no access to a flush toilet (75%), with access to clean water (70%) and low asset index (55%). Most of the children lived in crowded households (>2 people/room)(71%) with poor nutritional status (hungry often/sometimes)(62%).

There were fewer births during 1998 compared to 1993-1997. By province of residence, the largest proportion was from the Eastern Cape (30%), nearly equal proportions from KwaZulu-Natal, Mpumalanga and Limpopo (12-15%) and smallest from Western Cape (2%).

In the univariate analysis only mother's age at birth, fuel use, access to clean water, asset index and people/room were significantly associated with an increase risk in 1-59 month old mortality (Table 2). In the adjusted model, all of them, except asset index, are still significantly associated with an increase risk in mortality (Table 3).

The impact of mother's age at birth on under-five mortality is not affected much in the adjusted model. Children born to mothers aged 19-24 and 25-34 were significantly less at risk from dying compared to those born to mothers younger than 19 years, whilst older mothers (≥ 35 years) had no significant health benefit. Compared to households using clean fuels, children in households using dirty fuels had a substantially higher mortality rate: from 2.22 (CI: 1.22-4.04) to 1.95 (CI: 1.32-2.90) in the univariate and adjusted analyses. Indeed, the negative effect of dirty fuels in the univariate model exceeded that of lack of clean water supplies. However, their effects were similar in the adjusted model. Children living in household without clean water supplies were nearly twice more at risk from dying compared to their counterparts (adjusted model: 1.96, CI: 1.32-2.90). The risk posed by lack of access to clean water decreased somewhat (7%) from the univariate to adjusted analyses. Surprisingly, children living in crowded households (> 2 persons/room) had a significantly lower risk of dying (37%) compared to their counterparts. The influence of a crowded household decreased somewhat (11%) from the univariate to adjusted analyses.

7.4 Discussion and conclusions

Results of this study suggest that exposure to cooking and heating smoke from dirty fuels is significantly associated with 1-59 month mortality, after controlling for mother's age at birth, water source, asset index and household crowdedness (OR = 1.99, CI: 1.04-3.68). It is intriguing that a household crowdedness decreases the risk of

Table 2 Relative risk of mortality with 95% confidence intervals obtained from univariate Cox regression analyses, stratified by area of residence

Risk factor	Relative risk	95% CI
Age (months)		
1-11★	1.00	-
12-59	1.54	0.95, 2.49
Sex		
Boy★	1.00	-
Girl	0.84	0.61, 1.17
Birth order		
1★	1.00	-
>1	0.87	0.62, 1.24
Mother's age at birth (years)		
<19★	1.00	-
19-24	0.60	0.37, 0.97†
25-34	0.44	0.27, 0.71†
≥35	0.71	0.42, 1.20
Preceding birth interval (months)		
<24★	1.00	-
≥24	1.35	0.83, 2.19
Breastfed		
Yes★	1.00	
No	1.11	0.67, 1.81
Fuel use		
Clean★	1.00	-
Dirty	2.22	1.22, 4.04†
Toilet		
Flush★	1.00	-
No flush	1.52	0.82, 2.83
Water source		
Clean★	1.00	-
Dirty	2.11	1.44, 3.10†
Asset index		
≥35★	1.00	-
<35	1.59	1.08, 2.32†
Persons per room		
≤ 2★	1.00	-
>2	0.71	0.50, 0.99†
Household going hungry		
Never/seldom[a]	1.00	-
Often/sometimes	1.21	0.84, 1.73
Province		
Western Cape★	1.00	-
Eastern Cape	0.92	0.21, 3.91
Northern Cape	1.01	0.18, 5.51
Free State	0.90	0.19, 4.21
KwaZulu-Natal	0.96	0.22, 4.15
North West	0.42	0.09, 2.09
Gauteng	0.88	0.19, 4.15
Mpumalanga	0.62	0.14, 2.81
Limpopo	0.50	0.11, 2.28
Year of birth	1.00	0.88, 1.13

★Referent category, †$p<0.05$ for stratum RR

Table 3 Relative risk of mortality with 95% confidence intervals obtained from multivariate Cox regression analyses, stratified by area of residence

Risk factor	Relative risk	95% CI
Mother's age at birth (years)		
<19★	1.00	-
19-24	0.58	0.36, 0.95†
25-34	0.43	0.26, 0.70†
≥35	0.67	0.40, 1.13
Fuel use		
Clean★	1.00	-
Dirty	1.99	1.04, 3.68†
Water source		
Clean★	1.00	-
Dirty	1.96	1.32, 2.90†
Asset index		
≥35★	1.00	-
<35	1.25	0.82, 1.88
Persons per room		
≤2★	1.00	-
>2	0.63	0.44, 0.89†

★Referent category
†$p<0.05$ for stratum RR

mortality in the multivariate model. A possible explanation might be that fewer fuels are burnt for space heating when more people are sharing a dwelling.

The potential risk estimates from the multivariate model were in general consistent with other developing country studies that focused on toilet facilities and clean water as risk factors for infant and childhood mortality. Macassa et al observed an 80% higher risk for 12-59 month mortality when households in urban areas of Mozambique had no flush toilet in a multivariate model.[54] They did not report any significant health benefits when households had clean water sources or for mothers to have babies when they are older than 19 years, as opposed to this study. Woldemicael reported that children born in urban areas of Eritrea had 59% and 44% decreased postneonatal and childhood mortality compared to children born to households without a flush toilet and piped water, respectively.[55] Gubhaju et al reported that the risk of death was 44% lower among Nepalese children born to households, which used piped water compared to their counterparts, even after controlling for socio-economic and demographic factors.[56] The study also indicated that the risk of death was 64% lower among infants born to households which had their own toilets compared to their counterparts.

Although most available evidence relating air pollution and mortality was obtained for adults using outdoor exposure data from developed countries, pollution has been also associated with increased mortality in children, but in a significantly smaller number of studies. Furthermore studies conducted in developing countries rather focused on outdoor instead of indoor air pollution. Conceicao et al reported estimated proportions of childhood respiratory deaths attributed to outdoor CO, SO_2 and PM_{10} exposure, when considered individually, are around 15, 13 and 7%, respectively in the city of São Paulo, Brazil.[57] Loomis et al found that excess infant mortality was associated with PM_{10} levels during the days before death, with the strongest association observed for the average concentration of fine particles during the 3-5 preceding days in Mexico City.[58] A 10 $\mu g.m^{-3}$ increase in the mean level of PM_{10} during those 3-5 days was associated with a 6.9% excess of infant deaths. Infant mortality was also associated with the levels of NO_2 and O_3 3-5 days before death, but not as consistently as with PM_{10}. Penna et al observed that, with the income variable included in the regression, a statistically significant association between the average annual level of particulates and infant mortality from pneumonia from an investigation in the Rio de Janeiro Metropolitan Area.[59]

A recent report by Statistics South Africa, listed the ten leading underlying natural causes of death for different age groups during 1997, 1999 and 2001.[60] Table 4 presents the variation across the three years of leading underlying natural causes of death for infants (< 12 months) and children (12-48 months).

Infant deaths were mostly due to causes related to the perinatal stage, as also reported in the literature.[10-23] This particular analysis excluded the perinatal age group. However, between 22.6% and 18.7% of infant deaths were due to unexplained causes. It is likely that some of these premature deaths may be attributed to high indoor air pollution exposure. Multiplying the number of deaths due to other causes with the attributable fraction (0.44, CI: 0.03-0.68) it is estimated that 2 684 (CI: 187-4 154), 2 697 (CI: 188-4 174) and 2521 (CI: 176-3 902) infant deaths could have been prevented if dirty fuel use were completely eliminated during 1997, 1999 and 2001, respectively. The top 3 leading causes of child mortality (12-48 months) across the three years were intestinal

Table 4 Ten leading underlying natural causes of death for each age group: 1997, 1999 and 2001[60]

Broad group of causes of death	1997			1999			2001		
	Rank	No.	%	Rank	No.	%	Rank	No.	%
Less than 1 year									
All causes		27 044	100.0		30 359	100.0		30 750	100.0
Respiratory and cardiovascular - perinatal (P20 - P29)★	1	6 797	25.1	1	7 983	26.3	1	9 388	30.5
Digestive disorders foetus and newborn (P75 - P78)	2	4 814	17.8	2	6 323	20.8	2	5 498	17.9
Other disorders - perinatal (P90 - P96)	3	3 368	12.5	3	3 308	10.9	3	3 419	11.1
Disorders related to gestation and foetal growth (P05 - P08)	4	1 598	5.9	4	1 563	5.1	5	1 418	4.6
Infections specific to the perinatal period (P35 - P39)	5	1 310	4.8	5	1 446	4.8	4	1 626	5.3
Foetus and newborn complications (P00 - P04)	6	1 216	4.5	6	1 125	3.7	7	690	2.2
Haemorrhagic and haematological. disorders of the foetus & newborn (P50 - P61)	7	610	2.3	8	645	2.1	8	682	2.2
Malnutrition (E40 - E46)	8	424	1.6	9	495	1.6	10	514	1.7
Transitory endocrine and metabolic disorders specific to foetus and newborn (P70 - P74)	9	404	1.5	…	…	…	9	563	1.8
Certain immune disorders (D80 - D89)	10	387	1.4	7	842	2.8	6	1 207	3.9
Human immunodeficiency virus [HIV] diseases (B20 - B24)	…†	…	…	10	484	1.6	…	…	…
Other causes		6 116	22.6		6 145	20.2		5 745	18.7
1-4 years									
All causes		7 735	100.0		9 780	100.0		11 170	100.0
Intestinal infectious diseases (A00 - A09)	1	1 611	20.8	1	2 274	23.3	1	2 308	20.7
Malnutrition (E40 - E46)	2	733	9.5	3	886	9.1	3	910	8.1
Influenza and pneumonia (J10 - J18)	3	626	8.1	2	931	9.5	2	1 453	13.0
Tuberculosis (A15 - A19)	4	288	3.7	4	373	3.8	4	640	5.7
Human immunodeficiency virus [HIV] diseases (B20 - B24)	5	219	2.8	6	338	3.5	6	334	3.0
Other forms of heart disease (I30 - I52)	6	164	2.1	7	141	1.4	9	153	1.4
Certain immune disorders (D80 - D89)	7	111	1.4	5	349	3.6	5	454	4.1
Inflammatory diseases of the central nervous system (G00 - G09)	8	110	1.4	9	125	1.3	8	175	1.6
Other bacterial diseases (A30 - A49)	9	102	1.3	…	…	…	…	…	…
Metabolic disorders (E70 - E88)	10	92	1.2	10	112	1.1	10	131	1.2
Other viral diseases (B25 - B34)	…	…	…	8	137	1.4	7	226	2.0
Other causes		3 679	47.6		4 114	42.1		4 386	39.3

★The causes of death were coded using procedures described in the Stats SA manual Guidelines for coders using ICD-10. ICD-10 is the tenth revision of the International Classification of Diseases developed by the World Health Organisation (WHO), which is followed worldwide in order to have a uniform way of classifying morbidity as well as causes of death.

†Category not in top ten

infectious diseases, malnutrition and influenza and pneumonia. Furthermore, there was a steady increase in the number and percentage of deaths due to intestinal infectious diseases and influenza and pneumonia. Certain disorders involving the immune mechanism appeared in all three years, as did HIV-related diseases. It is anticipated that indoor air pollution may exert its impact in unison with all the causes of death listed, except perhaps those from malnutrition and metabolic disorders. Thus from the 6 910, 8 782 and 10 129 deaths in 1997, 1999 and 2001 3 033 (CI: 212-4 693), 3 854 (CI: 269-5 965) and 4 445 (CI: 310-6 880) may be attributed to high indoor air pollution exposure.

While this study achieved its objective to determine the association between dirty cooking and heating fuel use and 1-59 month old mortality whilst allowing for a number of confounders or effect modifiers, there are numerous limitations. Since it was a cross-sectional design, it was hard to inspect any possible temporal relationships. The data signifies a specific point in time and determining a cause and effect relationship, therefore, can be limited. Any association reported in this study therefore does not necessarily indicate causation. The proportional hazards assumption was confirmed for each independent variable. However, this test cannot indicate slight changes in exposure variables over the 5 year period. A large proportion (43%) of women still lived in their current house when the child was born, so we assumed that the toilet facility and water source variables remained constant over time. Mother's education was not included in the analyses as it is assumed not to be constant over the 5 year period.

Some biases within the study may influence the generalisability of the results. There is probable recall bias among respondents answering questions involving events occurring in the past, such as household going hungry and breastfeeding patterns immediately after birth. Reporting bias may be present, since the possibility exists for respondents to answer in such a fashion that may be more socially desirable or in ways that they perceive would get a more approving response from the interviewers. There is also the potential for information bias. Other factors that might be detrimental to children's health, such as mother's size and pre-pregnancy weight, child's birthweight, mother's exposure to other pollution sources and risk factors during pregnancy, outdoor air

pollution, insecticide and fertiliser use, meteorological variables along with current HIV/AIDS and TB epidemics, were not recorded. Furthermore, information was not collected on mother's smoking status. Excluding environmental tobacco smoke from the analysis might introduce substantial bias as 24% of women in South Africa smoke.[52] The current HIV/AIDS epidemic along with escalating number of TB infections could also influence the association between 1-59 month mortality and exposure to smoke from dirty fuels. As the SADHS survey did not collect indicators of potential HIV/AIDS status of the child, biological mother or any household member, the attributable fraction due to HIV/AIDS could not be calculated. However, the TB prevalence rate was low (2.4% among 13 826 adults).[52] Adult household members diagnosed with TB and who lived in households with smokers (34.2% among 13 826 adults), were not linked with the dataset as this reduced the number of deaths considerably.[52] Furthermore, although only 28% of children were vaccinated, no deaths were reported for unvaccinated children. Most women went for antenatal care check ups (97%).

Substantial evidence had shown that parents perceive the inherent healthiness of a child through knowledge of their own genetic endowments and health characteristics of the household and neighborhood.[61-63] If parents perceive that their child's inherent healthiness is low, they may alter their fertility decisions, leading to selection bias in the sample of potential births. However, we could not only consider first births in the analysis as this reduced the sample size considerably. Also, the number of children per age group decreased with increasing age and this is likely to be due to two contributing factors. First, close to 7% of the children in South Africa die by the time they reach 5 years of age and this high mortality rate may result in fewer older children in the sample.[64] Also, as children get older they are more likely to be sent away to live with relatives in urban areas. It was found that 331 black children did not live with their mothers (8.54%, N=4 114 with no missing values).

The most common indicators used for measurement of socioeconomic status are income level, occupation, and educational level.[65] We cannot rule out that the results would have been different with another measure instead of using an asset index. More than a

third (35%) of the women never married, so we could not assume that the current partner's job or education remained the same over the 5 year period.

It is hoped that future analytic studies will scrutinise the potential risk factors of 1-59 month mortality that were identified in this investigation. This is important given the fact that large proportions of households in this country and other developing countries are still relying on these fuels for household energy. However, Yach et al addressed the methodological difficulties in undertaking epidemiological studies in developing countries.[66] They pointed out the use of ecological and cross-sectional studies in determining the relationship between risk factors and disease and consequently applying detailed analytical studies to determine the reasons for these relationships. In South Africa, detailed analytic epidemiology studies will have to compete with the demands on the public and research purse for work on common diseases of pressing current importance (e.g. HIV/AIDS). Therefore analytical studies should not merely redocument the impact of known risk factors, but should provide a basis for designing interventions, albeit technical or socio-behavioural.

In the last decade a program of providing electricity to three million homes has been underway in South Africa. However, the county's heavy reliance on coal for electricity generation confers substantial external costs, which need to be taken into account.[67] Nevertheless, it is much easier to control pollutant emissions from a few power plants than from millions of households where exposure is extremely high due to the proximity of the source. Furthermore, changing the type of fuel used is not that easily assimilated by the South African community. A local ongoing, community-based electrification programme has found that about 50% of households continue to use wood for cooking and heating after electrification. Economic, political, educational and cultural factors account for why so many households continue to use wood.[68]

Therefore, the efforts need to focus on providing improved cook stoves designed to reduce exposure to smoke by means of improved combustion and improved venting, and designing public information campaigns to inform people about the health risks of exposure to indoor smoke. For such programmes to be effective, local needs and

community participation should receive high priority. It is important to note that 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.[28] Thus more than 8 million (20%) South Africans may not be able to benefit from health promotion material that is designed for the more educated population.

Socio-behavioural interventions may include preventing children and pregnant women from being exposed to smoke from the dirty fuels. The national department of Health is implementing secondary and tertiary prevention strategies, such as the Integrated Management of Childhood Illness (IMCI) to reduce infant and child mortality.[69] All three components of IMCI (case management, improvement of the health system, and household/community care components) are being implemented in South Africa. However, the expansion of IMCI is slow due to lack of resources (financial, human and material in the form of transport) and the lack of a dedicated budget for child health. Chapter 6 suggested that South African children (< 60 months) living in households using dirty fuels for cooking and heating were 25-29% more likely to have suffered from ARI as children from households using clean fuels, after adjusting for child's age, sex, birth order, number of children per household, mother's age at childbirth, mother's education, household living standard, province of residence, race and treatment received. These results are consistent with international studies that have reported strong associations between exposure to dirty fuel smoke and ARI in preschool children.[70-73] Two other local studies established a significant link with indoor air pollution indicators and respiratory health of preschool children.[74,75] Thus as acute respiratory infections (ARI)(such as pneumonia) are currently the second biggest cause of death amongst young children in large parts of South Africa it is important that mothers recognise the signs of ARI in order to seek timely medical care to prevent premature death of their young children.[60] Kauchali et al have indeed found in a local study that mothers do recognise the signs of ARI and that traditional remedies were the preferred treatment.[76]

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Chapter 8

GENERAL DISCUSSION

8.1 Main Findings

First the status quo of air pollution epidemiology in the country was elucidated in a narrative review (Chapter 2). The objectives of this review were (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 literature search strategy and selection criteria involved a MEDLINE search until June 2005. Fourteen out of 267 journal articles were found which focused on air pollution epidemiology (excluding active smoking and on internal dose as a proxy for health outcomes).[1-18] Two studies were also located by word of mouth or through the references of the selected studies.[19,20] The local studies do provide some evidence of associations with a range of serious and common health problems. Three of the studies established a strong significant relationship with air pollution indicators and deteriorated human health.[2,3,13-16] Seven studies established a significant link with air pollution indicators and disease.[1,4,8-10,17,19] Four studies did not report any significant link between air pollution indicators and disease.[5,11,12,20] The lower limit of many of the association measures of some of the reviewed studies is more or less one.[1,4,8] 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. Two studies mention a correlation between air pollution indicators and disease, but did not quote the p-value or the confidence interval of the association measure.[7,18] All the studies investigated the impact of air pollution on respiratory health. Studies also focused on birth weight, learning difficulties, immunological, hematological conditions[17] and gastrointestinal, dermal and occular conditions.[19] Eight of the studies addressed chronic respiratory health effects[1-3,9,10,12-16,20], whilst fifteen studies investigated acute respiratory health effects.[1-5,7-9,11-17,19] Ten of the studies had a cross-sectional design[1-5,9,11,12,19,20], two a case-control design[8,10] and four were prospective cohort studies.[7,13-16,17,18] No time-series or any ecological study was located. Local researchers Sitas and Thompson discussed the value of ecological epidemiological studies in developing countries.[21] They pointed 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. These study designs are also implemented sometimes without acknowledging their limitations. These limitations are exacerbated when measures of exposure and disease are based on single measurements and where the population under investigation is homogenous with regard to exposure. The former is responsible for regression dilution bias (underestimation of effect) and the latter for a lack of contrasts between exposure groups. Both limitations would attenuate any exposure disease relationship. 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. As discussed in Chapter 2, indoor and personal air pollution concentrations often correlate poorly with outdoor air levels.[22-33] Indoor, personal and outdoor correlations are dependent on the pollutant under investigation. Nevertheless, time-series studies are at least steppingstones to address air pollution epidemiology in the country as they are 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 resulting from the absence of an electronic health data management system together with electronic birth and cancer registries.

However, most of the local studies discussed in the review are fraught with systematic and random errors. This limits their validity and precision. Methodological limitations included lack of detailed and systematic pollution and/or exposure measurement and variations between studies in the way that disease outcomes are defined and cases found. Most of the studies to date have been observational rather than intervention studies which may ultimately result in more robust evidence on the nature of the relationship between air pollution and health. None of the studies established exposure-response curves for the criteria pollutants (CO, SO_2, NO_x, O_3, particulate matter and lead) due to the applied study design and the lack of quantitative air pollution exposure assessment. It is therefore impossible to use the results of the studies in risk assessment studies or to trace intervention efficiency in lowering air pollution concentrations. Lastly some studies have dealt inadequately with confounding factors, such as socio-economic status, family history of asthma symptoms and age.

Morbidity and mortality are caused by a complex network of risk factors, such as poor nutrition, rapid urbanisation, HIV/AIDS and TB, which usually act in concert. It may thus be tricky to recognise the signal attributable to air pollution beside a myriad of opposing causes of disease and death. Having reviewed the studies conducted in South Africa, it is obvious that environmental epidemiology studies need to be planned and executed better in order to notice the health impacts of air pollution in this country. Von Schirnding and Ehrlich also highlighted this important issue.[34] This issue will be elaborated upon later in Section 8.3.

In the light of the lack of unique South African exposure-response curves, the next chapter addressed the question whether outdoor air pollution is homogenously distributed in Cape Town, South Africa (Chapter 3). This is useful to know when conducting time-series studies, which outputs can be used to derive exposure-response curves. Many pollutants show complex spatio-temporal profiles, which complicates measuring or modelling exposure patterns and obscures subsequent estimation of human exposure. Nevertheless, the majority of air pollution epidemiological studies are based on exposure data from a single central outdoor monitor. This may introduce information bias, which impairs the validity of epidemiological studies. The temporal correlation among outdoor concentrations of a particular air pollutant measured at different sites (inter-site correlation) may be investigated to control information bias. The higher the inter-site correlation, the lower the anticipated information bias when using data from one site for another with similar air pollution sources and the more valid the exposure assessment of the epidemiological study. Exposure-response curves deduced from studies conducted in developed and even other developing countries are not merely applicable in this country. The three global factors that directly or indirectly impact on health – that is the community and social environment, the physical environment and the family and individual environment – are different between and amongst developed and developing countries.[35] In the South African context economical, social and cultural factors may render the population more vulnerable to increased air pollution exposure, due to factors such as poor hygiene, overcrowding, dusty environments, poor nutrition, open dwellings, outdoor lifestyles and the escalating HIV/AIDS epidemic. Cape Town was selected from the main air pollution hotspots in the country (such as Durban, Johannesburg, Pretoria, Richards Bay, Witbank, Vaal

Triangle) as the city adheres to stringent quality assurance guidelines from the US-EPA when monitoring outdoor air pollution.[36]

Air pollution monitoring sites in Cape Town are located in different types of areas. It is therefore anticipated that the spatial correlations for the different pollutants will differ. However, it is still necessary to quantify the temporal inter-site correlations instead of describing the situation qualitatively. It was found that in general 24-h average concentrations of PM_{10} mass, NO_2, NO, SO_2, O_3 and CO are not homogenously distributed during all seasons (Tables 3 to 5, Chapter 3). On average, the most homogeneously distributed pollutant is NO_2, followed by PM_{10} (including Khayelitsha data), PM_{10} (excluding Khayelitsha data), NO, CO, O_3 and finally SO_2 in the Cape Town air shed. Inter-site correlation coefficients for NO_2, NO, SO_2, CO and O_3 vary from 0.456 to 0.832; 0.212 to 0.791; -0.100 to 0.662; 0.302 to 0.676 and 0.123 and 0.557, respectively. PM_{10} measured at Bothasig, City Centre, Goodwood and Tableview presents correlations from 0.261 to 0.859. The PM_{10} inter-site correlation coefficients between Khayelitsha and the other sites (Bothasig, City Centre, Goodwood and Tableview) vary from 0.396 to 0.769. It is suggested that the City of Cape Town do not reduce the number of measuring sites. Limited resources constantly constrain outdoor air pollution measurement. Given the lack of air pollution epidemiological studies in the country, local cities should thus attempt to optimise and update their air quality monitoring networks in such a manner in order to serve both compliance monitoring and epidemiological exposure assessment. Furthermore, as outdoor concentrations of a particular air pollutant were heterogeneously distributed within the city area it is anticipated that there will be adequate outdoor exposure variation. Consequently health data can be linked to these outdoor air pollutant concentrations during future time-series analyses.

Detailed analytic epidemiology studies have to compete with the demands on limited public and research funds for research on common diseases of pressing current importance (such as HIV/AIDS). Another approach for risk estimation is that of health risk assessment studies or even more simply, compliance monitoring. Thus in Chapter 4, the current and future health implications due to outdoor PM_{10} mass exposure in the Khayelitsha sub-district in the City of Cape Town was investigated. The results indicate that elevated PM_{10} mass concentrations are frequently present. During autumn 1999 and winter 2003 the UK Daily Limit value

(50 $\mu g.m^{-3}$)[38] was exceeded 44 times. The number of times the UK DLV may be exceeded should not be more than 35 during a year. Although this requirement was only surpassed twice, the preliminary trend analysis indicates an increase in monthly average and maximum daily average PM_{10} concentrations. The implication is that within a few years the UK Annual Limit value (40 $\mu g.m^{-3}$) will be exceeded. Consequently a higher proportion of the community might be at risk from suffering various detrimental health effects.

Due to the lack of a computerised health data management system in the country, it was not feasible to conduct a time-series study using the high quality outdoor air pollution data from Cape Town. Instead, the 1998 South African Demographic and Health Survey (SADHS) data were analysed in more detail than reported in the SADHS report.[37] The SADHS collected data on various household characteristics, including the type of fuels used for cooking and heating purposes. This endeavour resulted in the calculation of unique South African risk estimates for adult respiratory health (Chapter 5), under five respiratory health (Chapter 6) and 1-59 month old mortality (Chapter 7) due to exposure to indoor air pollution from using dirty fuels for cooking and heating purposes. Previously, most environmental epidemiological studies in South Africa focused only on children's health.[1,4,8,9,13-17,19]

The aim of the analysis reported in Chapter 5 was to determine the prevalence and determinants (occupational and environmental, socio-demographic, BMI, TB) of various respiratory symptoms and diseases in a representative adult population (15 years and older) of South Africa. Data from 13 826 adults from 6 457 households were included in the analysis. The survey revealed relatively low crude prevalence rates for doctor diagnosed asthma (3.7%), chronic bronchitis (2.4%), doctor diagnosed emphysema/bronchitis (4.2%) and TB (2.7%), but higher respiratory symptoms: wheeze and shortness of breath (11.1%), cough with phlegm (6.8%); nocturnal coughing (13.1%), nocturnal wheezing/tight chest (10.8%) (Table 3, Chapter 5). This reinstated the importance of recognising that clinical disease diagnosis only indicates the tip of the iceberg. The majority of people have symptoms. Nearly 1.7% of the respondents reported using asthma medication, whilst 0.5% was using TB medication. In general most of the potential risk factors were significantly related to the respiratory diseases and symptoms in the unadjusted models (Table 4, Chapter 5). The multivariate logistic regression analyses suggested

that the prevalence of respiratory symptoms and diseases could be diminished in South Africa by health promotion predictors (increasing connection to electricity, having a medical aid and improved education) (Table 6, Chapter 5). This preliminary data analyses suggests that the following potential risk factors should be lessened in order to have a beneficial influence on the prevalence rates of respiratory symptoms and diseases: households going hungry, years smoked, households with smokers, exposure at work to fumes, smoke, dust or strong smells and period worked in such a job as well as BMI increase for the underweight and decrease of the obese. Other potential risk factors included age and race. Although there is potential for residual confounding despite adjustment in this preliminary analysis, the documented international evidence on most of the potential risk factors suggests that these associations may be real. The results from the SADHS can also be used in secondary or tertiary prevention, i.e. assess the need and demand for health services based on location of disease and risk hotspots and to evaluate intervention programs in specific target populations.

Chapter 6 reports on the connection between household dirty fuels use for cooking and heating with acute respiratory infections (ARI) in preschool children (<5 years) in South Africa. Two-thirds (67%) of the 4 679 children included in the analysis lived in households using wood, dung, coal and/or paraffin. Nineteen percent suffered from ARI during the 2 weeks prior to the survey (Table 1, Chapter 6). After adjustment, children in households using high and medium polluting fuels for cooking and heating were 26-29% more likely to have an ARI event than children from households using cleaner fuels (OR 1.26; 95% CI : 1.00-1.58 and OR 1.29; 95% CI : 1.02-1.62, respectively). Although there is potential for residual confounding despite adjustment, the better documented international evidence on indoor air pollution and ARI suggests that this association may be real. As nearly half of the 11 million households in South Africa still rely on polluting fuels, the attributable risk arising from this association, if confirmed, could be substantial. Thus the acceleration of implementing interventions to lower exposure to indoor air pollution derived from dirty fuel combustion is imperative in order to improve the health of millions of South African children. Chapter 2 reported on intervention strategies that had already been implemented in the country and recommendations were made regarding future intervention strategies.

The aim of Chapter 7 was to determine the association between dirty cooking and heating fuel use and 1-59 month old mortality in the country whilst controlling for a number of confounders or effect modifiers. Data from 3 556 African/Black children (142 deaths) living in 2 828 households were analysed. The overall mortality incidence rate in the 5 years within the study was 1.473/1000 person-months. The majority of the children lived in households that use dirty fuels for cooking and heating either exclusively or in combination with clean fuels (79%), with no access to a flush toilet (75%), with access to clean water (70%) and low asset index (55%). Most of the children lived in crowded households (>2 people/room)(71%) with poor nutritional status (hungry often/sometimes)(62%). The multivariate analysis suggested that exposure to cooking and heating smoke from dirty fuels is significantly associated with 1-59 month mortality, after controlling for mother's age at birth, water source, asset index and household crowdedness (OR=1.99, 95% CI=1.04-3.68)(Table 3, Chapter 7). Although there is potential for residual confounding despite adjustment, the better documented evidence on outdoor air pollution and mortality suggest this association may be real. As nearly half of households in South Africa still rely on polluting fuels and women of childbearing age perform most cooking tasks, the attributable risk arising from this association, if confirmed, could be substantial. Suggested interventions to reduce 1-59 month old mortality are for women to give birth when they are 19-34 years as well as for households to use clean water and clean cooking and heating fuels. These interventions are based on the adjusted association measures derived in Chapter 7. Sections 8.3 and 8.6 will address recommendations regarding future intervention strategies.

8.2 Potential Bias and Limitations

There are some important study specific limitations in the analyses from Chapter 3 to 6, which should be taken into account when interpreting the results. These coincide with those addressed by Ritz et al.[40] They discussed the pros and cons of various systems for the examination of environmental health questions. Hill's causation guidelines should be kept in mind (Table 1, Chapter 1).

The SADHS had a cross-sectional design. Cross-sectional studies are weak to prove causation as they are subject to difficulties interpreting the temporal sequence of events since health status and determinants are measured simultaneously. In Chapter

7 the proportional hazards assumption was confirmed for each independent variable. However, this test cannot indicate slight changes in exposure variables over the 5 year period. A large proportion (43%) of women still lived in their current house when the child was born, so it was assumed that the toilet facility and water source variables remained constant over time.

There is a good chance that differential or nondifferential misclassification of disease and exposure status may be present in using the 1998 SADHS data due to recall bias, reporting bias and information bias. This results in statistical significance arising by chance with either an over- or underestimation of the association measures. Consequently the direction of bias on the association is not easy to predict. Recall bias may be present as respondents answered questions involving events occurring in the past, such as household going hungry and breastfeeding patterns immediately after birth. Reporting bias may be present, since the possibility exists for respondents to answer in such a fashion that may be more socially desirable or in ways that they perceive would get a more approving response from the interviewers. Information bias may be due to the reliance on self-reported data. Respondents with current symptoms and diseases may be more likely to report exposures and remember past TB infections than asymptomatic respondents.

Self-reporting of emphysema and bronchitis can be used only as a very rough guide to the prevalence of chronic lung diseases for a variety of reasons. First, use of diagnostic terms reflects health service access, which in South Africa varies considerably by socio-economic status and geography. A term such as emphysema is likely to be used inconsistently by medical practitioners based on varying clinical criteria. Lung function testing, which contributes important information to diagnosis, is uncommon at primary care level. Bronchitis also is a non-specific term that would elicit reports of acute bronchitis as well as chronic bronchitis. Acute bronchitis is a common ailment, often a mild and self-limiting viral infection, which may occur without underlying chronic disease. A literature review of asthma symptoms assessed by questionnaire found that "physician-diagnosed asthma" had a mean specificity of 99% and a mean sensitivity of 68% for asthma defined by symptoms, suggesting that underdiagnosis is more likely than overdiagnosis.[41] Finally, asthma in adults is probably frequently misdiagnosed as bronchitis. On the one hand, self-reporting of asthma is likely to reflect some degree of under-

diagnosis. On the other hand, asthma rates may be inflated by confusion with emphysema and chronic bronchitis, particularly in older age groups. Reporting of symptoms is less likely to be influenced by contact with health services than is reporting of diagnoses. The chronic bronchitis symptom complex is defined by chronic bronchitis every day for at least 3 months a year, for at least 2 successive years. It was one of the earliest symptom complexes to be defined by standard respiratory questionnaires, and has entered into common usage as both a clinical and epidemiological definition.

Information on ARI is based on mothers' reports and no clinical measurements were undertaken and smoke exposure was ascertained from type of fuel used for cooking and heating. Although the symptomatic definition used here is aimed to assess acute lower respiratory infections (ALRI) in children, some acute upper respiratory illness may have been integrated in the conveyed prevalence. As it is impossible to separate ALRI from these data, the term ARI is used in this study, not ALRI. In developing countries such as South Africa, where clinical data on ARI are frequently unattainable or very weak, the symptomatic definition of illness used here is assumed to present a reasonably accurate estimation of ARI in the population. Furthermore, Kauchali et al conducted a local rural study on maternal perceptions of ARI in children under 5 years of age.[42] Maternal recognition of respiratory distress was good (sensitivity 91.3%, 95% CI=86.8-95.8%; specificity 95%, 95% CI=89.5–100%), with little variation between mothers (kappa = 0.704). Thus, following the results from Kauchali et al it is anticipated that most mothers' would at least have identified ARI among their children.[42] However, it is not so clear whether differential or nondifferential misclassification may be minimised by the supporting results of Kauchali et al.[42]

The generalisibility of data is determined by the non-response rate. The response rate at the household level was 97% of 12 860 households in 966 EAs. Of the 6 457 households selected for the adult survey, 95.3% were completed. At the individual level, 92.6% of eligible adults were included in the survey, although not all of them had all the measurements taken. The overall response rate for the adult survey was 89.7%. It was substantially lower in Gauteng (67.5%) where a large proportion of adults were not at home (13%). The response rate was higher in the non-urban than urban area. For the women's health survey, the overall response rate

was 92.3%. Thus the bias that might be introduced by non-response is relatively low for the SADHS data.

Other factors that might contribute to adult and childhood morbidity and mortality, such as outdoor and indoor air pollution sources (location of household close to industry, transportation, waste fill site, insecticide and fertiliser use), town/city of household to request meteorological variables from South Africa Weather Bureau, mother's pre-pregnancy weight, child's birthweight, mother's exposure to other pollution sources and risk factors during pregnancy, along with current HIV/AIDS and TB epidemics, were not recorded. Furthermore, information was not collected on mother's smoking status. Excluding environmental tobacco smoke from the analysis might introduce substantial bias (differential or nondifferential) as 24% of women in South Africa smoke.[37] Thus the direction of bias on the calculated association measures is not easy to predict. The current HIV/AIDS epidemic along with escalating number of TB infections could also influence the association between 1-59 month mortality and exposure to smoke from dirty fuels. As the SADHS survey did not collect indicators of potential HIV/AIDS status of the child, biological mother or any household member, the attributable fraction due to HIV/AIDS could not be calculated. However, the TB prevalence rate was low (2.7% among 13 826 adults)(Table 3, Chapter 5). Adult household members diagnosed with TB and who lived in households with smokers (34.2% among 13 826 adults), were not linked with the 1-59 month mortality dataset as this reduced the number of deaths considerably.

Substantial evidence had shown that parents perceive the inherent healthiness of a child through knowledge of their own genetic endowments and health characteristics of the household and neighbourhood.[43-47] If parents perceive that their child's inherent healthiness is low, they may alter their fertility decisions, leading to selection bias in the sample of potential births. However, considering first births exclusively in the analysis was not feasible as this reduced the sample size considerably. Also, the number of children per age group decreased with increasing age and this is likely to be due to three contributing factors. First, close to 7% of the children in South Africa die by the time they reach 5 years of age and this high mortality rate may result in fewer older children in the sample.[48] Secondly, as children get older they are more likely to be sent away to live with relatives in urban

areas. It was found that 331 black children did not live with their mothers (8.5%, N=4 114 with no missing values)(Chapter 7). Lastly, the survey was conducted between January and September 1998, resulting in lower birth counts compared to 1993-1997.

Notwithstanding the lack in the measurement of smoke exposure, the uniformity in the significance of crude and adjusted effects of dirty fuel use on adult respiratory, childhood ARI and 1-59 month mortality implies a probable 'exposure–response' relationship. Many households in South Africa in general use a combination of cooking and heating fuels. The calculated effects may be underestimated if only considering using high polluting fuels (wood and dung) exclusively and not in combination with paraffin, coal, LPG/natural gas and/or electricity. This is also expected when using medium polluting fuels (paraffin, coal) exclusively and not in combination with LPG/natural gas and/or electricity.

The most common indicators used for measurement of socioeconomic status are income level, occupation, and educational level.[49] However, Demographic and Health Surveys traditionally do not include questions on income and expenditure. Educational level measures one aspect of socioeconomic status and it cannot be ruled out that the results would have been different with another measure. However, the relation between socioeconomic status (SES) and socioeconomic factors with respiratory health in adults is not well understood. Existing studies are heterogeneous regarding the definition of the socioeconomic indicators used. More than a third (35%) of the women never married, so it could not be assumed that the current partner's job or education remained the same over the 5 year period in the 1-59 month mortality analysis. Mishra analysed the Zimbabwean DHS data and calculated a household standard of living index as a possible confounder in the association between household cooking fuel use and ARI in preschool age children (<5 years).[50] In Chapters 6 and 7 a similar approach was followed.

Another bias is that employed low-income men are underrepresented, as they work overtime, shifts or away from home. Furthermore, male worker hostels, a common form of housing for African migrant workers in mining and certain urban areas, were not surveyed. Nevertheless the results presented here are the first national survey of the symptoms and prevalence of chronic lung disease in South Africa.

Previous morbidity information was derived from surveys of selected adult populations only.[51,52]

There is a chance of some selection bias in the sample due to ARI-related mortality. The risk estimates of effect of cooking smoke on ARI are downwardly biased as children living in households using medium and high polluting fuels are more likely to die from ARI. Then again, given high prevalence of ARI and relatively small number (n=269, 5.28% of the sample, N=5 093) of deaths in the sample, the impact of this bias on the estimated effect is likely to be little.

Although the influence of susceptibility to the detrimental health impact of being exposed to emissions from dirty fuels and the selected confounders was controlled by adjusting for age, socio-economic status and gender, some residual susceptibility might still be present. Bias in risk estimates will arise if individuals with similar exposures but different susceptibilities are treated the same. This issue will be addressed later in Section 8.3.

Only controlling for confounding is not enough. Attention should also be paid to confounder measurement errors (random or systematic). Greenland has pointed out that errors in measurement of a confounding variable will tend to cause partial loss of an ability to eliminate confounding bias.[53] Kupper has shown that an inaccurate surrogate confounder can produce seriously misleading inferences.[54] The presence of random and/or systematic errors in the measurement of confounders will either influence the true value of the association measure between the exposure variable or health outcome towards or away from an insignificant association.

During the analysis it was assumed that confounding is additive and not multiplicative. If confounding is additive, then the confounding variable would produce the same additional risk of a health outcome in the exposed and unexposed; but if the health outcome is rare in unexposed, it would follow that the confounder might account for a much larger proportion of health outcome in that group. Conversely, if the two exposures act multiplicatively, the proportional increase in health outcome rates due to confounding would be the same in exposed and unexposed; but if the health outcome is more prevalent in the exposed group, the absolute increase would be larger in the exposed. This issue thus has important risk

assessment and public health policy implications. Again, Greenland has shown that errors in measurement of a covariate can distort its modifying effect and possibly introduce an apparent interaction where none exists.[53]

Limitations of using air pollution data from the City of Cape Town included the low PM_{10} data availability at Khayelitsha for most of the period under investigation, lack of meteorological data (temperature, relative humidity, wind speed, wind direction, rainfall) collected at each monitoring site and inter-pollutant correlations at each monitoring site.

8.3 Improving Secondary Health and Exposure Data Sources to enhance Public Health

As mentioned in Chapter I public health may be improved by primary, secondary or tertiary prevention. This thesis focuses on primary prevention, that is the identification of environmental risk factors, such as outdoor air pollution and the use of highly polluting fuels for cooking and space heating, whilst controlling for confounding. Chapter I also addressed the ultimate endeavour of epidemiology, namely to identify modifiable determinants of disease occurrence and progression and to contribute in testing the efficacy and effectiveness of interventions on these determinants including the health services. For public health to be improved by epidemiological studies it is imperative that these studies should attempt to minimise systematic and random errors and subsequently strengthen their validity and accuracy.

Recently, Ritz et al discussed the pros and cons of various systems for the examination of environmental health questions.[40] They highlighted that environmental public health tracking networks should be synonymous with a dynamic process requiring regular system updates to a) incorporate new technologies to improve population-level exposure and disease assessment, b) allow public dissemination of new data that become available, c) allow the policy community to address new and emerging exposures and disease "threads," and d) evaluate the effectiveness of these networks over some appropriate time interval. It will be necessary to weigh the benefits of surveillance against its costs, but the major challenge will be to maintain support for this important new system. They concluded that prevention might be the only rationale option (discussed later in this

section and Sections 8.6), even if multifactorial diseases do not lend themselves to surveillance data driven evaluations of intervention strategies.

Given the lack of resources for outdoor air pollution measurement and the dearth of air pollution epidemiological studies in the country, local cities should thus attempt to optimise and update their air quality monitoring networks in such a manner as to serve both compliance monitoring and epidemiological exposure assessment (addressing issues raised by Ritz et al above).[40]

Stemming from the preliminary results of this investigation along with its associated limitations, the following thoughts are suggested to the City of Cape Town:

- Review the number and location of the air pollution monitors in the City;
- Increase the number of pollutants measured at Khayelitsha and Oranjezicht;
- Increase the data response at Khayelitsha for PM_{10};
- Collect meteorological data (temperature, relative humidity, wind speed, wind direction, rainfall) collected at each monitoring site;
- Increase the number of O_3 monitors in background areas;
- Place O_3 monitors downwind from precursor sources;
- Investigate the current air pollution trends against health guidelines and
- Establish the surrogate or confounder relationship between air pollutants in a time-series analyses (i.e inter-pollutant correlations at each monitoring site)

Although O_3 is not a primary pollutant, it is at the moment unclear whether it is generated in the Khayelitsha area. Furthermore, it is suggested not to reduce the number of measurements per day as hourly measurements are needed to indicate peaks and for acute health effects assessment. These suggested recommendations aim at increasing the accuracy and reliability of using the air quality network data in epidemiological studies. It is furthermore recommended that similar analyses be conducted using air quality data from the other major cities in the country that maintain relatively extensive air quality monitoring networks under rigorous quality assurance guidelines. If quality assurance guidelines are not adhered to, it is recommended that immediate provision be made to fulfil this criterion. It is

recommended that all air quality monitoring networks in the country should apply for accreditation in the near future by the South African National Accreditation System (SANAS). Ideally a comprehensive exposure assessment study with outdoor, indoor and personal measurements for SO_2, NO_2, O_3 and $PM_{2.5}$ should also be conducted in the immediate future on a representative sample of the South African population along with information on distal and proximal causes of health outcomes, such as poverty, home design and socio-behavioural practices. Correlation coefficients between distal and proximal causes and quantitative personal, indoor and outdoor air pollution measurements can be determined and used to develop exposure assessment models. Validating and applying these models in future studies will save time and money by circumventing the measurement of quantitative personal, indoor and outdoor air pollution measurements in every study. The indoor and outdoor air pollution measurements as well as the individual distal and proximal causes might also be used as surrogates for personal air pollution measurements. However, explicit criteria for acceptable surrogate measures along with the need to take error into account when surrogates are used, should be set. Rosner et al have shown that for correlations between surrogate and true measures of exposure less than 0.8, the odds ratios estimated by logistic regression will differ markedly for the surrogate and the true exposure measure, while much less bias will occur when correlations between the two measures are 0.8 or greater.[55]

The local authorities are encouraged to consult the latest scientific literature with the assistance of research and academic institutions when updating or designing their air quality monitoring networks. Issues regarding improved collaboration in the field of air pollution epidemiology will be deliberated upon in Section 8.5. Chang and Tseng discussed the optimal evaluation of expansion alternatives for existing air quality monitoring networks in a growing metropolitan region, which poses many uncertainties such as the changing population density and the changing emission sources in the urban environment.[56] They discussed the principles for siting air quality monitoring stations through a multiobjective analysis and suggested that monitoring stations should be located in areas

- of high population density;
- where pollution concentrations are expected to be highest;
- where the highest frequency of violation can be detected;

- where significant growth is expected to occur and
- near major downwind sources.

The optimisation modelling they addressed considered three objectives: the maximisation of

- protection capability of the highest population density;
- detection capability of the highest pollution concentrations and
- detection capability of the highest frequency of violation of health guidelines/standards, along with cost, effectiveness and efficiency factors.

Chow et al reviewed the design of monitoring networks to present outdoor human exposure.[57] Kanaroglou et al discussed the establishment of an air pollution monitoring network for intra-urban population exposure assessment, which is quite relevant in epidemiological studies.[58] The impetus for their study was to address the limitation of locating monitors in an ad hoc fashion, which favours the placement of monitors in source "hot spots" or in areas deemed subjectively to be of interest. Their study addressed the development of a formal method of optimally locating a dense network of air pollution monitoring stations and the subsequent development of an exposure assessment model based on these monitoring data and related land use, population and biophysical information.

Before addressing issues on how to improve the next SADHS to ultimately reduce random and systematic errors and consequently improve the accuracy and validity of derived association measures, it is important to remember the general purpose and characteristics of health surveys as addressed in Section 1.2. The topics that the 1998 SADHS addressed can be classified according to the following three categories: Health status (disease specific morbidity, chronic conditions, self-assessed health, height and weight as well as lung function tests), lifestyle and health habits (various indicators) and medical consumption (hospitalisations, general practitioner consultations and use of medicines).

The SADHS addressed adequately the health status and medical consumption sections as it covered serious prevalent health conditions (albeit self-reported) in the

country, such as ARI, chronic bronchitis and child mortality. However future SADHS should ideally also include other direct and indirect health consequences related to use of dirty fuels identified by Smith, Bruce et al, Ezzati et al and a WHO report (Table 1).[59-62]

However including more questions in any established survey will be a challenge as there are many other competing health risk needs and interests and against the overall limitations of questionnaire length. From the direct and indirect health consequences listed in Table 2 it is suggested to prioritise on the following: Birth defects, low birth weight, paraffin poisoning, restrictions on opportunities for education and issues arising from gender power imbalance.

Table 1 Other direct and indirect health consequences related to use of dirty fuels

Indicator
Direct
Birth defects
Low birth weight
Cataracts
Burns
Paraffin poisoning
Indirect
House fires
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
Issues arising from gender power imbalance
Decision-making about the use of energy and appliances
Impact of inter-relationships between scarcity of fuel and stressed local environments

Mental and physical disability is not high on the agenda in South Africa, thus including a question on birth defects will improve this field of study. In general, birth weights should be better recorded in the country as most of the children had no birth weight indicated in the SADHS. Studies have found that underweight babies tend to be sicker later in health.[63-66] The use of paraffin is encouraged over the use of coal or wood for household energy needs, but the direct health impacts of paraffin use should also be highlighted, such as paraffin poisoning among young children. Restrictions on opportunities for education and issues arising from gender

power imbalance are important to address in order to alleviate poverty, a distal cause of ill health. Of these five prioritised direct and indirect health consequences, low birth weight is the most likely to be influenced by random and systematic error (such as errors in reporting birth weight, recall bias). Although birth certificates are issued in the country (which are called "The Road to Health Cards"), they are lost or destroyed in most cases after a few years. An alternative should thus be sought to record birthweights in South Africa.

Comparing the limitations identified in using the 1998 SADHS data to those from the review in Chapter 2, it is evident that not much progress has been made in air pollution epidemiology in the country during this analysis. However, due to the large sample size and many risk factors identified in the 1998 SADHS, it had the advantage over previous local studies in that it attempted to deal more adequately with confounding factors (Table 2).

In order to address at least more confounders in air pollution epidemiology analyses (Table 3) it is recommended that the lifestyle and health habits sections of future SADHS need to be planned better. Furthermore, methodological limitations of previous South African air pollution epidemiology studies included the lack of detailed and systematic pollution and/or exposure measurements and variations between studies in the way that disease outcomes are defined and cases found. These limitations were also present in the 1998 SADHS. Neither the analyses of the 1998 SADHS data nor previous local studies established exposure-response curves for the criteria pollutants. This is due to the applied study designs and the lack of quantitative air pollution exposure assessment. It is hence not feasible to employ the results of the studies in risk assessment studies or to track intervention efficiency in reducing air pollution concentrations. As it is not feasible to collect personal, indoor and outdoor air pollution measurements in a DHS, it is thus important to capture more indoor air pollution exposure indicators in future SADHS (Table 4).

Table 2 Air pollution exposure indicators and confounders addressed in this thesis

Characteristics/feature	Adult respiratory health (Chapter 5)	Childhood ARI (Chapter 6)	I-59 month old mortality (Chapter 7)
Fuel use for cooking and heating	✓	✓	✓
Residence in urban/rural area	✓	✓	✓
Connected to electricity	✓		
Persons per room	✓		✓
Household going hungry	✓		✓
Covered by medical aid/medical benefit scheme	✓		
Payment of medicine	✓		
Age distribution in years/months	✓	✓	✓
Ethnic identity	✓	✓	
Ever smoked tobacco, used snuff or chewed tobacco	✓		
Ever smoked at least 100 cigarettes in lifetime	✓		
Years smoked on a daily basis – distribution	✓		
Frequency smoking	✓		
Household with smokers	✓		
Job with smokers	✓		
Ever worked in job where regularly exposed to smoke, dust, fumes or strong smells	✓		
Period worked in job exposed to smoke, dust, fumes or strong smells – distribution in years	✓		
Gender	✓	✓	✓
Educational status	✓		
BMI	✓		
Province of residence	✓	✓	✓
Birth order		✓	✓
Number of children in household		✓	
Mother's age at birth (years)		✓	✓
Mother's education (in years)		✓	
Household standard of living		✓	✓
Treatment received for ARI		✓	
Preceding birth interval (months)			✓
Breastfed			✓
Type of toilet facility			✓
Water source			✓
Year of birth			✓

Table 3 Confounders identified to be addressed in future SADHS

Confounder	Adult respiratory health (Chapter 5)	Childhood ARI (Chapter 6)	I-59 month old mortality (Chapter 7)	Feasibility to include confounder in SADHS
Mother's smoking status before birth of each child		✓	✓	✓
Mother's smoking status after birth of each child		✓	✓	✓
Mother's educational level at birth of child		✓	✓	✓
Partner's job/educational status at child birth			✓	✓
Mother's pre-pregnancy weight		✓	✓	
Child's birth weight		✓	✓	Perhaps
Mother's exposure to other air pollution sources during pregnancy		✓	✓	
Mother's exposure to other confounding risk during pregnancy		✓	✓	
Insecticide use at household during past 2 weeks	✓	✓	✓	✓
Fertiliser use at household during past 2 weeks	✓	✓	✓	✓
Meteorological variables	✓	✓	✓	
HIV/AIDS status of participant/mother/child	✓	✓	✓	Perhaps
Clinically diagnosed TB status of participant/mother/child	✓	✓	✓	✓
Perception of health risk due to dirty fuel use	✓	✓	✓	✓
Diet: list food consumed during past 2 weeks	✓	✓	✓	✓
Garbage removal service present	✓	✓	✓	✓
Outdoor air pollution sources near household (i.e. industrial, transportation, burning of residential waste close to home or waste fill site located close to home)	✓	✓	✓	✓
Susceptibility (i.e. family members with the health outcome; number, ages and relationships of family members at risk)	✓	✓		

Once more, including more questions in any established survey will be a challenge as mentioned previously. In order to prioritise on which confounders and air pollution

indicators to focus in future SADHS, it is important to remember the need to measure confounders and indicators with minimum measurement errors (random or systematic). Table 3 indicates the confounders that are most feasible to include in a DHS with limited recall bias. HIV/AIDS status of participant/mother/child will be a challenge to determine due to the stigma attached to HIV/AIDS. However, it is important to address this challenge in order to compare the attributable burden of indoor air pollution exposure and HIV/AIDS on overlapping morbidity and mortality outcomes in South Africa. Black et al used a prediction model to estimate the distribution of deaths in children younger than 5 years by cause (namely diarrhoea, pneumonia, malaria, measles, AIDS, neonatal causes other causes and unknown causes).[67] No uncertainty bounds are available for the AIDS for 42 countries (including South Africa), which contributed 90% of all such deaths in 2000.[68] Estimates and uncertainty bounds were: 22% of deaths attributed to diarrhoea (14–30%), 21% to pneumonia (14–24%), 9% to malaria (6–13%), 1% to measles (1–9%), 3% to AIDS, 33% to neonatal causes (29–36%), 9% to other causes and fewer than 1% to unknown causes. No uncertainty bounds are available for the AIDS estimate as the model did not produce these data (country-level estimates from UNAIDS were used).[69]

The current SADHS included a question on the length of stay at the current dwelling. Thus bias due to migration is controlled for when posing a question on outdoor air pollution sources near household. Maternal education status and smoking status before birth and after a child's birth as well as the partner's job/educational status have been found to be important indicators of child health outcomes in previous studies.[70-74] Diet and nutritional status is also important for health. Although the frequency of a household going hungry was determined, a report by the South African Medical Research Council (MRC) on poverty and chronic diseases in the country also emphasised the lack of detailed adult nutritional data collected in the 1998 SADHS.[75]

Due to the high concentrations detected indoors when using insecticides it is important to address this confounder. Fertiliser use also has a huge influence on human health. Clinically diagnosed TB status of participant/mother/child is an important confounder in respiratory health. The presence of garbage removal service and other outdoor air pollution sources near households also warrant attention. It is

necessary to address perception of health risk due to dirty fuel use for health promotion strategies.

Table 4 Indoor air pollution exposure indicators to be addressed in future SADHS

Indicator	Feasibility to include confounder in SADHS
Types of fuels used for cooking during past 12 months	✓
Types of fuels used for heating during past 12 months	✓
Types of fuels used for lighting during past 12 months	✓
Stove type used during past 12 months	✓
Current housing characteristics (i.e location of cooking; number and size of rooms; the relation between stove location and other rooms; number and size of doors and windows)	✓
Current socio-behavioural characteristics (i.e. length of cooking and heating intervals; status of doors and windows during cooking and heating; participation of individual household members in cooking and other energy related tasks; amount of time spent indoors and near the stove when burning)	✓

The issue of susceptibility raised in the previous section warrants some attention. There are a number of epidemiologic designs for assessing sensitivity to environmental exposures.[76] As a measurement problem, the central issue is whether the marker for sensitivity being examined is a measurement of the genotype itself, some host characteristic or family history. As it is not feasible to assess susceptibility via genotyping in SADHS data, it may be assessed by host characteristic or family history. For family history as a marker of susceptibility to a disease, the basic minimal information that needs to be collected is the identification of the family members with the disease and the number, ages and relationships of family members at risk. This information should be collected systematically for all first-degree relatives (parents, siblings, and offspring) and possibly for all second-degree relatives. As the objective is to examine family history as a marker of sensitivity to an environmental exposure, every effort should be made to obtain exposure information on all relatives, not just the affected ones. However, due to recall bias, it is not feasible to include questions on family history in the future SADHS.

Meteorological variables may be addressed in future SADHS if a question is included in which city/town the household is located. Data may then be requested

from the South African Weather Service. All the indoor air pollution exposure indicators are feasible to address in the next SADHS. Lastly, future SADHS need to include male worker hostels, a common form of housing for Black migrant workers in mining and certain urban areas in order to reduce selection bias.

Chapter 2 identified that ten of the studies had a cross-sectional design, like the 1998 SADHS.[1-4,5,9,11,12,19,20] The inability to minimise measurement errors (random or systematic) of health, exposure and confounder variables is due to inherent limitations of any DHS study design (i.e. cross-sectional). It is also important to realise that most of the current and identified confounders as well as exposure indicators are only applicable in analyses involving acute diseases.

Future options to address the limitations posed by questionnaire length and the inherent study design of the SADHS need to be explored. A specific questionnaire can be designed for half of the households interviewed in the next SADHS or other surveys could include relevant questions for confounders and exposure indicators identified, such as the annual October household survey or smaller surveys conducted in typical South African settings. An indoor air pollution exposure questionnaire could also be applied in the three INDEPTH (International Network of field sites with continuous Demographic Evaluation of Populations and Their Health in developing countries) operational in the country.[77-80] INDEPTH has 33 demographic surveillance sites in 18 different countries. Its vision is to harness the collective potential of the world's community-based longitudinal demographic surveillance initiatives in resource constrained countries to provide a better, empirical understanding of health and social issues, and to apply this understanding to alleviate the most severe health and social challenges.

An option to circumvent the issue on measurement errors, participants can be asked whether they would like to be contacted in the future for inclusion on follow-up studies. Another identifying indicator than the South African identification number should be used in tracking people's use of health services. In general people do not inform the Department of Home Affairs of their new addresses after relocating. One might also ask them if they would mind that researchers track their future use of clinics and hospitals for application in follow-up studies. This would of course require a properly updated electronic health information system in the country.

Selection bias may be introduced in these suggested follow-up studies, as approximately 14.9% of the South African population was covered by a medical aid scheme in 2003.[81] People covered by medical aid schemes access private hospitals and clinics, whose databases are in general inaccessible to research institutions. Furthermore, the majority of the White population had access to a medical aid scheme (65.2%), followed by Indians/Asians (35.0%), and then Coloureds (19.3%). The African population had the smallest proportion of people with access to a medical aid scheme (8.0%). In a speech by the Health Minister during July 2005, it was announced that only 15-20% of South Africans have a high degree of access to health services.[82]

The psychosocial stress that may be associated with exposure to a perceived environmental hazard can potentially confound, mediate or modify any associations between the exposure and disease.[76] This issue will become a challenge in future SADHS as communities (hopefully) will be sensitised around the detrimental health impact of using dirty fuels. Stress might operate indirectly and cause exposed individuals to alter risk behaviours. Stress also could have an artificial association with the health outcome of concern due to changes in care seeking, diagnostic practices or self-reported health states. Alternatively, concern about environmental exposures could cause adverse outcomes other than those potentially associated with the perceived hazard, such as anxiety and depression.

The issue of stress as a confounder, effect modifier, mediator, indicator of some methodological bias or even as an exposure or outcome needs to be explicitly addressed in future environmental epidemiological research conducted on sensitised populations. Ozonoff et al and Roht et al developed some relevant methodology in studies of communities near toxic wastes to distinguish between biologic effects of exposure to hazardous substances at such sites and either symptoms of stress or altered symptom reporting.[83,84] These efforts include use of a scale to measure hypochondriasis and stratified analysis of self-reported symptoms to take account of subjects' perception about the source of pollution. Hatch and Thomas emphasised that environmental epidemiologists need to learn when and how to address the issue of psychosocial stress in order to clarify interpretation of health effects studies and to estimate the importance of stress in its own right.[76] Consideration should be given to measuring perceived stress and physiologic indicators of stress as well as to

collecting data on methodological covariates such as motivation to participate, interest in receiving health care, and beliefs about the exposure in question as a cause of adverse health effects.

The influence of psychosocial stress may be controlled by clinical diagnosis during intervention studies. This approach is however impractical in future SADHS. The inclusion of specifically clinical diagnosed health outcomes should then be preferred above self-diagnosed ones. Questions should also be included whether the participant sought health care for the health outcome of concern.

The need for a central location for data storage of all exposure, demographic and health data is crucial in South Africa – linking back to the issues raised by Ritz et al above.[40] The collection of high quality outdoor air pollution data in all major cities in the country is needed, together with the implementation of an electronic health data management system along with electronic birth and cancer registries as soon as possible in order to conduct time-series studies.

8.4 Usefulness of Secondary Health and Exposure Data in enhancing Public Health

Despite the limitations mentioned in Section 8.2, the air quality data in the City of Cape Town is quite useful in conducting trend analysis or perform preliminary epidemiological studies. From the results in Chapter 3 it is advisable when conducting epidemiological studies to link clinic/hospital data with air pollution data collected from the nearest air quality monitor. This will reduce information bias. The air quality analysers continuously assess real-time concentrations of criteria pollutants using US-EPA equivalent methods in accordance with ISO 17025 guidelines.[36] These instruments measure the concentration of ambient air pollutants in 20 second scans and values can be expressed in short term (10 min), one hour, 24 hour, monthly or annual averages. Thus the data can be used to check compliance with air quality guidelines/standards (usually applying 24 hour average values), for air pollution alerts (usually applying values averaged over a few minutes to one hour), determination of acute and chronic human health and ecological effects (usually applying values averaged over one hour and 24 hours). Furthermore, the community can view the data on the City's web site soon after statistical analysis.[85]

Air quality data in the City of Cape Town can also be linked to school absenteeism as a proxy for child health until a health management system has been established for the country. While school absenteeism is a crude proxy measure of morbidity, which is also influenced by non-health-related factors, the majority of school absences are health-related and attributable to either respiratory infections or gastroenteritis. Excluding data from Mondays and Fridays may control confounding due to non-health-related factors. These days are often associated with non-health-related absence. Houghton et al suggested that school attendance data should be computerised.[86] This will facilitate the routine analysis of the relationship between pollution data and school absenteeism.

Table 5 lists the advantages and disadvantages of using 1998 SADHS data in air pollution epidemiological analyses. Furthermore, Hupkens et al pointed out that health surveys are especially relevant for health indicators that cannot be collected by means of hospital or clinic records, such as indicators on health status (for example as the prevalence of chronic conditions and self-assessed health as a measure for people's well-being), lifestyles (like smoking habits and alcohol consumption) and medical consumption (such as the use of medicines).[87]

Also modern health indicators like health expectancies and disability-adjusted life years (DALYs) can only be calculated with the help of survey data. Furthermore, the abundance of national health interview survey data makes these data valuable for studying the diversity in health in different country settings, which may advance the understanding of the determinants of health and disease. However, the value of these survey data for international comparisons depends on the comparability of the survey questions, frequency and methodology of the surveys (such as type of survey, sample frame and interview method). Given the large number of national health interview surveys, harmonisation may be a long, but also a promising endeavour. However, as most studies addressing air pollution epidemiology in Europe and North America are specifically designed for their unique purpose, the use of health survey data in these parts of the world are mainly to fulfil the objectives of the health survey and in international comparisons.

Table 5 Advantages and disadvantages of using 1998 SADHS data in air pollution epidemiological analyses

Advantages	Disadvantages
• Less time-consuming and costly than more rigorous prospective cohort studies	• Cross-sectional design and associated disadvantages
• Information on national representative sample of population	• Information on important confounders not collected
• Provide information on intercorrelations among numerous variables	• Unable to minimise measurement errors (random and systematic) of health, exposure and confounding variables
• Detect high-risk groups	
• Give hints about causal relations	
• Generate hypotheses	
• Provide a baseline for health and risk factor prevalence rates to be compared with future measurements	
• Provide information on need and demand for health services	
• Provide information to evaluate intervention programs in specific target populations	

The Bellagio Study Group on child survival recently discussed the requirement to transform knowledge into action, which included leadership, strong health systems, targeted human and financial resources and modified health system to ensure that poor children and mothers benefit from health research.[67,88-91] In order for the public to benefit from epidemiological results, these must be translated from theory into public health practice more efficiently. Population attributable fractions (PAFs) are useful for estimating the proportion of disease cases that could be prevented if one or more risk factors for that disease were reduced or eliminated.[92-99] Thus, how many cases of disease or premature mortality could be avoided if indoor air pollution due to combustion of dirty fuels for cooking and heating purposes could be eliminated completely in South Africa?

The results from Table 6 are calculated using equations 1 and 2.

$$\text{Attributable fraction} = \frac{(\%\ \text{population exposed} \times \text{relative risk}^* + \%\ \text{population unexposed} \times 1) - 1}{\%\ \text{population exposed} \times \text{relative risk}^* + \%\ \text{population unexposed} \times 1} \quad (1)$$

** The estimated odds ratios will be used in Table 6*

$$\text{Attributable burden} = \text{attributable fraction} \times \text{current disease level}^{**} \quad (2)$$

*** Product of population size and prevalence rate of health outcome in Table 6*

Table 6 reflects the huge impact of indoor air pollution in the country. However, one should realise that epidemiological results alone will not change policy. This thesis does not by all means want to dictate to policy-makers how to apply these results. Rather, it is paving the way to open communication between epidemiological research and policy in South Africa. The Science-Policy-Interface (SPI) is complex, as was reported by the Thematic Network on Air Pollution and Health in Europe (AIRNET) initiative.[100] Communication between science (epidemiology studies) and policy is a dynamic process that is influenced by numerous factors. A detailed discussion of these factors is beyond the scope of this thesis and the reader is directed to Baker et al for an introduction.[101]

Decision-making is not a simple process. A decision-maker must choose between competing alternatives, and may face uncertainties at every step. These difficulties, however, are no excuse for lack of action. Given the strengths and weakness of the results (Section 8.2), increasing evidence about potential health problems could certainly aid the decision-making process, but waiting for more evidence implies that someone has to endure the suffering in the mean time.[102] As far back as 1965, Hill noted, "*All scientific work is incomplete – whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge that we already have, or to postpone the action that it appears to demand at a given time.*"[103]

This thesis set out to identify the magnitude of health outcomes caused by indoor air pollution exposure. However, these results (when considering their strengths and weaknesses) can be utilised for economic analysis of exposure, environmental equity

and evaluation of intervention and policies. The results cannot be used for standard or guideline setting as no quantitative exposure data were collected nor was the study design appropriate.

Table 6 Number of cases attributable to indoor air pollution due to medium and high polluting fuels†

	Chapter and Table	Fraction exposed	RR (95% CI)	Attributable Fraction	Prevalence of health outcome %	Population size	Attributable Burden
Adult nocturnal coughing due to *no* access to electricity	Chapter 5 Tables 3 and 5	0.35	1.72 (1.09; 2.70)	0.20 (0.03; 0.37)	13.1	30 454 485*	803 006 (121 833; 1 488 260)
Adult cough with phlegm due to *no* access to electricity	Chapter 5 Tables 3 and 5	0.35	1.64 (1.12; 2.44)	0.18 (0.04; 0.34)	6.80	30 454 485*	378 989 (83 472; 693 973)
<5 years ARI due to *high* polluting fuels use	Chapter 6 Tables 1 and 2	0.39	1.29 (1.02; 1.62)	0.10 (0.01; 0.19)	19.26	4 449 816**	87 082 (6 633; 166 879)
<5 mortality due to *dirty* fuels use	Chapter 7 Tables 1 and 3	0.79	1.99 (1.04; 3.68)	0.44 (0.03; 0.68)	-	41 171***	18 068 (1 261; 27 963)

† *Refer to the individual chapters for the definition of fuel categories*
* *≥15 year group*[104]
** *<5 year group*[104]
*** *Total number of deaths for 0-4 year olds during 1998*[105]

These applications are dependent on one another and will be elaborated upon further. Economic analysis can utilise the attributable fractions calculated in Table 6 in valuing the health impacts of air pollution. Policymakers often need to assess relative importance of air pollution in setting priorities for health programs. Estimating the costs of ill health, suffering, and premature death is a highly debated, controversial subject. The valuation of health impacts is usually done using 'willingness to pay' or 'cost of illness' approaches. Both these approaches need to make some heroic assumptions about value of lost life, or lost workdays, pain, and suffering. The task is made more difficult by non-availability of local epidemiologic studies when additional assumptions about dose-response functions need to be made. Yet, policymakers cannot wait for more research and decisions need to be

made based on best available information, appropriately adapted to the local setting as these presented in this thesis.

The results are of use to environmental equity and advocacy. It is actually incomprehensible that for so long, the health impacts of indoor air pollution have not been communicated to the general public. If they know about these health effects, they might better implement the technical and socio-behavioural interventions available. Technical interventions might be to reduce electricity costs for the poor in order to use electrical heaters instead of dirty fuels, to improve the ventilation rate in the homes or proper housing may also reduce dirty fuel use, such as built to enhance sunshine during winter time, tight houses with chimneys. Socio-behavioural interventions may include preventing children and pregnant women from being exposed to smoke from the dirty fuels. However, many factors influence the perception of risk. The reader is referred to Baker et al for an introduction on these as a thorough discussion is not within the reach of this thesis.[102] Chapter 2 addressed published results from interventions applied in the country. However, it is proposed that advocacy be encouraged in South Africa and the appropriate communication media used in awareness raising by the Health Promotion Department of the Department of Health. 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.[104] More than 8 million South Africans may thus not be able to benefit from health promotion material that is designed for the more educated population. This should be considered when developing communication messages. The multi-cultural dimension of South African society furthermore represents a particular challenge to develop culturally appropriate interventions. The field of environmental health promotion should be nurtured in this country, to address the multidisciplinary actions needed to implement viable interventions.

Furthermore, Kauchali et al conducted a local rural study on maternal perceptions of ARI in children under 5 years of age.[42] The respiratory set of signs and symptoms were classifiable into five causative categories: supernatural, natural, tuberculosis, cold weather and unknown. They indicated that perceptions of causation differed greatly from biomedical concepts. For illnesses of perceived supernatural causation, mothers were reluctant to seek medical care and antibiotics were deemed

inappropriate. Traditional remedies were preferred instead. These results have important implications in treating ARI (such as pneumonia), which is one of the top 3 leading causes of child mortality (12-48 months) in the country.[105] It is imperative that these issues be addressed in health promotion strategies.

The results can be applied in the evaluation of intervention and policies. Chapter 2 reported on intervention strategies that had already been implemented in the country and recommendations were made regarding future intervention strategies. Results from future SADHS may be used in trend analysis, during which time dirty fuel use will hopefully have been reduced by interventions and policies.

8.5 Other Sources of Data, Collaboration and Capacity Building

Given the advantages and disadvantages of using 1998 SADHS data in air pollution epidemiological analyses it is imperative that the country does not just rely on secondary data. For local public health to be improved by local air pollution epidemiological studies, other sources of data, collaboration and capacity building need to be investigated and enhanced.

Local researchers Sitas et al addressed the scarcity of health and risk factor data in the country more than 10 years ago.[107] Back then and currently still the primary routine system for morbidity data collection in South Africa is the register of notifiable diseases in which a number of conditions are notifiable by law. However, the information from such passive disease surveillance systems is assumed to be a reasonable measure of the incidence of a health outcome. This may be erroneous and Sitas et al addressed a number of reasons, such as ability for patient to attend a health facility, accessibility of health facilities, geographical differences in diagnostic standards, problem of linking a case to its appropriate catchment population with unique risk factor profiles.[107] In order to address these limitations, they suggested that active disease monitoring be introduced in a number of surveillance sites where the population has been properly enumerated in order to improve the usefulness of vital statistical information.

The need for a health data management system is covered in one of the indicators of the Department of Health Goals 2004 document and is also covered by its Ten Point Plan.[108,109] The reader is also directed to the Department of Health document

on Essential National Health Research (ENHR) published in 2001.[110] The ENHR was introduced in Chapter I of this thesis. Several challenges remain in implementing the ENHR strategies, such as institutional roles and responsibilities, coordination of key components of ENHR among partners in health improvement, patterns of advocacy, capacity development and sources and levels of funding. In a recent series of editorials in the *Scandinavian Journal of Public Health* the value of an effective surveillance system for particularly poor countries has been discussed.[110-113]

Although cross-sectional studies cannot prove causation, they introduce hypotheses that could be tested in analytical intervention studies. This touches upon the issue of improved collaboration with the Department Environmental Affairs and Tourism, Department of Minerals and Energy, Department of Health, Medical Research Council, CSIR and tertiary academic institutions. The MRC report on poverty and chronic diseases in South Africa highlighted that in situations where data exists (such as records of medical aid societies of selected hospitals) they are seldom collated and presented in a format that could help inform the overall morbidity patterns. Research collaborations with private medical aid companies and the health services should thus start addressing this deficiency. This need for collaboration is to some extent addressed in the Ten Point Plan of the Department of Health, that is coordinating, supporting and conducting research and monitoring and evaluation activities.[109] The Ten Point Plan is a strategic plan for delivery of the Department of Health's goals in the next five years and recognises the importance of ENHR and partnership within the research community for achieving national health objectives. Thus far the institutional mechanisms have been strengthened by the constitution of a functional and widely representative ENHR committee with a wide range of representation. Sustainability and expansion of ENHR activities are guaranteed by the continued support from the Department of Health, Department of Arts, Culture, Science and Technology, Department of Education and other government, international and private sector organisations. However, several challenges remain and these include institutional roles and responsibilities, coordination of key components of ENHR among partners in health development, patterns of advocacy, capacity development and sources and levels of funding. Multi-sectoral collaboration is absolutely essential in order to benefit as much as possible from secondary data usage, such as conducting epidemiological studies whilst

implementing interventions, such as the Basa njengo Magogo project by the Department of Minerals and Energy in Orangefarm, Johannesburg during the winter of 2003.[114] The Basa njengo Magogo of lighting a coal fire was introduced in Chapter 2.

The report by the WHO on indoor air pollution research needs stressed the importance of collaborative action and highlighted that multi-sectoral collaboration is not usually straightforward and often requires active development and support.[60] The report addressed three issues in multi-sectoral collaboration: a) The role of each sector needs to be more clearly defined in order to avoid duplication and confusion about responsibilities, b) multi-sectoral action requires good co-ordination, a function that will need to be put in place and c) collaborative action is often difficult for partners in practice: typically this requires an institutional or programme focus, leadership and adequate time for partners to learn how best to work together.

Local researchers Parry et al emphasised local, regional and international strategies to strengthen capacity building in health research.[115,116] This thesis specifically wants to stress the importance of developing the country's capacity in technical and research in *environmental* epidemiology, which ultimately attempts to improve public health. A country's ability to develop more sustainably depends on the capacity of its people and institutions to understand complex environmental and development issues in order to make the right development choices. The Foresight Series by the South African Department of Arts, Culture, Science and Technology pointed out that there is a general lack of highly trained experts in environmental epidemiology and health.[117] The latter is addressed in one of the indicators of the Department of Health Goals 2004 document, namely the transformation of Schools of Public Health.[105] However, capacity building can also be fostered by including postgraduate students in multi-sectoral collaboration projects. Furthermore, as addressed previously (Chapter 1, Section 1.2) the new National Health Act (Act 61 of 2003) may offer relief to the lack of political commitment to environmental health and improve the dilapidated function (including air quality measurements) in South Africa.[118] Regrettably environmental health is not presently a main concern in municipality budgets. This in turn will stress quality assurance when collecting

environmental data and the application of the data in epidemiological analyses, which in turn can inform policy and boost public health.

As mentioned in Chapter 1, Ezzati et al pointed out that whilst conducting intervention and/or epidemiological studies, it does not help to just zoom in 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.[119] Epidemiological studies will then only focus on causation and miss its goal to introduce interventions to eliminate or reduce risk factors.

The use of biomarkers in local indoor air pollution epidemiology studies is also a debatable topic that warrants attention. Because of the difficulty of obtaining accurate and unbiased exposure information from study participants and the difficulty of estimating the doses that such exposures might produce, there has been great interest in the development of biologic markers (biomarkers).[76] These may be defined as "cellular, biochemical, or molecular alterations that are measurable in biological media, such as human tissue, cells, or fluids".[76] Biomarkers allow for considerable improvement in measurement of dose if used properly. Biomarkers have several advantages: (1) may prevent the errors arising from participants' lack of knowledge, memory failure, biased recall or deliberate misinformation; (2) prevent measurement errors arising due to individual pharmacokinetic and pharmacodynamic variations; (3) some markers can be used to detect biological interactions between the exposure of interest and critical tissues; (4) provide a quantitative or estimate of dose; (5) can serve as the gold standard for other information sources and (6) can provide a basis for error allowance procedures in studies that rely on less accurate exposure measures due to the cost of the marker.

To be valuable in environmental epidemiology studies, a biomarker should obviously be better than recall data or environmental measures; should permit differentiation between exposure levels; should be applicable on a large scale or should at least be acceptable to participants in a validation substudy.[76] Before markers are used in epidemiologic research, their sensitivity and specificity should be known from both the laboratory and epidemiologic perspectives; reproducibility of results within and between laboratories must also be known; and, very importantly,

the particular time frame they reflect and during which they can be measured *in vivo* must be established so that they provide interpretable data regarding time and dose.[76]

At present, few exposure biomarkers satisfy these requirements. Some markers may provide a record of cumulative exposure (such as bone lead measurement), but most can assess only relatively recent exposures. Studies of biomarkers that use a case-control design and a cross-sectional marker of exposure can be difficult to interpret because of ambiguity about the temporal sequence of the marker and the disease. Indeed, such studies can be misleading. In addition to such problems in interpretation, biological measurements are often costly to perform. Furthermore, the need to obtain specimens can reduce the cooperation of participants and introduce the potential for selection bias to occur through initial refusal or later attrition, although these problems are probably not insurmountable if they are anticipated and addressed. Thus it is recommended to first pay attention to personal, indoor and outdoor air pollution measurements as well as distal and proximal causes of disease before applying biomarkers in local indoor air pollution epidemiology studies.

8.6 Main Conclusions and Recommendations

This thesis attempted to investigate the usefulness of analysing secondary South African air pollution exposure and health data to project risk estimates for adult respiratory health (older than 15 years), under five ARI and 1-59 month mortality due to exposure to indoor air pollution from using dirty fuels for cooking and heating purposes and ultimately improve public health in the country.

Despite the limitations of using the 1998 SADHS data, the results indicated that there is a strong case for acknowledging the large public health risk arising from air pollution exposure in South Africa. The findings can be utilised for economic analysis of exposure, environmental equity and evaluation of intervention and policies. The results cannot be used for standard or guideline setting as no quantitative exposure data have been collected nor was the study design appropriate.

Comparing the limitations identified in using the 1998 SADHS data to those from other local air pollution epidemiological studies, it is evident that not much progress

has been made in air pollution epidemiology in the country during this analysis. However, due to the large sample size and many risk factors identified in the 1998 SADHS, it had the advantage over previous local studies in that it attempted to deal more adequately with confounding factors. Future SADHS surveys at regular intervals will allow the South African health care providers to assess if any progress has been made to relieve indoor air pollution exposure and if such progress has influenced health patterns in the country.

In order to improve the use of future SADHS data in air pollution epidemiological analyses it is important to capture more indoor air pollution exposure indicators and identified confounders. However, given the advantages and disadvantages of using 1998 SADHS data in air pollution epidemiological analyses it is imperative that the country does not just rely on secondary data. For local public health to be improved by local air pollution epidemiological studies, other sources of data, collaboration and capacity building need to be investigated and enhanced. Air pollution indicators can also be included in other national surveys or smaller surveys conducted in typical South African settings. An indoor air pollution exposure questionnaire could also be applied at demographic surveillance sites through out the country.

It is recommended that the country must develop environmental public health tracking networks, which incorporates various data sources from multi-sectoral collaborative intervention projects with an analytic study design, in all major cities in the country. The studies may address any of the following health outcomes: injuries, TB, cancer, respiratory infections or perinatal conditions. Ideally the studies 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 local exposure models can be used and validated in future studies. Interventions that may be addressed include technical or socio-behavioural ones, like the Basa njengo Magogo method, preventing vulnerable groups (such as children and pregnant women) from being exposed to smoke from the dirty fuels and opening windows and/doors when cooking. More research is needed in establishing socio-behavioural interventions when using dirty fuels for lighting and space heating. Future studies need to be planned well in advance. Postgraduate students should participate in these studies to strengthen capacity in air pollution epidemiology in the country. Future studies may also link air quality data in the City of Cape Town to school absenteeism as a proxy

for child health until a health management system has been established for the country. It is recommended to first pay attention to personal, indoor and outdoor air pollution measurements as well as distal and proximal causes of disease before applying biomarkers in local indoor air pollution epidemiology studies. The use of Geographical Information Systems is also encouraged in linking exposure to health outcomes.

The air quality data in the City of Cape Town is quite useful for a number of reasons, despite the limitations identified. It is furthermore recommended that similar inter-site correlation analyses be conducted using air quality data from the other major cities in the country that maintain relatively extensive air quality monitoring networks under rigorous quality assurance guidelines. It is also recommended that all air quality monitoring networks in the country should be accredited in the near future by the South African National Accreditation System (SANAS). Given the lack of resources for outdoor air pollution measurement and the dearth of air pollution epidemiological studies in the country, local cities should thus attempt to optimise and update their air quality monitoring networks with the assistance of research and academic institutions in such a manner as to serve both compliance monitoring and epidemiological exposure assessment

The need for a central location for data storage of all exposure, demographic and health data is crucial in South Africa. The collection of high quality outdoor air pollution data in all major cities in the country is needed, together with the implementation of an electronic health data management system as well as electronic birth and cancer registries as soon as possible.

There is a need to add health policy courses to the health sciences curriculum in order to improve communication between epidemiological research and policy in the country.

The multi-cultural dimension of South African society furthermore represents a particular challenge to develop culturally appropriate interventions. The field of environmental health promotion should be nurtured in this country, to address the multidisciplinary actions needed to implement viable interventions.

8.7 References

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Appendix I

1998 SADHS HOUSEHOLD QUESTIONNAIRE

19/1/98

SOUTH AFRICAN DEMOGRAPHIC AND HEALTH SURVEY
HOUSEHOLD SCHEDULE

IDENTIFICATION

PROVINCE _____

DISTRICT
_____

EA NUMBER .....

EA TYPE .....

SADHS CLUSTER NUMBER .....

HOUSEHOLD NUMBER .....

NAME OF HOUSEHOLD HEAD _____

IS HOUSEHOLD SELECTED FOR ADULT HEALTH 1 = YES 2 = NO

INTERVIEWER VISITS

	1	2	3	FINAL VISIT
DATE	_____	_____	_____	DAY
PHONE NUMBER _____				MONTH YEAR
INTERVIEWER'S NAME	_____	_____	_____	NAME
RESULT*	_____	_____	_____	RESULT
NEXT VISIT: DATE	_____	_____		TOTAL NO. OF VISITS
TIME	_____	_____		

*RESULT CODES:

1 COMPLETED
2 NO HOUSEHOLD MEMBER AT HOME OR NO COMPETENT RESPONDENT AT HOME AT TIME OF VISIT
3 ENTIRE HOUSEHOLD ABSENT FOR EXTENDED PERIOD OF TIME
4 POSTPONED
5 REFUSED
6 DWELLING VACANT OR ADDRESS NOT A DWELLING
7 DWELLING DESTROYED
8 DWELLING NOT FOUND
9 OTHER _____
(SPECIFY)

TOTAL IN HOUSEHOLD

TOTAL ADULTS 15 YEARS AND OVER

TOTAL WOMEN 15-49 YEARS

LINE NO. OF RESP. TO HOUSEHOLD SCHEDULE

SUPERVISOR	FIELD EDITOR	OFFICE EDITOR	KEYED BY
NAME_____	NAME _____		
DATE _____	DATE _____		

H-ENG1

HOUSEHOLD SCHEDULE

* CODES FOR Q.3

RELATIONSHIP TO HEAD OF HOUSEHOLD:
01 = HEAD
02 = WIFE/HUSBAND/PARTNER
03 = SON OR DAUGHTER
04 = SON-IN-LAW OR DAUGHTER-IN-LAW
05 = GRANDCHILD
06 = PARENT
07 = PARENT-IN-LAW
08 = BROTHER OR SISTER
09 =NIECE/NEPHEW
10 =OTHER RELATIVE
11 = ADOPTED/FOSTER/STEP CHILD
12 = NOT RELATED
98 = DON'T KNOW

** CODES FOR Q.10 (EDUCATION GRADE)
00 = LESS THAN 1 YEAR COMPLETED
71 = SUB A/CLASS 1
72 = SUB B/CLASS 2
01 = STANDARD 1
02 = STANDARD 2
03 = STANDARD 3
04 = STANDARD 4
05 = STANDARD 5
06 = STANDARD 6
07 = STANDARD 7
08 = STANDARD 8
09 = STANDARD 9
10 = STANDARD 10
11 = FURTHER STUDIES INCOMPLETE
12 = DIPLOMA/OTHER POSTSCHOOL COMPLETE
13 = FURTHER DEGREE COMPLETE
98 = DON'T KNOW

*** CODES FOR Q.14

11 = ASSAULT IN HOME
12 = POLITICAL VIOLENCE
13 = OTHER ASSAULT OUTSIDE OF HOME
14 = SELF INFLICTED VIOLENCE
21 = TRAFFIC COLLISION
22 = ACCIDENT AT WORK
23 = SPORT
96 = OTHER UNINTENTIONAL

*** Q.15 THROUGH Q.18:

These questions refer to the bidogical parents of the child. Record 00 if parent not member of household.

Now we would like some information about the people who usually live in your household or who are staying with you now.

LINE NO.	USUAL RESIDENTS AND VISITORS	RELATIONSHIP TO HEAD OF HOUSEHOLD*	RESIDENCE		SEX	AGE	GRANTS/ PENSION	EDUCATION			WORK	INJURIES IN THE LAST MONTH		PARENTAL SURVIVORSHIP AND RESIDENCE FOR PERSONS LESS THAN 15 YEARS OLD***				ELIGIBILITY	
									IF ATTENDED SCHOOL		AGE 10+			IF ALIVE		IF ALIVE		CIRCLE LINE NUMBER OF	
	Please give me the names of the persons who usually live in your household and guests of the household who stayed here last night, starting with the head of the household.	What is the relationship of (NAME) to the head of the household?	Does (NAME) usually live here?	Did (NAME) stay here last night?	Is (NAME) male or female?	How old is (NAME)? (WRITE 00 IF UNDER 1 YEAR). IF 95 OR OVER, WRITE '95'	Does (NAME) receive a child maintenance grant , a disability grant or a pension from the government?	Has (NAME) ever been to school?	What is the highest level of school (NAME) completed?**	IF AGE LESS THAN 25 YEARS: Is (NAME) still in school?	Did (NAME) work for pay during the last 7 days?	Did (NAME) have any injury that was treated by a doctor or nurse during the last 30 days?	IF INJURED IN LAST 1 MONTH: What type of injury*** did (NAME) have?	Is (NAME)'s natural mother alive?	Does (NAME)'s natural mother live in this household? IF YES: What is her name? RECORD MOTHER'S LINE NUMBER	Is (NAME)'s natural father alive?	Does (NAME)'s natural father live in this household? IF YES: What is his name? RECORD FATHER'S LINE NUMBER	ALL PERSONS 15 YEARS OR OLDER	ALL WOMEN AGE 15-49
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)
01			YES NO 1 2	YES NO 1 2	M F 1 2	IN YEARS	YES NO DK 1 2 8	YES NO 1 2		YES NO 1 2	YES NO 1 2	YES NO DK 1 2 8 GO TO (15)		YES NO DK 1 2 8		YES NO DK 1 2 8		01	01
02			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		02	02
03			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		03	03

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)
04			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		04	04
05			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		05	05
06			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		06	06
07			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		07	07
08			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		08	08
09			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		09	09
10			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		10	10
11			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		11	11
12			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		12	12
13			1 2	1 2	1 2		1 2 8	1 2		1 2	1 2	1 2 8 GO TO (15)		1 2 8		1 2 8		13	13

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)

TICK HERE IF CONTINUATION SHEET USED ☐

Just to make sure that I have a complete listing:

1)	Are there any other persons such as small children or infants that we have not listed?	YES ☐	E
2)	In addition, are there any other people who may not be members of your family, such as domestic workers, lodgers or friends who usually live here?	YES ☐	E
3)	Are there any guests or temporary visitors staying here, or anyone else who slept here last night that have not been listed?	YES ☐	E

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
21	Has anyone in the household died in the last 12 months?	YES ..... 1 NO 2	→25
22	In the last 12 months, how many people in your household died?	NUMBER OF PERSONS ..... ☐☐	
23	In the last 12 months, how many people in your household died from an injury sustained as a result of violence either between them and other people or from violence inflicted upon themselves?	NUMBER OF PERSONS ..... ☐☐	
24	In the last 12 months, how many persons in your household died from an unintentional injury they sustained such as from a traffic collision, or an injury (such as falls, burns or cuts) that happended at home/work/school/etc?	NUMBER OF PERSONS ..... ☐☐	
25	What is the main source of drinking water for members of your household?	PIPED WATER (TAP) IN DWELLING ..... 11 PIPED WATER (TAP) IN SITE/YARD ..... 12 PUBLIC TAP ..... 13 WATER CARRIER/TANKER ..... 21 BOREHOLE/WELL ..... 31 DAM/RIVER/STREAM/SPRING ..... 32 RAIN-WATER TANK ..... 41 BOTTLED WATER ..... 51 OTHER _____ 96	→28 →28 →28
26	How long does it take you to get there, get water, and come back?	MINUTES ..... ☐☐☐ ON PREMISES 996	
27	Who fetched the water yesterday? RECORD ALL MENTIONED.	FEMALE ADULT ..... A MALE ADULT ..... B FEMALE CHILD ..... C MALE CHILD ..... D DON'T KNOW ..... Z	
28	What kind of toilet facility does your household have?	FLUSH TOILET (OWN) ..... 11 FLUSH TOILET (SHARED) ..... 12 BUCKET LATRINE ..... 21 PIT LATRINE ..... 22 NO FACILITY/BUSH/FIELD ..... 31 OTHER _____ 96	
29	Does your household have: Electricity? A radio? A television? A telephone? A refrigerator? A personal computer (PC)? A washing machine?	YES NO ELECTRICITY ..... 1 2 RADIO ..... 1 2 TELEVISION ..... 1 2 TELEPHONE ..... 1 2 REFRIGERATOR ..... 1 2 PERSONAL COMPUTER ..... 1 2 WASHING MACHINE 1 2	
30	What does your household use for cooking and heating? RECORD ALL MENTIONED.	ELECTRICITY ..... A GAS ..... B PARAFFIN ..... C WOOD ..... D COAL ..... E ANIMAL DUNG ..... F OTHER _____ X	
31	How many rooms in your household are used for sleeping?	ROOMS ..... ☐☐	

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
32	MAIN MATERIAL OF THE FLOOR. RECORD OBSERVATION	EARTH/SAND/DUNG ..... 11 BARE WOOD PLANKS ..... 21 CEMENT ..... 31 VINYL ..... 32 CARPET ..... 33 CERAMIC TILES ..... 34 PARQUET OR POLISHED WOOD ..... 35 OTHER 96	
33	MAIN MATERIAL IN THE WALLS. RECORD OBSERVATION	PLASTIC/CARDBOARD ..... 11 MUD ..... 12 MUD AND CEMENT ..... 13 CORRUGATED IRON/ZINC ..... 21 PREFAB ..... 22 BARE BRICK/CEMENT BLOCK ..... 23 PLASTER/FINISHED ..... 31 OTHER 96	
34	Let us speak about the household and what it can afford. Would you say that the people here often, sometimes, seldom or never go hungry?	OFTEN ..... 1 SOMETIMES ..... 2 SELDOM ..... 3 NEVER 4	
35	Does any member of your household own: A bicycle? A motorcycle? A car? A donkey or a horse? Sheep or cattle?	YES NO BICYCLE ..... 1 2 MOTORCYCLE ..... 1 2 CAR ..... 1 2 DONKEY/HORSE ..... 1 2 SHEEP/CATTLE 1 2	

Appendix 2

1998 SADHS ADULT HEALTH QUESTIONNAIRE

SOUTH AFRICAN DEMOGRAPHIC AND HEALTH SURVEY
ADULT HEALTH QUESTIONNAIRE

12/1/98

IDENTIFICATION

PROVINCE _____

DISTRICT
_____

EA NUMBER .....

EA TYPE .....

SADHS CLUSTER NUMBER .....

HOUSEHOLD NUMBER .....

NAME AND LINE NUMBER OF ADULT _____

NAME OF HOUSEHOLD HEAD _____

INTERVIEWER VISITS

	1	2	3	FINAL VISIT
DATE				DAY / MONTH / YEAR
INTERVIEWER'S NAME				NAME
RESULT*				RESULT
NEXT VISIT: DATE				TOTAL NO. OF VISITS
TIME				

*RESULT CODES:
1 COMPLETED
2 NOT AT HOME
3 POSTPONED
4 REFUSED
5 PARTLY COMPLETED
6 INCAPACITATED
7 OTHER _____ (SPECIFY)

LANGUAGE

LANGUAGE OF QUESTIONNAIRE .....

LANGUAGE OF INTERVIEW .....

HOME LANGUAGE OF RESPONDENT .....

TRANSLATOR USED (YES = 1, NO = 2) .....

LANGUAGE CODES

01 ENGLISH	04 isi ZULU	07 SePEDI	10 ZITSONGA
02 AFRIKAANS	05 SeSOTHO	08 SISWATI	11 isiNDEBELA
03 isiXHOSA	06 SeTSWANA	09 TshiVENDA	

SUPERVISOR	FIELD EDITOR	OFFICE EDITOR	KEYED BY
NAME _____	NAME _____		
DATE _____	DATE _____		

SECTION 1: HEALTH SERVICE UTILIZATION

NO.	QUESTIONS AND FILTERS					
1.	During the last month have you been to any of the following health services for medical care for yourself : PROBE			2. Were you satisfied with the care you received at (PLACE)?		3. Why were you not satisfied with the care you received at (PLACE)?
A	Day Hospital?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	
B	Government Hospital/Government Clinic?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	
C	Private Hospital/Private Clinic?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	
D	District Surgeon?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	
E	Private Doctor?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	
F	Chemist Shop?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	
G	Faith Healer?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	
H	Traditional Healer or Herbalist?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	
I	Health Services at the Workplace?	YES	NO	YES	NO	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
		1	2	1	2	

J	Home Based Care Services/House visits?	YES 1 ▪ NO 2 ┐	YES 1 ┐ ▪ NO 2	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
K	Dentist/Oral hygienist/Oral therapist?	YES 1 ▪ NO 2 ┐	YES 1 ┐ ▪ NO 2	LONG WAIT ..... 01 SHORT CONSULTATION ..... 02 STAFF RUDE/UNKIND ..... 03 DIDN'T SEE DOCTOR ..... 04 OTHER_____ 96 (SPECIFY)
L	Other? _____ SPECIFY	YES 1 NO 2		

4.	Are you covered by a Medical Aid or Medical Benefit Scheme? (Any scheme that helps you pay for health/drug services)	YES ..... 1 NO 2	
5.	Have you had your blood pressure measured in the past 12 months?	YES ..... 1 NO 2	
6.	Do you know what your blood pressure is?	YES ..... 1 NO 2	→ 8
7.	Is it high, normal or low?	HIGH ..... 1 NORMAL ..... 2 LOW ..... 3 DON'T KNOW 8	

SECTION 2: FAMILY MEDICAL HISTORY

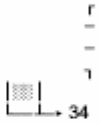
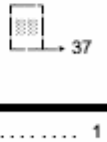
8	Now I would like to ask you about your family. Do you have a close blood relative (father, mother, brother, sister or child) who has ever had any of the following conditions:		
8A	High Blood Pressure?	YES ..... 1 NO ..... 2 DON'T KNOW 8	
8B	Heart attack or angina or chest pain when exerting himself/herself?	YES ..... 1 NO ..... 2 DON'T KNOW 8	┐→ 8D
8C	IF "YES", was it before the age of 50 years?	YES ..... 1 NO ..... 2 DON'T KNOW 8	
8D	Stroke?	YES ..... 1 NO ..... 2 DON'T KNOW 8	
8E	High blood cholesterol or Fats?	YES ..... 1 NO ..... 2 DON'T KNOW 8	
8F	Diabetes or Blood Sugar?	YES ..... 1 NO ..... 2 DON'T KNOW 8	
8G	Cancer?	YES ..... 1 NO ..... 2 DON'T KNOW 8	

SECTION 3: CLINICAL CONDITIONS

9	Now I would like to ask you about your own health. Has a doctor or nurse or staff member at a clinic or at hospital told you that you had or have any of the following conditions:		
9A	High Blood Pressure?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 9C
9B	IF "YES", when was the first time that you were told you had high blood pressure?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	
9C	Heart attack or angina?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 9E
9D	IF "YES", when was your heart attack or angina?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	
9E	Stroke?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 9G
9F	IF "YES", when did you have your stroke?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	
9G	High blood cholesterol or fats?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 9I
9H	IF "YES", when was the first time that you were told that you had blood cholesterol or fats?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	
9I	Diabetes or Blood Sugar?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 9K
9J	IF "YES", when was the first time that you were told that you had diabetes or blood sugar?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	
9K	Emphysema/Bronchitis?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 9M
9L	IF "YES", when was the first time that you were told that you had emphysema or bronchitis?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	
9M	Asthma?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 9O
9N	IF "YES" when was the first time that you were told that you had asthma?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	
9O	TB?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 9Q
9P	IF "YES" when was the first time that you were told that you had TB?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	
9PP	How many episodes of TB have you ever been treated for?		
9Q	Cancer?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 12
9R	IF "YES", when was the first time that you were told that you had cancer?	IN THE LAST 12 MONTHS . . . . . 1 MORE THAN A YEAR AGO 2	

10	Did the doctor/nurse/staff member at a hospital tell you what kind of cancer you have?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 12
11	What kind of cancer were you told you had or have? DO NOT READ THE LIST OF CANCERS.	LUNG CANCER . . . . . A CERVICAL/WOMB CANCER . . . . . B SKIN CANCER . . . . . C BREAST CANCER . . . . . D PROSTATE CANCER . . . . . E ESOPHAGEAL CANCER . . . . . F OTHER_____ X (SPECIFY)	
12	Do you feel you have less breath when exerting yourself when compared to other people your age?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	
13	During the last year have you had wheezing or tightness of your chest.	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 16
14	If "YES" were you also short of breath?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	
15	Do you only get wheezing when you have a cold?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	
16	Is your sleep ever interrupted by you coughing?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	
17	Is your sleep ever interrupted by wheezing or a tight chest?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	
18	Do you usually cough?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 21
19	When you cough, do you usually bring up phlegm from your chest?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 21
20	If "yes", have you brought up phlegm every day for at least three months during the last year?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→ 21
20A	If "yes" for how many years have you brought up phlegm in this way?		
21	IS THE RESPONDENT A MAN OR A WOMAN?	MAN ▼ WOMAN	→ 26
22	Now I am going to ask you some personal questions. Please remember that this information will be kept strictly confidential. Some men experience pain during urination or have a discharge from the penis. During the last 3 months, have you noticed any such pain or discharge?	YES . . . . . 1 NO 2	
24	Some men experience sores in the genital area. During the last 3 months, have you noticed any such sores?	YES . . . . . 1 NO 2	

SECTION 4: DENTAL HEALTH

26	Now I want to ask you about your teeth. Do you think that there is anything wrong in your mouth, teeth or gums?	YES ..... 1 NO 2	→2 8
27	Which of the following items do you feel is a problem: Your Teeth? Your Gums? Ulcers/sores in the mouth? Dentures? Any other problems? RECORD ALL MENTIONED.	 TEETH ..... A GUMS ..... B ULCERS/SORES IN THE MOUTH ... C DENTURES ..... D OTHER _____ X (SPECIFY)	
28	Have you ever visited a dentist, an oral hygienist, or an oral therapist ?	YES ..... 1 NO 2	
29	Have you lost any of your natural teeth?	YES ..... 1 NO 2	→34
30	Do you have any of your natural teeth?	YES ..... 1 NO 2	
31	Do you wear a denture (false teeth)?	YES, PARTIAL ..... 1 YES, TOTAL/COMPLETE ..... 2 NO 3	
32	CHECK 30: HAS NO NATURAL TEETH 	HAS NATURAL TEETH 	
33	Do you usually rinse or clean your mouth everyday?	YES ..... 1 NO 2	→ 38
34	What do you do to look after your teeth. Do you Clean/Brush your teeth? Watch your diet/Eat special foods? Visit the dentist? Anything else?	YES NO CLEAN/BRUSH ..... 1 2 DIET/FOOD ..... 1 2 VISIT DENTIST ..... 1 2 OTHER_____1 2 (SPECIFY)	
35	CHECK 34: CLEAN/BRUSH 	DOES NOT CLEAN/BRUSH 	
36	Do you usually brush/wash your teeth everyday?	YES ..... 1 NO 2	
37	Do you own a toothbrush?	YES ..... 1 NO 2	

38	Some people say that fluoride mineral in the water makes the children and adults' natural teeth strong and healthy; Other people say it does not. What do you think?	MAKES TEETH STRONG . . . . . 1 DOES NOT MAKE TEETH STRONG . 2 OTHER_____ 6 (SPECIFY) DON'T KNOW 8	

SECTION 5: OCCUPATIONAL HEALTH

39	In the last 12 months, have you worked for payment?	YES . . . . . 1 NO 2	→45A
40	In the last 12 months, have you had any injury or health problem related to your work?	YES . . . . . 1 NO 2	→43
41	Did you stay away from work because of this injury or problem?	YES . . . . . 1 NO 2	
42	What was the injury or health problem?	___ ___ ___ _____ _____ _____	
43	In the last 12 months, have you had an existing injury or health problem that was aggravated or became worse at work?	YES . . . . . 1 NO 2	→45A
44	Did you stay away from work because of this injury or problem?	YES . . . . . 1 NO 2	
45	What was the injury or health problem?	___ ___ ___ _____ _____ _____	
45A	Have you ever worked underground in a mine?	YES . . . . . 1 NO 2	→46
45B	If "yes", what kind of mine was it? RECORD ALL	GOLD . . . . . A COAL . . . . . B ASBESTOS . . . . . C OTHER _____ X (SPECIFY)	
45C	How many years in total did you work underground?	___ ___	

SECTION 6: MEDICATION

46	Now I want to ask you about any medication you take. Do you use any medicine regularly that has been prescribed by a doctor or nurse?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→65
47	How many different medicines do you use regularly?	NUMBER	
48	Do you know what the medication is for?	YES . . . . . 1 NO 2	→65
49	Is it for **High Blood Pressure**?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→51
50	Can you name the medication? WRITE DOWN THE NAME(S) OF THE MEDICATION.	YES . . . . . 1 NO . . . . . 2 _____ _____ _____ _____ _____	→51
51	Is it for **Diabetes/Sugar**?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→53

52	Can you name the medication? WRITE DOWN THE NAME(S) OF THE MEDICATION.	YES . . . . . 1 NO . . . . . 2 ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘	⟶53
53	Is it for **High Blood Cholesterol**?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→55
54	Can you name the medication? WRITE DOWN THE NAME(S) OF THE MEDICATION.	YES . . . . . 1 NO . . . . . 2 ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘	⟶55
55	Is it for **Angina/chestpain**?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→57

56	Can you name the medication? WRITE DOWN THE NAME(S) OF THE MEDICATION.	YES . . . . . 1 NO . . . . . 2 ┌─┐ └─┘ ┌─┐ └─┘ ┌─┐ └─┘ ┌─┐ └─┘ ┌─┐ └─┘	→57
57	Is it for any other **Heart condition**?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→59
58	Can you name the medication? WRITE DOWN THE NAME(S) OF THE MEDICATION.	YES . . . . . 1 NO . . . . . 2 ┌─┐ └─┘ ┌─┐ └─┘ ┌─┐ └─┘ ┌─┐ └─┘ ┌─┐ └─┘	→59
59	Is it for **Asthma, Emphysema or Bronchitis**?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→61

60	Can you name the medication? WRITE DOWN THE NAME(S) OF THE MEDICATION.	YES . . . . . 1 NO . . . . . 2 ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘	→61
61	Is it for **Tuberculosis**?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→63
62	Can you name the medication? WRITE DOWN THE NAME(S) OF THE MEDICATION.	YES . . . . . 1 NO . . . . . 2 ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘ ┌─┐ _____ |▒▒| └─┘	→63
63	Do you take it because you had a **Stroke**?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→65

64	Can you name the medication? WRITE DOWN THE NAME(S) OF THE MEDICATION.	YES . . . . 1 NO . . . . 2 ____________________ ____________________ ____________________ ____________________ ____________________	→65

65	NOW, ASK THE RESPONDENT TO SHOW YOU ALL THE MEDICATION TAKEN EVERY DAY DURING THE LAST MONTH. THEN WRITE DOWN THE NAMES OF ALL THE MEDICATIONS BELOW. NAME _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐ _____ ☐☐☐☐☐☐☐	MEDICATIONS ARE LISTED YES.....1 NO.....2 →66 NUMBER OF MEDICATIONS LISTED ..... ☐☐
65A	Who pays for most of the prescribed medication that you use?	RESPONDENT ..... 01 FAMILY ..... 02 MEDICAL AID ..... 03 PROVIDED AT CLINIC OR PUBLIC HOSPITAL ..... 04 EMPLOYER ..... 05 OTHER _____ 96 (SPECIFY)

SECTION 7: HABITS AND LIFESTYLE

Now I would like to ask you a few questions about your diet and other habits.

66	How old were you at your last birthday?	AGE IN COMPLETED YEARS .... ☐☐	
66a	Which race group do you consider yourself?	BLACK/AFRICAN ..... 1 COLOURED ..... 2 WHITE ..... 3 ASIAN/INDIAN 4	
67	Do you usually eat your food very salty, lightly salted or not salted?	VERY SALTY ..... 1 LIGHTLY SALTED ..... 2 NOT SALTED ..... 3 DON'T KNOW 8	
68	Do you usually add salt or Aromat/Fondor to your serving of food? IF YES, Before or after tasting the food?	NO, I NEVER ADD SALT/AROMAT ..... 1 YES, BUT I TASTE FIRST AND THEN ADD . 2 YES, EVEN BEFORE HAVING TASTED FOOD 3 DON'T KNOW 8	
69	Do you eat salty snacks more often than three times per week (Such as chips, nliknaks, salted peanuts, salty biscuits, biltong, dried sausage, dried fish)?	YES ..... 1 NO 2	
70	Do you personally think that you are underweight,normal weight or overweight?	UNDERWEIGHT ..... 1 NORMAL WEIGHT ..... 2 OVERWEIGHT..... 3 DON'T KNOW 8	
71	Have you ever smoked tobacco, used snuff or chewed tobacco?	YES ..... 1 NO 2	——→81
72	Have you ever smoked at least 100 cigarettes (5 packets of 20 cigarettes) or the equivalent amount of tobacco in your lifetime?	YES ..... 1 NO 2	
73	Have you ever smoked daily?	YES ..... 1 NO 2	
74	On average, what number of the following items do or did you smoke or use per day? PROBE AND FILL IN NUMBER FOR EACH ITEM.	MANUFACTURED CIGARETTES ☐☐ HAND-ROLLED CIGARETTES ☐☐ PIPEFULS OF TOBACCO ☐☐ CIGARS/CHEROOTS/CIGARILLOS ☐☐ SNUFF ☐☐ CHEWING TOBACCO/PRUIMPIE ☐☐	
75	CHECK 74: EVER SMOKED CIGARETTES, PIPES OR CIGARS ☐	USES SNUFF OR CHEWING TOBACCO ☐ → 81	
76	How many years have you smoked or did you smoke on a daily basis? (IF RESPONDENT HAS STOPPED AND STARTED AGAIN, ASK FOR TOTAL YEARS)	NUMBER OF YEARS ..... ☐☐	

ADULT DEMOGRAPHIC AND HEALTH SURVEY
ANTHROPOMETRIC DATA SHEET

94	DATE	d d m m y y y y: 1 9
95	FIELDWORKER NUMBER	
96	WEIGHT (KG)	
97	HEIGHT (CM)	
98	MID-UPPER-ARM CIRCUMFERENCE (CM)	
99	WAIST CIRCUMFERENCE (CM)	
100	HIP CIRCUMFERENCE (CM)	
101	SYSTOLIC BLOOD PRESSURE 1	
102	DIASTOLIC BLOOD PRESSURE 1	
103	PULSE 1	
104	SYSTOLIC BLOOD PRESSURE 2	
105	DIASTOLIC BLOOD PRESSURE 2	
106	PULSE 2	
107	SYSTOLIC BLOOD PRESSURE 3	
108	DIASTOLIC BLOOD PRESSURE 3	
109	PULSE 3	
110	PEAK EXPIRATORY FLOW RATE	

Appendix 3

1998 SADHS WOMEN'S HEALTH QUESTIONNAIRE

SOUTH AFRICAN DEMOGRAPHIC AND HEALTH SURVEY
WOMEN QUESTIONNAIRE

15/1/98

IDENTIFICATION

PROVINCE _____

DISTRICT _____

EA NUMBER .....

EA TYPE .....

SADHS CLUSTER NUMBER .....

HOUSEHOLD NUMBER .....

NAME AND LINE NUMBER OF WOMAN _____

NAME OF HOUSEHOLD HEAD _____

INTERVIEWER VISITS

	1	2	3	FINAL VISIT
DATE				DAY / MONTH / YEAR
INTERVIEWER'S NAME				NAME
RESULT*				RESULT
NEXT VISIT: DATE				TOTAL NO. OF VISITS
TIME				

*RESULT CODES:
1 COMPLETED
2 NOT AT HOME
3 POSTPONED
4 REFUSED
5 PARTLY COMPLETED
6 INCAPACITATED
7 OTHER _____ (SPECIFY)

LANGUAGE

LANGUAGE OF QUESTIONNAIRE 0 1

LANGUAGE OF INTERVIEW .....

HOME LANGUAGE OF RESPONDENT .....

TRANSLATOR USED (YES = 1, NO = 2) .....

LANGUAGE CODES

01 ENGLISH	04 isi ZULU	07 SePEDI	10 ZITSONGA
02 AFRIKAANS	05 SeSOTHO	08 SiSWATI	11 isiNDEBELA
03 isiXHOSA	06 SeTSWANA	09 TshiVENDA	

SUPERVISOR	FIELD EDITOR	OFFICE EDITOR	KEYED BY
NAME _____	NAME _____		

SUPERVISOR	FIELD EDITOR	OFFICE EDITOR	KEYED BY
DATE _____	DATE _____		

SECTION 1. RESPONDENT'S BACKGROUND

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
101	RECORD THE TIME.	HOUR . . . . . [ ][ ] MINUTES . . . . . [ ][ ]	
102	First I would like to ask some questions about you and your household. For most of the time until you were 12 years old, did you live in a city, in a large town, on a farm or in rural areas?	CITY . . . . . 1 TOWN . . . . . 2 RURAL/FARM 3	
103	How long have you been living continuously in (NAME OF CURRENT PLACE OF RESIDENCE)? IF LESS THAN 1 YEAR, WRITE '00'	YEARS . . . . . [ ][ ] ALWAYS . . . . . 95 VISITOR 96	→105
104	Just before you moved here, did you live in a city, in a town, or in the rural area /farm?	CITY . . . . . 1 TOWN . . . . . 2 RURAL/FARM 3	
105	In what month and year were you born?	MONTH . . . . . [ ][ ] DON'T KNOW MONTH . . . . . 98 YEAR . . . . . [ ][ ][ ][ ] DON'T KNOW YEAR 9998	
106	How old were you at your last birthday? COMPARE AND CORRECT 105 AND/OR 106 IF INCONSISTENT.	AGE IN COMPLETED YEARS [ ][ ]	
107	Have you ever attended school?	YES . . . . . 1 NO 2	→114
109	What is the highest (standard/year) you completed ?	LESS THAN ONE YEAR COMPLETED 00 SUB A/CLASS 1 . . . . . 71 SUB B/CLASS 2 . . . . . 72 STANDARD 1 . . . . . 01 STANDARD 2 . . . . . 02 STANDARD 3 . . . . . 03 STANDARD 4 . . . . . 04 STANDARD 5 . . . . . 05 STANDARD 6 . . . . . 06 STANDARD 7 . . . . . 07 STANDARD 8 . . . . . 08 STANDARD 9 . . . . . 09 STANDARD 10 . . . . . 10 FURTHER STUDIES INCOMPLETE . 11 DIPLOMA/OTHER POSTSCHOOL COMPLETE . . . . . 12 FURTHER DEGREE COMPLETE . . 13	
110	CHECK 106: AGE 24 OR BELOW [ ] AGE 25 OR ABOVE [ ]		→114
111	Are you currently attending school?	YES . . . . . 1 NO 2	→114

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
112	What was the main reason you stopped attending school?	GOT PREGNANT ..... 01 GOT MARRIED ..... 02 TO CARE FOR YOUNGER CHILDREN ..... 03 FAMILY NEEDED HELP ON FARM OR IN BUSINESS ..... 04 COULD NOT PAY SCHOOL FEES .. 05 NEEDED TO EARN MONEY ..... 06 GRADUATED/HAD ENOUGH SCHOOLING ..... 07 DID NOT PASS ENTRANCE EXAMS 08 DID NOT LIKE SCHOOL ..... 09 SCHOOL NOT ACCESSIBLE/ TOO FAR ..... 10 OTHER _____ 96 (SPECIFY) DON'T KNOW 98	
114	Can you read and understand a letter or newspaper in your home language easily, with difficulty, or not at all?	EASILY ..... 1 WITH DIFFICULTY ..... 2 NOT AT ALL 3	→116
115	Have you read a newspaper or magazine in the last week?	YES ..... 1 NO 2	
116	Do you usually listen to a radio every day?	YES ..... 1 NO 2	
117	Do you usually watch television at least once a week?	YES ..... 1 NO 2	
119	Which race group do you consider yourself?	BLACK/AFRICAN ..... 1 COLOURED ..... 2 WHITE ..... 3 ASIAN/INDIAN 4	
120	CHECK Q.4 IN THE HOUSEHOLD QUESTIONNAIRE THE WOMAN INTERVIEWED IS NOT A USUAL RESIDENT ☐ ▼ THE WOMAN INTERVIEWED IS A USUAL RESIDENT ☐		→201
121	Now I would like to ask about the place in which you usually live. What is the name of the place in which you usually live? _____ (NAME OF PLACE) Is that a large city, town, or rural area/farm?	CITY ..... 1 TOWN ..... 2 RURAL/FARM 3	
122	In which PROVINCE is that located?	EASTERN CAPE ..... 01 FREE STATE ..... 02 GAUTENG ..... 03 KWAZULU/NATAL ..... 04 MPUMALANGA ..... 05 NORTHERN CAPE ..... 06 NORTHERN PROVINCE ..... 07 NORTH WEST ..... 08 WESTERN CAPE ..... 09 OTHER COUNTRY 10	
123	Now I would like to ask about the household in which you usually live. What is the main source of drinking water for members of your household?	PIPED WATER (tap), IN DWELLING 11 PIPED WATER (tap), IN SITE/YARD 12 PUBLIC TAP ..... 13 WATER CARRIER/ TANKER ..... 21 BOREHOLE/WELL ..... 31 DAM /RIVER/STREAM/SPRING .... 32 RAIN-WATER TANK ..... 41 BOTTLED WATER ..... 51 OTHER _____ 96 (SPECIFY)	
125	What kind of toilet facility does your household have?	FLUSH TOILET (OWN) ..... 11 FLUSH TOILET (SHARED) ..... 12 BUCKET LATRINE ..... 21 PIT LATRINE ..... 22 NO FACILITY/BUSH/FIELD ..... 31 OTHER _____ 96 (SPECIFY)	

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
126	Does your household have: Electricity? A radio? A television? A telephone? A refrigerator? A personal computer (PC)? A washing machine?	YES NO ELECTRICITY . . . . . 1 2 RADIO . . . . . 1 2 TELEVISION . . . . . 1 2 TELEPHONE . . . . . 1 2 REFRIGERATOR . . . . . 1 2 PERSONAL COMPUTER . . . 1 2 WASHING MACHINE 1 2	
127	Could you describe the main material of the walls of your home?	PLASTIC/CARDBOARD . . . . . 11 MUD . . . . . 12 MUD AND CEMENT . . . . . 13 CORRUGATED IRON/ZINC . . . . . 21 PREFAB . . . . . 22 BARE BRICK/CEMENT BLOCK . . . . 23 PLASTER/FINISHED . . . . . 31 OTHER _____ 96 (SPECIFY)	

SECTION 2. REPRODUCTION

Now I would like to ask you about all the pregnancies that you have had in your lifetime. By this I mean all the children born to you, whether they were born alive or dead, whether still living or not, whether living with you or elsewhere, and all the pregnancies that you have had that did not result in a live birth. I understand that it is not easy to talk about children who have died, or pregnancies that have terminated before full term, but it is extremely important that you tell us about all of them, so that we can develop programs that will help the Government of South Africa improve children's health in the future.

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
201	Now I would like to ask about all the births you have had during your life. Have you ever given birth?	YES ..... 1 NO 2	→206
202	Do you have any sons or daughters to whom you have given birth who are living with you?	YES ..... 1 NO 2	→204
203	How many sons live with you? And how many daughters live with you? IF NONE, RECORD '00'.	SONS AT HOME ..... [ ][ ] DAUGHTERS AT HOME .. [ ][ ]	
204	Do you have any sons or daughters to whom you have given birth who are alive but do not live with you?	YES ..... 1 NO 2	→206
205	How many sons are alive but do not live with you? And how many daughters are alive but do not live with you? IF NONE, RECORD '00'.	SONS ELSEWHERE ..... [ ][ ] DAUGHTERS ELSEWHERE [ ][ ]	
206	Have you ever given birth to a boy or girl who was born alive but later died? IF NO, PROBE: Any baby who cried or showed signs of life but survived only a few hours or days?	YES ..... 1 NO 2	→208
207	How many boys have died? And how many girls have died? IF NONE, RECORD '00'.	BOYS DEAD ..... [ ][ ] GIRLS DEAD ..... [ ][ ]	
208	Women sometimes have pregnancies that do not result in a live born child. That is, a pregnancy can end very early, in a miscarriage or an abortion or the child can be born dead. Have you had any such pregnancy that did not result in a live birth?	YES ..... 1 NO 2	→210
209	In all, how many such pregnancies have there been?	PREGNANCY LOSSES ... [ ][ ]	
210	SUM ANSWERS TO 203, 205, 207 AND 209, AND ENTER TOTAL. IF NONE, RECORD '00'.	TOTAL ..... [ ][ ]	
212	CHECK 210: ONE OR MORE PREGNANCIES [ ] NO PREGNANCIES [ ]		→234

213 Now I would like to ask you about all of your pregnancies, whether born alive, born dead, or lost before full term, starting with the first one you had.
RECORD ALL THE PREGNANCIES. RECORD TWINS AND TRIPLETS ON SEPARATE LINES.

214 Think back to the time of your (first/next) pregnancy.	215 Was that a single or multiple pregnancy?	216 Was the baby born alive, born dead, or lost before full term?	217 Did that baby cry, move, or breathe when it was born?	218 What was the name given to that child?	219 Is (NAME) a boy or a girl?	220 In what month and year was (NAME) born? PROBE: What is his/her birthday? OR: In what season was he/she born?	221 Is (NAME) still alive?
01	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM 3 (SKIP TO 225)	YES . . 1 NO . . . 2 225	_____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR . 19	YES 1 NO . . 2 224
02	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM 3 (SKIP TO 225)	YES . . 1 NO . . . 2 225	_____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR . 19	YES 1 NO . . 2 224
03	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM 3 (SKIP TO 225)	YES . . 1 NO . . . 2 225	_____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR . 19	YES 1 NO . . 2 224
04	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM 3 (SKIP TO 225)	YES . . 1 NO . . . 2 225	_____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR . 19	YES 1 NO . . 2 224
05	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM 3 (SKIP TO 225)	YES . . 1 NO . . . 2 225	_____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR . 19	YES 1 NO . . 2 224
06	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM 3 (SKIP TO 225)	YES . . 1 NO . . . 2 225	_____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR . 19	YES 1 NO . . 2 224
07	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM 3 (SKIP TO 225)	YES . . 1 NO . . . 2 225	_____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR . 19	YES 1 NO . . 2 224
08	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM 3 (SKIP TO 225)	YES . . 1 NO . . . 2 225	_____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR . 19	YES 1 NO . . 2 224

IF BORN ALIVE AND STILL LIVING:		IF BORN ALIVE BUT NOW DEAD:	IF BORN ALIVE BUT NOW DEAD:	IF BORN DEAD OR LOST BEFORE FULL TERM:			
222 How old was (NAME) at his/her last birthday? RECORD AGE IN COMPLETED YEARS.	223 Is (NAME) living with you?	224 How old was (NAME) when he/she died? IF '1 YR.', PROBE: How many months old was (NAME)? RECORD DAYS IF LESS THAN 1 MONTH; MONTHS IF LESS THAN TWO YEARS; OR YEARS.	224A Did (NAME) die from diarrhoea?	225 In what year and month did this pregnancy end?	226 How many months did the pregnancy last? RECORD IN COMPLETED MONTHS.	228 FROM YEAR OF THIS PREGNANCY SUBTRACT YEAR OF PREVIOUS PREGNANCY. IS THE DIFFERENCE 2 OR MORE YEARS?	229 Were there any other pregnancies between the previous pregnancy mentioned and this pregnancy?
01 AGE IN YEARS	YES 1 NO . 2 (NEXT PREG.)	DAYS . . 1 MONTHS 2 YEARS . 3	YES . . . . 1 NO . . . . . 2 DK . . . . . 8 (NEXT PREG.)	MONTH YEAR 19	MONTHS (next preg.)		
02 AGE IN YEARS	YES 1 NO . 2 (GO TO 228)	DAYS . . 1 MONTHS 2 YEARS . 3	YES . . . . 1 NO . . . . . 2 DK . . . . . 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES . . . . . 1 NO . . . . . 2 (NEXT PREGNANCY)	YES . . . . 1 NO 2
03 AGE IN YEARS	YES 1 NO . 2 (GO TO 228)	DAYS . . 1 MONTHS 2 YEARS . 3	YES . . . . 1 NO . . . . . 2 DK . . . . . 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES . . . . . 1 NO . . . . . 2 (NEXT PREGNANCY)	YES . . . . 1 NO 2
04 AGE IN YEARS	YES 1 NO . 2 (GO TO 228)	DAYS . . 1 MONTHS 2 YEARS . 3	YES . . . . 1 NO . . . . . 2 DK . . . . . 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES . . . . . 1 NO . . . . . 2 (NEXT PREGNANCY)	YES . . . . 1 NO 2
05 AGE IN YEARS	YES 1 NO . 2 (GO TO 228)	DAYS . . 1 MONTHS 2 YEARS . 3	YES . . . . 1 NO . . . . . 2 DK . . . . . 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES . . . . . 1 NO . . . . . 2 (NEXT PREGNANCY)	YES . . . . 1 NO 2
06 AGE IN YEARS	YES 1 NO . 2 (GO TO 228)	DAYS . . 1 MONTHS 2 YEARS . 3	YES . . . . 1 NO . . . . . 2 DK . . . . . 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES . . . . . 1 NO . . . . . 2 (NEXT PREGNANCY)	YES . . . . 1 NO 2
07 AGE IN YEARS	YES 1 NO . 2 (GO TO 228)	DAYS . . 1 MONTHS 2 YEARS . 3	YES . . . . 1 NO . . . . . 2 DK . . . . . 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES . . . . . 1 NO . . . . . 2 (NEXT PREGNANCY)	YES . . . . 1 NO 2
08 AGE IN YEARS	YES 1 NO . 2 (GO TO 228)	DAYS . . 1 MONTHS 2 YEARS . 3	YES . . . . 1 NO . . . . . 2 DK . . . . . 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES . . . . . 1 NO . . . . . 2 (NEXT PREGNANCY)	YES . . . . 1 NO 2

214 Think back to the time of your next pregnancy.	215 Was that a single or multiple pregnancy?	216 Was the baby born alive, born dead, or lost before full term?	217 Did that baby cry, move, or breathe when it was born?	218 What was the name given to that child?	219 Is (NAME) a boy or a girl?	220 In what month and year was (NAME) born? PROBE: What is his/her birthday? OR: In what season was he/she born?	221 Is (NAME) still alive?
09	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM . . . . . 3 (SKIP TO 225)	YES . . . 1 NO 2 225	_____ _____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR 19	YES 1 NO . . 2 224
10	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM . . . . . 3 (SKIP TO 225)	YES . . . 1 NO 2 225	_____ _____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR 19	YES 1 NO . . 2 224
11	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM . . . . . 3 (SKIP TO 225)	YES . . . 1 NO 2 225	_____ _____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR 19	YES 1 NO . . 2 224
12	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM . . . . . 3 (SKIP TO 225)	YES . . . 1 NO 2 225	_____ _ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR 19	YES 1 NO . . 2 224
13	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM . . . . . 3 (SKIP TO 225)	YES . . . 1 NO 2 225	_____ _____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR 19	YES 1 NO . . 2 224
14	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM . . . . . 3 (SKIP TO 225)	YES . . . 1 NO 2 225	_____ _____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR 19	YES 1 NO . . 2 224
15	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM . . . . . 3 (SKIP TO 225)	YES . . . 1 NO 2 225	_____ _____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR 19	YES 1 NO . . 2 224
16	SINGLE . . . 1 MULTIPLE 2	BORN ALIVE . . . . . 1 (SKIP TO 218) BORN DEAD . . . . . 2 LOST BEFORE FULL TERM . . . . . 3 (SKIP TO 225)	YES . . . 1 NO 2 225	_____ _____ (NAME)	BOY . 1 GIRL . 2	MONTH . . . YEAR 19	YES 1 NO . . 2 224

IF BORN ALIVE AND STILL LIVING:		IF BORN ALIVE BUT NOW DEAD:	IF BORN ALIVE BUT NOW DEAD:	IF BORN DEAD OR LOST BEFORE FULL TERM:			
222 How old was (NAME) at his/her last birthday? RECORD AGE IN COMPLETED YEARS.	223 Is (NAME) living with you?	224 How old was (NAME) when he/she died? IF '1 YR.', PROBE: How many months old was (NAME)? RECORD DAYS IF LESS THAN 1 MONTH; MONTHS IF LESS THAN TWO YEARS; OR YEARS.	224A Did (NAME) die from diarrhoea	225 In what year and month did this pregnancy end?	226 How many months did the pregnancy last? RECORD IN COMPLETED MONTHS.	228 FROM YEAR OF THIS PREGNANCY SUBTRACT YEAR OF PREVIOUS PREGNANCY. IS THE DIFFERENCE 2 OR MORE?	229 Were there any other pregnancies between the previous pregnancy mentioned and this pregnancy?
09 AGE IN YEARS	YES 1 NO 2 (GO TO 228)	DAYS 1 MONTHS 2 YEARS 3	YES 1 NO 2 DK 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES 1 NO 2 (NEXT PREGNANCY)	YES 1 NO 2
10 AGE IN YEARS	YES 1 NO 2 (GO TO 228)	DAYS 1 MONTHS 2 YEARS 3	YES 1 NO 2 DK 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES 1 NO 2 (NEXT PREGNANCY)	YES 1 NO 2
11 AGE IN YEARS	YES 1 NO 2 (GO TO 228)	DAYS 1 MONTHS 2 YEARS 3	YES 1 NO 2 DK 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES 1 NO 2 (NEXT PREGNANCY)	YES 1 NO 2
12 AGE IN YEARS	YES 1 NO 2 (GO TO 228)	DAYS 1 MONTHS 2 YEARS 3	YES 1 NO 2 DK 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES 1 NO 2 (NEXT PREGNANCY)	YES 1 NO 2
13 AGE IN YEARS	YES 1 NO 2 (GO TO 228)	DAYS 1 MONTHS 2 YEARS 3	YES 1 NO 2 DK 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES 1 NO 2 (NEXT PREGNANCY)	YES 1 NO 2
14 AGE IN YEARS	YES 1 NO 2 (GO TO 228)	DAYS 1 MONTHS 2 YEARS 3	YES 1 NO 2 DK 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES 1 NO 2 (NEXT PREGNANCY)	YES 1 NO 2
15 AGE IN YEARS	YES 1 NO 2 (GO TO 228)	DAYS 1 MONTHS 2 YEARS 3	YES 1 NO 2 DK 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES 1 NO 2 (NEXT PREGNANCY)	YES 1 NO 2
16 AGE IN YEARS	YES 1 NO 2 (GO TO 228)	DAYS 1 MONTHS 2 YEARS 3	YES 1 NO 2 DK 8 (GO TO 228)	MONTH YEAR 19	MONTHS	YES 1 NO 2 (NEXT PREGNANCY)	YES 1 NO 2

230	FROM YEAR OF INTERVIEW SUBTRACT YEAR OF LAST PREGNANCY. IS THE DIFFERENCE 2 YEARS OR MORE?	YES . . . . . 1 NO 2	 →232
231	Have you had any pregnancies since the last pregnancy mentioned?	YES . . . . . 1 NO 2	→214
232	COMPARE 210 WITH NUMBER OF PREGNANCIES IN HISTORY ABOVE AND MARK: NUMBERS ARE SAME ☐ NUMBERS ARE DIFFERENT ☐→ (PROBE AND RECONCILE) CHECK: FOR EACH PREGNANCY: YEAR IS RECORDED IN 220 OR 225. FOR EACH LIVING CHILD: CURRENT AGE IS RECORDED IN 222. FOR EACH DEAD CHILD: AGE AT DEATH IS RECORDED IN 224. FOR EACH PREGNANCY LOSS: DURATION IS RECORDED IN 226. FOR AGE AT DEATH 12 MONTHS OR 1 YR.: PROBE TO DETERMINE EXACT NUMBER OF MONTHS.		☐
233	CHECK 220 AND ENTER THE NUMBER OF BIRTHS SINCE JANUARY 1993. IF NONE, RECORD '0'.		☐

234	Are you pregnant now?	YES . . . . . 1 NO . . . . . 2 UNSURE 8	→237 →237
235	How many months pregnant are you?	MONTHS ☐☐	
236	At the time you became pregnant, did you want to become pregnant then, did you want to wait until later, or did you not want to have anymore children at all?	THEN . . . . . 1 LATER . . . . . 2 NOT WANT MORE CHILDREN 3	
237	When did your last menstrual period start? _____ (DATE, IF GIVEN)	DAYS AGO . . . . . 1 ☐☐ WEEKS AGO . . . . . 2 ☐☐ MONTHS AGO . . . . . 3 ☐☐ YEARS AGO . . . . . 4 ☐☐ IN MENOPAUSE . . . . . 994 BEFORE LAST BIRTH . . . . . 995 NEVER MENSTRUATED 996	

238	Do you have any of the following problems:		YES	NO
	Wet yourself when you cough, sneeze or lift heavy weights?	WET WHEN COUGH/SNEEZE . . .	1	2
	Are you constantly wet?	CONSTANTLY WET	1	2
	Are you constantly soiled?	CONSTANTLY SOILED	1	2

SECTION 3. CONTRACEPTION

Now I would like to talk about family planning - the various ways or methods that a couple can use to delay or avoid a pregnancy.

CIRCLE CODE 1 IN 301 FOR EACH METHOD MENTIONED SPONTANEOUSLY.
THEN PROCEED DOWN COLUMN 302, READING THE NAME AND DESCRIPTION OF EACH METHOD NOT MENTIONED SPONTANEOUSLY. CIRCLE CODE 2 IF METHOD IS RECOGNIZED, AND CODE 3 IF NOT RECOGNIZED.
THEN, FOR EACH METHOD WITH CODE 1 OR 2 CIRCLED IN 301 OR 302, ASK 303.

301	Which ways or methods have you heard about?	302 Have you ever heard of (METHOD)? SPONTANEOUS YES	PROBED YES	NO	303 Have you ever used (METHOD)?
01	PILL Women can take a pill every day.	1	2	3 →	YES . . . . . 1 NO 2
02	IUD Women can have a loop or coil placed inside them by a doctor or a nurse.	1	2	3 →	YES . . . . . 1 NO 2
03	INJECTIONS Women can have an injection by a doctor or nurse which stops them from becoming pregnant for several months.	1	2	3 →	YES . . . . . 1 NO 2
04	DIAPHRAGM, FOAM, JELLY Women can place a sponge, suppository, diaphragm, jelly, or cream inside themselves before intercourse.	1	2	3 →	YES . . . . . 1 NO 2
05	CONDOM Men can put a rubber sheath on their penis during sexual intercourse.	1	2	3 →	YES . . . . . 1 NO 2
06	FEMALE STERILIZATION Tie the tubes. Women can have an operation to avoid having any more children.	1	2	3 →	Have you ever had an operation to avoid having any more children? YES . . . . . 1 NO 2
07	MALE STERILIZATION Men can have an operation to avoid having any more children.	1	2	3 →	Have you ever had a partner who had an operation to avoid having children? YES . . . . . 1 NO 2
08	RHYTHM, CALENDAR METHOD Every month that a woman is sexually active she can avoid having sexual intercourse on the days of the month she is most likely to get pregnant.	1	2	3 →	YES . . . . . 1 NO 2
09	WITHDRAWAL Men can be careful and pull out before climax.	1	2	3 →	YES . . . . . 1 NO 2
10	HERBS. Women use natural herbs or Dutch remedies to avoid pregnancy	1	2	3 →	YES . . . . . 1 NO 2
11	Have you heard of any other ways or methods that women or men can use to avoid pregnancy?	1 _____ (SPECIFY) _____ (SPECIFY)		3	YES . . . . . 1 NO . . . . . 2 YES . . . . . 1 NO 2

304	CHECK 303:
	NOT A SINGLE "YES" → (NEVER USED)
	AT LEAST ONE "YES" → (EVER USED) → SKIP TO 308

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
305	Have you ever used anything or tried in any way to delay or avoid getting pregnant?	YES . . . . . 1 NO 2	→331
307	What have you used or done? CORRECT 303 AND 304 (AND 302 IF NECESSARY).		
308	Now I would like to ask you about the first time that you did something or used a method to avoid getting pregnant. What was the first method you ever used?	PILL . . . . . 01 IUD . . . . . 02 INJECTIONS . . . . . 03 DIAPHRAGM/FOAM/JELLY . . . . . 04 CONDOM . . . . . 05 FEMALE STERILIZATION . . . . . 06 MALE STERILIZATION . . . . . 07 RHYTHM/ CALENDER METHOD . . . 08 WITHDRAWAL . . . . . 09 HERB/REMEDIES . . . . . 10 OTHER _____ 96 (SPECIFY)	
309	How many living children did you have at that time, if any? IF NONE, RECORD '00'.	NUMBER OF CHILDREN . . [ ][ ]	
309A	How old were you when you first used something to avoid getting pregnant?	AGE . . . . . [ ][ ]	
309B	From whom did you first get information about methods to avoid pregnancy?	MOTHER . . . . . A SISTER . . . . . B FATHER . . . . . C OTHER RELATIVE . . . . . D FRIEND . . . . . E TEACHER . . . . . F NURSE . . . . . G DOCTOR . . . . . H POSTER/LEAFLET/MAGAZINE . . . . . I RADIO/TELEVISION . . . . . J OTHER _____ X (SPECIFY)	
309C	CHECK 309A: AGE LESS THAN 19 YEARS [ ] ▼ AGE 19 YEARS OR OLDER [ ]		→311
309E	Did your parent(s) or guardian give advice on contraceptives or explain how to use them?	YES . . . . . 1 NO 2	
311	CHECK 303: WOMAN NOT STERILIZED [ ] ▼ WOMAN STERILIZED [ ]		→314A
312	CHECK 234: NOT PREGNANT OR UNSURE [ ] ▼ PREGNANT [ ]		→331
313	Are you currently doing something or using any method to delay or avoid getting pregnant?	YES . . . . . 1 NO 2	→331
314	Which method are you using?	PILL . . . . . 01 IUD . . . . . 02 INJECTIONS . . . . . 03 DIAPHRAGM/FOAM/JELLY . . . . . 04 CONDOM 05	→328
314A	CIRCLE '06' FOR FEMALE STERILIZATION.	FEMALE STERILIZATION . . . . . 06 MALE STERILIZATION . . . . . 07 RHYTHM, CALENDER METHOD . . . 08 WITHDRAWAL . . . . . 09 HERB/REMEDIES . . . . . 10 OTHER _____ 96 (SPECIFY)	→323 →332

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
318	Where did the sterilization take place? IF SOURCE IS HOSPITAL, HEALTH CENTER, OR CLINIC, WRITE THE NAME OF THE PLACE. PROBE TO IDENTIFY THE TYPE OF SOURCE AND CIRCLE THE APPROPRIATE CODE. _____ (NAME OF PLACE)	PUBLIC SECTOR GOVERNMENT HOSPITAL .... 11 DAY HOSPITAL/CLINIC/ COMMUNITY HEALTH CENTRE 12 FAMILY PLANNING CLINIC .... 13 OTHER PUBLIC _____ 16 (SPECIFY) PRIVATE MEDICAL SECTOR PRIVATE HOSPITAL/CLINIC ... 21 PRIVATE DOCTOR ..... 23 OTHER PRIVATE MEDICAL _____ 26 (SPECIFY) OTHER _____ 96 (SPECIFY) DON'T KNOW 98	
319	Do you regret that (you/your partner) had the operation not to have any (more) children?	YES ..... 1 NO 2	→ 321
320	Why do you regret the operation?	RESPONDENT WANTS ANOTHER CHILD ..... 01 PARTNER WANTS ANOTHER CHILD 02 SIDE EFFECTS ..... 03 CHILD DIED ..... 04 OTHER _____ 96 (SPECIFY)	
321	In what month and year was the sterilization performed?	MONTH ..... ▒▒ YEAR ..... ▒▒▒▒	→ 335
323	How do you determine which days of your monthly cycle not to have sexual relations?	BASED ON CALENDAR ..... 01 BASED ON BODY TEMPERATURE . 02 BASED ON CERVICAL MUCUS (BILLINGS METHOD) ..... 03 BASED ON BODY TEMPERATURE AND CERVICAL MUCUS ..... 04 NO SPECIFIC SYSTEM ..... 05 OTHER _____ 96 (SPECIFY)	→ 332
328	Where did you obtain (METHOD) the last time? IF SOURCE IS HOSPITAL, HEALTH CENTER, OR CLINIC, WRITE THE NAME OF THE PLACE. PROBE TO IDENTIFY THE TYPE OF SOURCE AND CIRCLE THE APPROPRIATE CODE. _____ (NAME OF PLACE)	PUBLIC SECTOR GOVERNMENT HOSPITAL .... 11 DAY HOSPITAL/CLINIC/ COMMUNITY HEALTH CENTER 12 FAMILY PLANNING CLINIC ... 13 MOBILE CLINIC ..... 14 COMMUNITY HEALTH WORKER 15 OTHER PUBLIC _____ 16 (SPECIFY) PRIVATE MEDICAL SECTOR PRIVATE HOSPITAL/CLINIC .. 21 PHARMACY ..... 22 PRIVATE DOCTOR/ GYNECOLOGIST ..... 23 OTHER PRIVATE MEDICAL _____ 26 (SPECIFY) OTHER SOURCE SHOP ..... 31 CHURCH ..... 32 FRIEND/RELATIVE ..... 33 OTHER _____ 96 (SPECIFY)	→ 330A

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
330	Do you agree with the following statements about the family planning service you use? The staff shout and scold The staff do not explain much about the Family Planning method The staff ignore problems which you report The staff are unfriendly	AGREE DISAGREE 1 2 1 2 1 2 1 2	
330A	People select the place where they get family planning services for various reasons. What were the reasons you went to (NAME OF PLACE IN Q.328) instead of some other place you know about? RECORD ALL RESPONSES AND CIRCLE CODES. What is the Main Reason?	ACCESS-RELATED REASONS CLOSER TO HOME ..... A CLOSER TO MARKET/WORK .. B AVAILABILITY OF TRANSPORT C SERVICE-RELATED REASONS STAFF MORE COMPETENT/ FRIENDLY ..... D CLEANER FACILITY ..... E OFFERS MORE PRIVACY ..... F SHORTER WAITING TIME ..... G LONGER HRS. OF SERVICE ... H USE OTHER SERVICES AT THE FACILITY ..... I LOWER COST/CHEAPER ..... J WANTED ANONYMITY ..... K OTHER _____ X (SPECIFY) DON'T KNOW ..... Z	
330B	Over the last 12 months have you had a break in your contraceptive use for any reason?	YES ..... 1 NO 2	→ 335
330C	Over the last 12 months, why have you had a break in your contraceptive use?	WAS PREGNANT ..... 01 NO BOYFRIEND/ SEXUALLY INACTIVE ..... 02 WANTED TO SEE MENSTRUATION 03 HEALTH REASONS ..... 04 OTHER _____ . 96 (SPECIFY)	→ 335
331	What are the main reasons you are not using a method of contraception to avoid pregnancy? RECORD ALL MENTIONED What is the Main Reason?	NEVER HAD SEX ..... A FERTILITY-RELATED REASONS NOT HAVING SEX ..... B INFREQUENT SEX ..... C MENOPAUSAL/HYSTERECTOMY D INFERTILE ..... E POSTPARTUM/BREASTFEEDING F WANTS (MORE) CHILDREN .... G PREGNANT ..... H OPPOSITION TO USE RESPONDENT OPPOSED ..... I HUSBAND/PARTNER OPPOSED J OTHERS OPPOSED ..... K RELIGIOUS PROHIBITION ..... L LACK OF KNOWLEDGE KNOWS NO METHOD ..... M KNOWS NO SOURCE ..... N METHOD-RELATED REASONS HEALTH CONCERNS ..... O FEAR OF SIDE EFFECTS..... P LACK OF ACCESS/TOO FAR ... Q COST TOO MUCH ..... R INCONVENIENT TO USE ..... S INTERFERES WITH BODY'S NATURAL PROCESSES .. T OUT OF STOCK ..... U OTHER _____ X (SPECIFY) DON'T KNOW ..... Z	
332	Do you know of a place where you can obtain a method of family planning?	YES ..... 1 NO 2	→ 335

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
333	Where is that? IF SOURCE IS HOSPITAL, HEALTH CENTER, OR CLINIC, WRITE THE NAME OF THE PLACE. PROBE TO IDENTIFY THE TYPE OF SOURCE AND CIRCLE THE APPROPRIATE CODE. _____ (NAME OF PLACE)	PUBLIC SECTOR GOVERNMENT HOSPITAL .... 11 DAY HOSPITAL/CLINIC/ COMMUNITY HEALTH CENTER 12 FAMILY PLANNING CLINIC ... 13 MOBILE CLINIC ..... 14 COMMUNITY HEALTH WORKER 15 OTHER PUBLIC _____ 16 (SPECIFY) PRIVATE MEDICAL SECTOR PRIVATE HOSPITAL/CLINIC .. 21 PHARMACY ..... 22 PRIVATE DOCTOR/ GYNECOLOGIST ..... 23 OTHER PRIVATE MEDICAL _____ 26 (SPECIFY) OTHER SOURCE SHOP ..... 31 CHURCH ..... 32 FRIEND/RELATIVE ..... 33 OTHER _____ 96 (SPECIFY)	
335	Have you visited any type of health facility for any reason in the last 12 months?	YES ..... 1 NO 2	→337
336	Did any staff member at the health facility speak to you about family planning methods?	YES ..... 1 NO 2	
337	During which times of the monthly cycle does a woman have the greatest chance of becoming pregnant?	DURING HER PERIOD ..... 01 RIGHT AFTER HER PERIOD HAS ENDED ..... 02 IN THE MIDDLE OF THE CYCLE ... 03 JUST BEFORE HER PERIOD BEGINS 04 OTHER _____ 96 (SPECIFY) DON'T KNOW 98	
338	I would like to ask you a question about the law on abortion in South Africa. Does the present law allow a woman in early pregnancy, which is up to 12 weeks, to have an abortion?	YES ..... 1 NO ..... 2 DON'T KNOW 8	

SECTION 4A. PREGNANCY AND CHILD HEALTH

401	CHECK 233: ONE OR MORE BIRTHS SINCE JAN. 1993 ☐ * NO BIRTHS SINCE JAN. 1993 ☐ → (SKIP TO 465)		
402	ENTER THE NAME, LINE NUMBER, AND SURVIVAL STATUS OF EACH BIRTH SINCE JANUARY 1993 IN THE TABLE. ASK THE QUESTIONS ABOUT ALL OF THESE BIRTHS. BEGIN WITH THE LAST BIRTH. (IF THERE ARE MORE THAN 2 BIRTHS, USE ADDITIONAL QUESTIONNAIRES). Now I would like to ask you some questions about your pregnancies and the health of all your children born in the last five years. (We will talk about one child at a time.)		
403	LINE NUMBER FROM Q214	LAST BIRTH LINE NUMBER .... ☐☐	NEXT-TO-LAST BIRTH LINE NUMBER .... ☐☐
404	FROM Q218 AND Q221	NAME _____ ALIVE ☐ * DEAD ☐ *	NAME _____ ALIVE ☐ * DEAD ☐ *
405	At the time you became pregnant with (NAME), did you want to become pregnant then, did you want to wait until later, or did you want no (more) children at all?	THEN .... 1 (SKIP TO 407) LATER .... 2 NO MORE .... 3 (SKIP TO 407)	THEN .... 1 (SKIP TO 407) LATER .... 2 NO MORE .... 3 (SKIP TO 407)
406	How much longer would you like to have waited?	MONTHS .... 1 ☐☐ YEARS .... 2 ☐☐ DON'T KNOW 998	MONTHS .... 1 ☐☐ YEARS .... 2 ☐☐ DON'T KNOW 998
407	When you were pregnant with (NAME), did you go for antenatal care for this pregnancy? IF YES: Whom did you see? Anyone else? PROBE FOR THE TYPE OF PERSON AND RECORD ALL PERSONS SEEN.	HEALTH PROFESSIONAL DOCTOR .... A NURSE/MIDWIFE .... B OTHER PERSON TRADITIONAL BIRTH ATTENDANT .... D OTHER _____ X (SPECIFY) NO ONE .... Y (SKIP TO 410)	HEALTH PROFESSIONAL DOCTOR .... A NURSE/MIDWIFE .... B OTHER PERSON TRADITIONAL BIRTH ATTENDANT .... D OTHER _____ X (SPECIFY) NO ONE .... Y (SKIP TO 410)
407A	Where did you go the majority of times? PROBE FOR THE ONE PLACE VISITED MOST OFTEN	PUBLIC HOSPITAL .... 01 PRIVATE HOSPITAL .... 02 PUBLIC CLINIC .... 03 PRIVATE CLINIC/SURGERY 04 PRIVATE MIDWIFE'S OFFICE 05 OTHER _____ 96 (SPECIFY)	PUBLIC HOSPITAL .... 01 PRIVATE HOSPITAL .... 02 PUBLIC CLINIC .... 03 PRIVATE CLINIC/SURGERY 04 PRIVATE MIDWIFE'S OFFICE 05 OTHER _____ 96 (SPECIFY)
408	How many months pregnant were you when you first received antenatal care?	MONTHS .... ☐☐ DON'T KNOW 98	MONTHS .... ☐☐ DON'T KNOW 98
409	How many times did you receive antenatal care during this pregnancy?	NO. OF TIMES .... ☐☐ DON'T KNOW 98	NO. OF TIMES .... ☐☐ DON'T KNOW 98
410	When you were pregnant with (NAME) were you given an injection in the arm to prevent the baby from getting tetanus, that is, convulsions after birth?	YES .... 1 NO .... 2 DON'T KNOW 8	YES .... 1 NO .... 2 DON'T KNOW 8

412	Where did you give birth to (NAME)? _____ NAME OF PLACE	HOME ..... 11 PUBLIC SECTOR GOVT. HOSPITAL ..... 21 DAY HOSP/CLINIC COMMUNITY HEALTH CENTER ..... 22 GOVT. MOU ..... 23 OTHER PUBLIC _____ 26 (SPECIFY) PRIVATE MEDICAL SECTOR PVT. HOSPITAL/CLINIC .. 31 OTHER PRIVATE MEDICAL _____ 36 (SPECIFY) OTHER _____ 96 (SPECIFY)	HOME ..... 11 PUBLIC SECTOR GOVT. HOSPITAL ..... 21 DAY HOSP/CLINIC/ COMMUNITY HEALTH CENTER ..... 22 GOVT. MOU ..... 23 OTHER PUBLIC _____ 26 (SPECIFY) PRIVATE MEDICAL SECTOR PVT. HOSPITAL/CLINIC ... 31 OTHER PRIVATE MEDICAL _____ 36 (SPECIFY) OTHER _____ 96 (SPECIFY)
413	Who assisted with the delivery of (NAME)? Anyone else? PROBE FOR THE TYPE OF PERSON AND RECORD ALL PERSONS ASSISTING.	HEALTH PROFESSIONAL DOCTOR ..... A NURSE/MIDWIFE ..... B OTHER PERSON TRADITIONAL BIRTH ATTENDANT ..... D RELATIVE/FRIEND ..... E OTHER _____ X (SPECIFY) NO ONE ..... Y	HEALTH PROFESSIONAL DOCTOR ..... A NURSE/MIDWIFE ..... B OTHER PERSON TRADITIONAL BIRTH ATTENDANT ..... D RELATIVE/FRIEND ..... E OTHER _____ X (SPECIFY) NO ONE ..... Y
415	Was (NAME) delivered by caesarian section?	YES ..... 1 NO 2	YES ..... 1 NO 2
417	Was (NAME) weighed at birth?	YES ..... 1 NO ..... 2 (SKIP TO 419)	YES ..... 1 NO ..... 2 (SKIP TO 420)
418	How much did (NAME) weigh? RECORD WEIGHT FROM HEALTH CARD, IF AVAILABLE.	GRAMS FROM CARD .. 1 [ ][ ][ ][ ] GRAMS FROM RECALL 2 [ ][ ][ ][ ] DON'T KNOW 99998	GRAMS FROM CARD .. 1 [ ][ ][ ][ ] GRAMS FROM RECALL 2 [ ][ ][ ][ ] DON'T KNOW 99998
419	Has your period returned since the birth of (NAME)?	YES ..... 1 (SKIP TO 421) NO ..... 2 (SKIP TO 422)	
420	Did your period return between the birth of (NAME) and your next pregnancy?		YES ..... 1 NO ..... 2 (SKIP TO 424)
421	For how many months after the birth of (NAME) did you not have a period?	MONTHS ..... [ ][ ] DON'T KNOW 98	MONTHS ..... [ ][ ] DON'T KNOW 98
422	CHECK 234: RESPONDENT PREGNANT?	NOT PREGNANT [ ] PREG- [ ] NANT * OR UNSURE [ ] (SKIP TO 424)	
423	Have you resumed sexual relations since the birth of (NAME)?	YES ..... 1 NO ..... 2 (SKIP TO 425)	
424	For how many months after the birth of (NAME) did you not have sexual relations?	MONTHS ..... [ ][ ] DON'T KNOW 98	MONTHS ..... [ ][ ] DON'T KNOW 98
425	Did you ever breastfeed (NAME)?	YES ..... 1 NO ..... 2 (SKIP TO 431)	YES ..... 1 NO ..... 2 (SKIP TO 431)

426	How long after birth did you first put (NAME) to the breast? IF LESS THAN 1 HOUR, RECORD '00' HOURS. IF LESS THAN 24 HOURS, RECORD HOURS. OTHERWISE, RECORD DAYS.	IMMEDIATELY . . . . . 000 HOURS . . . . . 1 ☐☐ DAYS . . . . . 2 ☐☐	IMMEDIATELY . . . . . 000 HOURS . . . . . 1 ☐☐ DAYS . . . . . 2 ☐☐
427	CHECK 404: CHILD ALIVE?	ALIVE ☐ DEAD ☐ ↓ (SKIP TO 429)	ALIVE ☐ DEAD ☐ ↓ (SKIP TO 429)
428	Are you still breastfeeding (NAME)?	YES . . . . . 1 (SKIP TO 432) NO 2	YES . . . . . 1 (SKIP TO 432) NO 2
429	For how many months did you breastfeed (NAME)?	MONTHS . . . . . ☐☐ DON'T KNOW 98	MONTHS . . . . . ☐☐ DON'T KNOW 98
430	Why did you stop breastfeeding (NAME)?	MOTHER ILL/WEAK . . . . . 01 CHILD ILL/WEAK . . . . . 02 CHILD DIED . . . . . 03 NIPPLE/BREAST PROBLEM . 04 NOT ENOUGH MILK . . . . . 05 MOTHER WORKING . . . . . 06 CHILD REFUSED . . . . . 07 WEANING AGE/AGE TO STOP . . . . . 08 BECAME PREGNANT . . . . . 09 STARTED USING CONTRACEPTION . . . . . 10 OTHER _____ 96 (SPECIFY)	MOTHER ILL/WEAK . . . . . 01 CHILD ILL/WEAK . . . . . 02 CHILD DIED . . . . . 03 NIPPLE/BREAST PROBLEM . 04 NOT ENOUGH MILK . . . . . 05 MOTHER WORKING . . . . . 06 CHILD REFUSED . . . . . 07 WEANING AGE/AGE TO STOP . . . . . 08 BECAME PREGNANT . . . . . 09 STARTED USING CONTRACEPTION . . . . . 10 OTHER _____ 96 (SPECIFY)
431	CHECK 404: CHILD ALIVE?	ALIVE ☐ DEAD ☐ ↓ ↓ (SKIP TO 434) (GO BACK TO 405 IN NEXT COLUMN OR, IF NO MORE BIRTHS, GO TO 440)	ALIVE ☐ DEAD ☐ ↓ ↓ (SKIP TO 434) (GO BACK TO 405 IN NEXT COLUMN OR, IF NO MORE BIRTHS, GO TO 440)
432	How many times did you breastfeed last night between sunset and sunrise? IF ANSWER IS NOT NUMERIC, PROBE FOR APPROXIMATE NUMBER	NUMBER OF NIGHTTIME FEEDINGS . . . . . ☐☐	NUMBER OF NIGHTTIME FEEDINGS . . . . . ☐☐
433	How many times did you breastfeed yesterday during the daylight hours? IF ANSWER IS NOT NUMERIC PROBE FOR APPROXIMATE NUMBER.	NUMBER OF DAYLIGHT FEEDINGS . . . . . ☐☐	NUMBER OF DAYLIGHT FEEDINGS . . . . . ☐☐
434	Did (NAME) drink anything from a bottle with a nipple yesterday or last night?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8

435	At any time yesterday or last night, was (NAME) given any of the following: Plain water? Sugar water/Juice Herbal tea/Rooibos? Baby formula? Any kind of milk? Any other liquid? Any food made from [MAIZE or RICE or WHEAT], such as PORRIDGE or BREAD Eggs, fish or poultry? Meat? Fruits or vegetables? Any other solid or semi-solid foods?	YES NO DK PLAIN WATER . . . . . 1 2 8 SUGAR WATER/JUICE 1 2 8 HERBAL/ROOIBOS TEA 1 2 8 BABY FORMULA . . . . 1 2 8 ANY KIND OF MILK . . 1 2 8 OTHER LIQUIDS . . . . 1 2 8 FOOD MADE FROM MAIZE/RICE/WHEAT 1 2 8 EGGS/FISH/POULTRY 1 2 8 MEAT . . . . . 1 2 8 FRUITS OR VEG. . . . 1 2 8 OTHER SOLID/ SEMI-SOLID FOODS . 1 2 8	YES NO DK PLAIN WATER . . . . . 1 2 8 SUGAR WATER/JUICE 1 2 8 HERBAL/ROOIBOS TEA 1 2 8 BABY FORMULA . . . . 1 2 8 ANY KIND OF MILK . . 1 2 8 OTHER LIQUIDS . . . . 1 2 8 FOOD MADE FROM MAIZE/RICE/WHEAT 1 2 8 EGGS/FISH/POULTRY 1 2 8 MEAT . . . . . 1 2 8 FRUITS OR VEG. . . . 1 2 8 OTHER SOLID/ SEMI-SOLID FOODS . 1 2 8
436	CHECK 435: FOOD OR LIQUID GIVEN YESTERDAY?	"YES" TO ONE MORE → ; "NO/DK" TO ALL → (SKIP TO 439)	"YES" TO ONE MORE → ; "NO/DK" TO ALL → (SKIP TO 439)
437	(Aside from breast-feeding,) how many times did (NAME) eat yesterday, including both meals and snacks? IF 7 OR MORE TIMES, RECORD '7'.	NUMBER OF TIMES . . . . ▒▒ DON'T KNOW 8	NUMBER OF TIMES . . . . ▒▒ DON'T KNOW 8
439		GO BACK TO 405 IN NEXT COLUMN; OR, IF NO MORE BIRTHS, GO TO 440.	GO BACK TO 405 IN NEXT COLUMN; OR, IF NO MORE BIRTHS, GO TO 440.

SECTION 4B: IMMUNIZATION AND HEALTH

440	ENTER THE NAME, LINE NUMBER, AND SURVIVAL STATUS OF EACH BIRTH SINCE JANUARY 1993 IN THE TABLE. ASK THE QUESTIONS ABOUT ALL OF THESE BIRTHS. BEGIN WITH THE LAST BIRTH. (IF THERE ARE MORE THAN 2 BIRTHS, USE ADDITIONAL QUESTIONNAIRES).		
441	LINE NUMBER FROM Q214	LAST BIRTH LINE NUMBER	NEXT-TO-LAST BIRTH LINE NUMBER ....
442	FROM Q218 AND Q221	NAME _____ ALIVE ☐ DEAD ☐ (GO TO 442 IN NEXT COLUMN; OR, IF NO MORE BIRTHS, GO TO 465.)	NAME _____ ALIVE ☐ DEAD ☐ (GO TO 442 IN NEXT COLUMN; OR, IF NO MORE BIRTHS, GO TO 465.)
443	Do you have a card where (NAME'S) vaccinations are written down? IF YES: May I see it please?	YES, SEEN ..... 1 (SKIP TO 445) YES, NOT SEEN ..... 2 (SKIP TO 447) NO CARD 3	YES, SEEN ..... 1 (SKIP TO 445) YES, NOT SEEN ..... 2 (SKIP TO 447) NO CARD 3
444	Did you ever have a vaccination card for (NAME)?	YES ..... 1 (SKIP TO 447) NO 2	YES ..... 1 (SKIP TO 447) NO 2
445	(1) COPY VACCINATION DATE FOR EACH VACCINE FROM THE CARD (2) WRITE '44' IN 'DAY' COLUMN IF CARD SHOWS THAT A VACCINATION WAS GIVEN, BUT NO DATE IS RECORDED BCG Polio 0 (at birth) Polio 1 Polio 2 Polio 3 DPT 1 DPT 2 DPT 3 Hep. B 1 Hep. B 2 Hep. B 3 Measles	DAY MO YR BCG ☐☐ ☐☐ 19☐☐ P0 ☐☐ ☐☐ 19☐☐ P1 ☐☐ ☐☐ 19☐☐ P2 ☐☐ ☐☐ 19☐☐ P3 ☐☐ ☐☐ 19☐☐ D1 ☐☐ ☐☐ 19☐☐ D2 ☐☐ ☐☐ 19☐☐ D3 ☐☐ ☐☐ 19☐☐ H1 ☐☐ ☐☐ 19☐☐ H2 ☐☐ ☐☐ 19☐☐ H3 ☐☐ ☐☐ 19☐☐ Meas ☐☐ ☐☐ 19☐☐	DAY MO YR BCG ☐☐ ☐☐ 19☐☐ P0 ☐☐ ☐☐ 19☐☐ P1 ☐☐ ☐☐ 19☐☐ P2 ☐☐ ☐☐ 19☐☐ P3 ☐☐ ☐☐ 19☐☐ D1 ☐☐ ☐☐ 19☐☐ D2 ☐☐ ☐☐ 19☐☐ D3 ☐☐ ☐☐ 19☐☐ H1 ☐☐ ☐☐ 19☐☐ H2 ☐☐ ☐☐ 19☐☐ H3 ☐☐ ☐☐ 19☐☐ Meas ☐☐ ☐☐ 19☐☐
446	Did (NAME) receive any vaccinations that are not recorded on this card? RECORD 'YES' ONLY IF RESPONDENT MENTIONES BCG, POLIO 0-3, DPT 1-3, AND/OR MEASLES VACCINE(S).	YES 1 (PROBE FOR VACCINATIONS AND WRITE '66' IN THE CORRESPONDING DAY COLUMN IN 445) NO ..... 2 DON'T KNOW ..... 8 (SKIP TO 450)	YES 1 (PROBE FOR VACCINATIONS AND WRITE '66' IN THE CORRESPONDING DAY COLUMN IN 445) NO ..... 2 DON'T KNOW ..... 8 (SKIP TO 450)
447	Did (NAME) ever receive any vaccinations to prevent him/her from getting diseases?	YES ..... 1 NO ..... 2 (SKIP TO 450) DON'T KNOW 8	YES ..... 1 NO ..... 2 (SKIP TO 450) DON'T KNOW 8

448	Please tell me if (NAME) received any of the following vaccinations:		
448A	A BCG vaccination against tuberculosis, that is, an injection in the left arm or shoulder that caused a scar?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8
448B	Polio vaccine, that is, drops in the mouth?	YES . . . . . 1 NO . . . . . 2 (SKIP TO 448E) DON'T KNOW 8	YES . . . . . 1 NO . . . . . 2 (SKIP TO 448E) DON'T KNOW 8
448C	How many times?	NUMBER OF TIMES . . . . . [ ] DON'T KNOW 8	NUMBER OF TIMES . . . . [ ] DON'T KNOW 8
448D	When was the first polio vaccine given, just after birth or later?	JUST AFTER BIRTH. . . . . 1 LATER 2	JUST AFTER BIRTH. . . . . 1 LATER 2
448E	DPT vaccination, that is, an injection usually given at the same time as polio drops?	YES . . . . . 1 NO . . . . . 2 (SKIP TO 448G) DON'T KNOW 8	YES . . . . . 1 NO . . . . . 2 (SKIP TO 448G) DON'T KNOW 8
448F	How many times?	NUMBER OF TIMES . . . . . [ ] DON'T KNOW 8	NUMBER OF TIMES . . . . [ ] DON'T KNOW 8
448G	An injection to prevent measles?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8
448H	An injection to prevent hepatitis B?	YES . . . . . 1 NO . . . . . 2 (SKIP TO 450) DON'T KNOW 8	YES . . . . . 1 NO . . . . . 2 (SKIP TO 450) DON'T KNOW 8
448I	How many times?	NUMBER OF TIMES . . . . . [ ] DON'T KNOW 8	NUMBER OF TIMES . . . . [ ] DON'T KNOW 8
450	Has (NAME) been ill or feverish with a cough at any time in the last 2 weeks?	YES . . . . . 1 NO . . . . . 2 (SKIP TO 454) DON'T KNOW 8	YES . . . . . 1 NO . . . . . 2 (SKIP TO 454) DON'T KNOW 8
451	When (NAME) was ill with a cough, did he/she breathe with difficulty or faster than usual with short, fast breaths?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8
452	Did you seek advice or treatment for the illness?	YES . . . . . 1 NO . . . . . 2 (SKIP TO 454)	YES . . . . . 1 NO . . . . . 2 (SKIP TO 454)
453	Where did you seek advice or treatment? Anywhere else? RECORD ALL MENTIONED. _____ NAME OF PLACE	PUBLIC SECTOR GOVT. HOSPITAL . . . . . A DAY HOSP/CLINIC/ COMMUNITY HEALTH CENTER . . . . . B MOBILE CLINIC . . . . . D COMM. HEALTH WORKER . . E OTHER PUBLIC _____ F (SPECIFY) PRIVATE MEDICAL SECTOR PVT. HOSPITAL/CLINIC . . . G PHARMACY . . . . . H PRIVATE DOCTOR . . . . . I OTHER PRIVATE MEDICAL _____ J (SPECIFY) OTHER SOURCE SHOP . . . . . K TRAD. HEALER . . . . . L OTHER _____ X (SPECIFY)	PUBLIC SECTOR GOVT. HOSPITAL . . . . . A DAY HOSP/CLINIC/ COMMUNITY HEALTH CENTER . . . . . B MOBILE CLINIC . . . . . D COMM. HEALTH WORKER . E OTHER PUBLIC _____ F (SPECIFY) PRIVATE MEDICAL SECTOR PVT. HOSPITAL/CLINIC . . . G PHARMACY . . . . . H PRIVATE DOCTOR . . . . . I OTHER PRIVATE MEDICAL _____ J (SPECIFY) OTHER SOURCE SHOP . . . . . K TRAD. HEALER . . . . . L OTHER _____ X (SPECIFY)

454	Has (NAME) had diarrhoea in the last 2 weeks?	YES ..... 1 NO ..... 2 (SKIP TO 464) DON'T KNOW 8	YES ..... 1 NO ..... 2 (SKIP TO 464) DON'T KNOW 8
455	Was there any blood in the stools?	YES ..... 1 NO ..... 2 DON'T KNOW 8	YES ..... 1 NO ..... 2 DON'T KNOW 8
456	On the worst day of the diarrhoea, how many bowel movements did (NAME) have?	NUMBER OF BOWEL MOVEMENTS ..... [ ][ ] DON'T KNOW 98	NUMBER OF BOWEL MOVEMENTS ..... [ ][ ] DON'T KNOW 98
457	Was he/she given the same amount to drink as before the diarrhoea, or more, or less?	SAME ..... 1 MORE ..... 2 LESS ..... 3 DON'T KNOW 8	SAME ..... 1 MORE ..... 2 LESS ..... 3 DON'T KNOW 8
458	Was he/she given the same amount of food to eat as before the diarrhoea, or more, or less?	SAME ..... 1 MORE ..... 2 LESS ..... 3 DON'T KNOW 8	SAME ..... 1 MORE ..... 2 LESS ..... 3 DON'T KNOW 8
459	When (NAME) had diarrhoea, was he/she given any of the following to drink: A fluid, made from a special rehydration packet? Thin watery porridge? Soup? Home-made sugar-salt-water solution? Milk or infant formula? Yoghurt-based drink? Black Tea? Water? Coke? Any other liquid?	YES NO DK FLUID FROM ORS PKT 1 2 8 THIN WATERY PORRIDGE ..... 1 2 8 SOUP ..... 1 2 8 SUG.-SALT-WAT. SOL. 1 2 8 MILK/INFANT FORM. .. 1 2 8 YOGHURT-BASED DR. 1 2 8 BLACK TEA ..... 1 2 8 WATER ..... 1 2 8 COKE ..... 1 2 8 OTHER LIQUID 1 2 8	YES NO DK FLUID FROM ORS PKT 1 2 8 THIN WATERY PORRIDGE ..... 1 2 8 SOUP ..... 1 2 8 SUG.-SALT-WAT. SOL. 1 2 8 MILK/INFANT FORM. .. 1 2 8 YOGHURT-BASED DR. 1 2 8 BLACK TEA ..... 1 2 8 WATER ..... 1 2 8 COKE ..... 1 2 8 OTHER LIQUID 1 2 8
460	Was anything (else) given to treat the diarrhoea?	YES ..... 1 NO ..... 2 (SKIP TO 462) DON'T KNOW 8	YES ..... 1 NO ..... 2 (SKIP TO 462) DON'T KNOW 8
461	What was given to treat the diarrhoea? Anything else? RECORD ALL MENTIONED.	HOMEMADE SUGAR-SALT-WATER SOLUTION ..... A PILL OR SYRUP ..... B INJECTION ..... C (I.V.) INTRAVENOUS ..... D HOME REMEDIES/ HERBAL MEDICINES .... E OTHER _____ X (SPECIFY)	HOMEMADE SUGAR-SALT-WATER SOLUTION .... A PILL OR SYRUP ..... B INJECTION ..... C (I.V.) INTRAVENOUS ..... D HOME REMEDIES/ HERBAL MEDICINES ... E OTHER _____ X (SPECIFY)
462	Did you seek advice or treatment for the diarrhoea?	YES ..... 1 NO ..... 2 (SKIP TO 464)	YES ..... 1 NO ..... 2 (SKIP TO 464)

463	Where did you seek advice or treatment? Anywhere else? RECORD ALL MENTIONED.	PUBLIC SECTOR GOVT. HOSPITAL . . . . . A DAY HOSP/CLINIC/ COMMUNITY HEALTH CENTER . . . . . B MOBILE CLINIC . . . . . D COMM. HEALTH WORKER . . E OTHER PUBLIC _____ F (SPECIFY) PRIVATE MEDICAL SECTOR PVT. HOSPITAL/CLINIC . . . G PHARMACY . . . . . H PRIVATE DOCTOR . . . . . I OTHER PRIVATE MEDICAL _____ J (SPECIFY) OTHER SOURCE SHOP . . . . . K TRAD. HEALER . . . . . L OTHER _____ X (SPECIFY)	PUBLIC SECTOR GOVT. HOSPITAL . . . . . A DAY HOSP/CLINIC/ COMMUNITY HEALTH CENTER . . . . . B MOBILE CLINIC . . . . . D COMM. HEALTH WORKER . E OTHER PUBLIC _____ F (SPECIFY) PRIVATE MEDICAL SECTOR PVT. HOSPITAL/CLINIC . . . G PHARMACY . . . . . H PRIVATE DOCTOR . . . . . I OTHER PRIVATE MEDICAL _____ J (SPECIFY) OTHER SOURCE SHOP . . . . . K TRAD. HEALER . . . . . L OTHER _____ X (SPECIFY)
464		GO BACK TO 442 IN NEXT COLUMN; OR, IF NO MORE BIRTHS, GO TO 465.	GO BACK TO 442 IN NEXT COLUMN; OR, IF NO MORE BIRTHS, GO TO 465.

465	Now I am going to ask you some general questions about caring for children with diarrhoea and cough. When a child has diarrhoea, should he/she be given less to drink than usual, about the same amount, or more than usual?	LESS TO DRINK . . . . . 1 ABOUT SAME AMOUNT TO DRINK . 2 MORE TO DRINK . . . . . 3 DON'T KNOW 8	
466	When a child has diarrhoea, should he/she be given less to eat than usual, about the same amount, or more than usual?	LESS TO EAT . . . . . 1 ABOUT SAME AMOUNT TO EAT . . . 2 MORE TO EAT . . . . . 3 DON'T KNOW 8	
467	When a child is sick with diarrhoea, what signs of illness would tell you that he or she should be taken to a health facility or health worker? RECORD ALL MENTIONED. DO NOT PROBE	REPEATED WATERY STOOLS . . . . . A ANY WATERY STOOLS . . . . . B REPEATED VOMITING . . . . . C ANY VOMITING . . . . . D BLOOD IN STOOLS . . . . . E FEVER . . . . . F MARKED THIRST . . . . . G NOT EATING/NOT DRINKING WELL . H GETTING SICKER/VERY SICK . . . . . I NOT GETTING BETTER . . . . . J SUNKEN FONTANELLE . . . . . K OTHER _____ X (SPECIFY) DON'T KNOW . . . . . Z	
468	CHECK 459, ALL COLUMNS: NO CHILD RECEIVED ORS ☐ → ANY CHILD RECEIVED ORS ☐ →		→470
469	Have you ever heard of a special product called ORSOL OR SOROL that you can get for the treatment of diarrhoea?	YES . . . . . 1 NO 2	
470	When a child is sick with a cough, what signs of illness would tell you that he or she should be taken to a health facility or health worker? RECORD ALL MENTIONED.	FAST BREATHING . . . . . A DIFFICULT BREATHING . . . . . B NOISY BREATHING . . . . . C FEVER/HIGH TEMPERATURE . . . . . D UNABLE TO DRINK . . . . . E NOT EATING/NOT DRINKING WELL . F GETTING SICKER/VERY SICK . . . . . G NOT GETTING BETTER . . . . . H COUGHING A LOT . . . . . I OTHER _____ X (SPECIFY) DON'T KNOW . . . . . Z	

SECTION 5. MARITAL AND SEXUAL RELATIONS

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
501	PRESENCE OF OTHERS AT THIS POINT.	YES NO CHILDREN UNDER 10 . . . . . 1 2 HUSBAND/PARTNER . . . . . 1 2 OTHER MALES . . . . . 1 2 OTHER FEMALES 1 2	
	Now I am going to ask you some sensitive questions about your marital and sexual relations. All information you give me is completely confidential.		
502	Are you currently married or living with a man?	YES, CURRENTLY MARRIED . . . . . 1 YES, LIVING WITH A MAN . . . . . 2 NO, NOT IN UNION 3	→507
503	Do you currently have a regular sexual partner, an occasional sexual partner, or no sexual partner at all?	REGULAR SEXUAL PARTNER . . . . . 1 TWO OR MORE REGULAR PARTNERS . . . 2 OCCASIONAL SEXUAL PARTNER . . . . . 3 NO SEXUAL PARTNER 4	
504	Have you ever been married or lived with a man?	YES, FORMERLY MARRIED . . . . . 1 YES, LIVED WITH A MAN . . . . . 2 NO 3	→511 →514
506	What is your marital status now: are you widowed, divorced, or separated?	WIDOWED . . . . . 1 DIVORCED . . . . . 2 SEPARATED 3	→511
507	Is your husband/partner living with you now or is he staying elsewhere?	LIVING WITH HER . . . . . 1 STAYING ELSEWHERE 2	
508	Does your husband have any other wives besides yourself?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→511
509	How many other wives does he have?	NUMBER OF OTHER WIVES . . . . [ ][ ] DON'T KNOW 98	
511	Have you been married or lived with a man only once, or more than once?	ONCE . . . . . 1 MORE THAN ONCE 2	
512	CHECK 511: MARRIED/LIVED WITH A MAN ONLY ONCE [ ] In what month and year did you start living with your husband/partner? MARRIED/LIVED WITH A MAN MORE THAN ONCE [ ] Now we will talk about your first husband/partner. In what month and year did you start living with him?	MONTH . . . . . [ ][ ] DON'T KNOW MONTH . . . . . 98 YEAR . . . . . [ ][ ][ ][ ] DON'T KNOW YEAR 9998	→514
513	How old were you when you started living with him?	AGE . . . . . [ ][ ]	
514	How old were you when you had your first period?	AGE . . . . . [ ][ ]	
515	Now I need to ask you some questions about sexual activity in order to gain a better understanding of some health and family planning issues. When was the last time you had sexual intercourse (if ever)?	NEVER . . . . . 000 DAYS AGO . . . . . 1 [ ][ ] WEEKS AGO . . . . . 2 [ ][ ] MONTHS AGO . . . . . 3 [ ][ ] YEARS AGO . . . . . 4 [ ][ ] BEFORE LAST BIRTH 996	→608 →517

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
515A	Can you describe your relationship with the person you last had sexual intercourse with?	MARITAL PARTNER . . . . . 01 OTHER REGULAR PARTNER . . . . . 02 CASUAL ACQUAINTANCE . . . . . 03 SOMEONE JUST MET . . . . . 04 COMMERCIAL SEX WORKER . . . . . 05 OTHER _____ . . . . . 96 (SPECIFY)	
516	CHECK 301 AND 302: KNOWS CONDOM ☐ → The last time you had sex, was a condom used? DOES NOT KNOW CONDOM ☐ → Some men use a condom, which means that they put a rubber sheath on their penis during sexual intercourse. The last time you had sex, was a condom used?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→516B →516B
516A	If not, what are the reasons why you didn't use one? RECORD ALL MENTIONED What is the Main Reason?	WANTS CHILDREN . . . . . A PERCEIVED LOW OR NO RISK OF STD/HIV . . . . . B RESPONDENT DISLIKE . . . . . C PARTNER DISLIKE . . . . . D CULTURAL/RELIGIOUS PROHIBITION . . . . . E DID NOT KNOW CONDOMS . . . . . F DID NOT KNOW HOW TO USE CONDOM . . . . . G BAD PREVIOUS EXPERIENCE WITH CONDOM . . . . . H INCONVENIENT TO USE . . . . . I LACK OF SPONTANEITY . . . . . J DID NOT KNOW SOURCE OF CONDOMS . . . . . K EMBARRASSED TO GET . . . . . L INCONVENIENT TO GET . . . . . M DIDN'T HAVE A CONDOM . . . . . N COST TOO MUCH . . . . . O NO/LESS SENSATION WITH CONDOM . . . . . P SUGGESTS LACK OF TRUST OF PARTNER . . . . . Q SUGGESTS LACK OF LOVE OF PARTNER . . . . . R FEAR OF LOSING IT INSIDE . . . . . S WASTES SPERM . . . . . T RUBBER SMELL . . . . . U PARTNER OR SELF HAS BURNING/ DISCOMFORT WHEN USING CONDOM . . . . . V PREFER SEX 'FLESH TO FLESH' . . . . . W CONDOM USE NOT COOL/MANLY/ TRENDY . . . . . Y OTHER _____ X (SPECIFY) DON'T KNOW . . . . . Z ☐	
516B	In the last 12 months, with how many different men have you had sexual intercourse?	NUMBER . . . . . ☐☐	
517	Do you know of a place where you can get condoms?	YES . . . . . 1 NO 2	→519

518	Where is that? IF SOURCE IS HOSPITAL, HEALTH CENTER, OR CLINIC, WRITE THE NAME OF THE PLACE. PROBE TO IDENTIFY THE TYPE OF SOURCE AND CIRCLE THE APPROPRIATE CODE. _____ (NAME OF PLACE)	PUBLIC SECTOR GOVERNMENT HOSPITAL . . . . . 11 DAY HOSP/CLINIC COMMUNITY HEALTH CENTER . . . . 12 FAMILY PLANNING CLINIC . . . . . 13 MOBILE CLINIC . . . . . 14 COMMUNITY HEALTH WORKER . . 15 OTHER PUBLIC . . . . . 16 _____ (SPECIFY) PRIVATE MEDICAL SECTOR PRIVATE HOSPITAL/CLINIC . . . . . 21 PHARMACY . . . . . 22 PRIVATE DOCTOR . . . . . 23 OTHER PRIVATE MEDICAL . . . . . 26 _____ (SPECIFY) OTHER SOURCE SHOP . . . . . 31 CHURCH . . . . . 32 FRIENDS/RELATIVES . . . . . 33 OTHER_____ 36 (SPECIFY)	
519	How old were you when you first had sexual intercourse?	AGE . . . . . [ ][ ] FIRST TIME WHEN MARRIED 96	

SECTION 6. FERTILITY PREFERENCES

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
601	CHECK 314: NEITHER STERILIZED ☐ → HE OR SHE STERILIZED ☐		→612
602	CHECK 234: NOT PREGNANT OR UNSURE ☐ → Now I have some questions about the future. Would you like to have (a/another) child, or would you prefer not to have any (more) children? PREGNANT ☐ → Now I have some questions about the future. After the child you are expecting now, would you like to have another child, or would you prefer not to have any more children?	HAVE (A/ANOTHER) CHILD . . . . . 1 NO MORE/NONE . . . . . 2 SAYS SHE CAN'T GET PREGNANT . . 3 UNDECIDED/DON'T KNOW 8	→604 →606 →604
603	CHECK 234: NOT PREGNANT OR UNSURE ☐ → How long would you like to wait from now before the birth of (a/another) child? PREGNANT ☐ → After the child you are expecting now, how long would you like to wait before the birth of another child?	MONTHS . . . . . 1 ☐☐ YEARS . . . . . 2 ☐☐ SOON/NOW . . . . . 993 SAYS SHE CAN'T GET PREGNANT 994 AFTER MARRIAGE . . . . . 995 OTHER _____ 996 (SPECIFY) DON'T KNOW 998	→606
604	CHECK 234: NOT PREGNANT OR UNSURE ☐ → PREGNANT ☐		→607
605	If you became pregnant in the next few weeks, would you be happy, unhappy, or would it not matter very much?	HAPPY . . . . . 1 UNHAPPY . . . . . 2 WOULD NOT MATTER 3	
606	CHECK 313: USING A METHOD? NOT ASKED ☐ → NOT CURRENTLY USING ☐ → CURRENTLY USING ☐		→612
607	Do you think you will use a method to delay or avoid pregnancy within the next 12 months?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→609
608	Do you think you will use a method to delay or avoid pregnancy at any time in the future?	YES . . . . . 1 NO . . . . . 2 DON'T KNOW 8	→610
609	Which method would you prefer to use?	PILL . . . . . 01 IUD . . . . . 02 INJECTIONS . . . . . 03 DIAPHRAGM/FOAM/JELLY . . . . . 04 CONDOM . . . . . 05 FEMALE STERILIZATION . . . . . 06 MALE STERILIZATION . . . . . 07 CALENDER/RHYTHM . . . . . 08 WITHDRAWAL . . . . . 09 HERB/REMEDIES . . . . . 10 OTHER _____ 96 (SPECIFY) UNSURE 98	→612

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
610	What is the main reason that you think you will never use a method?	FERTILITY-RELATED REASONS INFREQUENT SEX . . . . . 22 MENOPAUSAL/HYSTERECTOMY 23 INFERTILE . . . . . 24 WANTS MORE CHILDREN . . . . . 26 OPPOSITION TO USE RESPONDENT OPPOSED . . . . . 31 HUSBAND/PARTNER OPPOSED . 32 OTHERS OPPOSED . . . . . 33 RELIGIOUS PROHIBITION . . . . . 34 LACK OF KNOWLEDGE KNOWS NO METHOD . . . . . 41 KNOWS NO SOURCE . . . . . 42 METHOD-RELATED REASONS HEALTH CONCERNS . . . . . 51 FEAR OF SIDE EFFECTS . . . . . 52 LACK OF ACCESS/TOO FAR . . . . 53 COST TOO MUCH . . . . . 54 INCONVENIENT TO USE . . . . . 55 INTERFERES WITH BODY'S NORMAL PROCESSES . . . . . 56 OTHER _____ 96 (SPECIFY) DON'T KNOW 98	
612	CHECK 216: HAS LIVING CHILDREN ☐ → If you could go back to the time you did not have any children and could choose exactly the number of children to have in your whole life, how many would that be? NO LIVING CHILDREN ☐ → If you could choose exactly the number of children to have in your whole life, how many would that be? PROBE FOR A NUMERIC RESPONSE.	NUMBER . . . . . ▒▒ OTHER _____ 96 (SPECIFY)	
614	Would you say that you approve or disapprove of couples using a method to avoid getting pregnant?	APPROVE . . . . . 1 DISAPPROVE . . . . . 2 NO OPINION 3	
615	Is it acceptable or not acceptable to you for information on family planning to be provided: On the radio? On the television?	ACCEPTABLE / NOT ACCEPTABLE / DK RADIO . . . . . 1 2 8 TELEVISION 1 2 8	
616	In the last few months have you heard about family planning and sterilization: On the radio? On the television? In a newspaper or magazine? From a poster? From leaflets or brochures?	YES / NO RADIO . . . . . 1 2 TELEVISION . . . . . 1 2 NEWSPAPER OR MAGAZINE . 1 2 POSTER . . . . . 1 2 LEAFLETS OR BROCHURES . 1 2	
618	In the last few months have you discussed the practice of family planning with your friends, neighbours, or relatives?	YES . . . . . 1 NO 2	→ 620
619	With whom? Anyone else? RECORD ALL MENTIONED.	HUSBAND/PARTNER . . . . . A MOTHER . . . . . B FATHER . . . . . C SISTER(S) . . . . . D BROTHER(S) . . . . . E DAUGHTER . . . . . F MOTHER-IN-LAW . . . . . G FRIENDS/NEIGHBOURS . . . . . H OTHER _____ X (SPECIFY)	

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
620	CHECK 502: YES, CURRENTLY MARRIED • ☐ YES, LIVING WITH A MAN • ☐ NO, NOT IN UNION ☐		→701
621	Spouses/partners do not always agree on everything. Now I want to ask you about your husband's/partner's views on family planning. Do you think that your husband/partner approves or disapproves of couples using a method to avoid pregnancy?	APPROVES . . . . . 1 DISAPPROVES . . . . . 2 DON'T KNOW 8	
622	How often have you talked to your husband/partner about family planning in the past year?	NEVER . . . . . 1 ONCE OR TWICE . . . . . 2 MORE OFTEN 3	
623	Do you think your husband/partner wants the same number of children that you want, or does he want more or fewer than you want?	SAME NUMBER . . . . . 1 MORE CHILDREN . . . . . 2 FEWER CHILDREN . . . . . 3 DON'T KNOW 8	
624	Who makes the decisions about using methods to avoid pregnancy?	REPONDENT DECIDES . . . . . 01 HUSBAND/PARTNER DECIDES . . . . 02 JOINTLY . . . . . 03 OTHER _____ 96 (SPECIFY)	

SECTION 7: TREATMENT OF WOMEN IN THE HOUSEHOLD

Now I would like to ask you some difficult questions about how you have been treated in your life by other people.

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
701	CHECK 502: MARRIED, LIVING WITH A MAN ☐	NO UNION ☐	→703
702	Within the last year, has your partner/husband regularly not provided money you need for food, rent or bills but has money for other things?	YES . . . . . 1 NO 2	
703	Over the last year, has anyone ever kicked, bitten, slapped, hit you with a fist, threaten you with a weapon, such as a knife, a stick, or a gun, or thrown something at you?	YES . . . . . 1 NO . . . . . 2 NO ANSWER 3	→705
704	Have any of your boyfriends or husbands ever kicked, bitten, slapped, hit you with a fist, threaten you with a weapon, such as a knife, a stick, or a gun, or thrown something at you?	YES . . . . . 1 NO . . . . . 2 NO ANSWER 3	→707 →712
705	Can you tell me who has done this to you? Anyone else? RECORD ALL MENTIONED PROBE IF NOT MENTIONED	CURRENT HUSBAND/PARTNER . . . . . A FORMER HUSBAND/PARTNER . . . . . B BOYFRIEND . . . . . C FATHER . . . . . D BROTHER . . . . . E SON . . . . . F DAUGHTER . . . . . G MOTHER . . . . . H FATHER-IN-LAW . . . . . I MOTHER-IN-LAW . . . . . J OTHER MALE RELATIVE . . . . . K OTHER FEMALE RELATIVE . . . . . L MANAGER/FOREMAN/EMPLOYER . . . . M ASSAILANT . . . . . N OTHER _____ X (SPECIFY) NO ANSWER . . . . . Y	
706	Who is the person who did or does beat you most often?	CURRENT HUSBAND/PARTNER . . . . . 01 FORMER HUSBAND/PARTNER . . . . . 02 BOYFRIEND . . . . . 03 FATHER . . . . . 04 BROTHER . . . . . 05 SON . . . . . 06 DAUGHTER . . . . . 07 MOTHER . . . . . 08 FATHER-IN-LAW . . . . . 09 MOTHER-IN-LAW . . . . . 10 OTHER MALE RELATIVE . . . . . 11 OTHER FEMALE RELATIVE . . . . . 12 MANAGER/FOREMAN/EMPLOYER . . . . 13 OTHER _____ 96 (SPECIFY) NO ANSWER 98	
707	Is or was this person always, sometimes or never "on something" (drugs or alcohol) when he/she did this to you?	ALWAYS . . . . . 1 SOMETIMES . . . . . 2 NEVER . . . . . 3 NO ANSWER 8	

708	In the past one year, approximately how many times did this happen to you? IF NONE WRITE '00'	TIMES . . . . . ▒▒ NO ANSWER 96	
709	Have you ever left a husband/partner because you were being beaten?	YES . . . . . 1 NO 2	
710	When you were pregnant, has anyone ever kicked, bitten, slapped, hit you with a fist, threaten you with a weapon, such as a knife, a stick, or a gun, or thrown something at you?	YES . . . . . 1 NO . . . . . 2 NEVER BEEN PREGNANT 3	
711	In the past year, have you ever been so seriously hurt during a beating that you needed medical attention even if you did not see a doctor?	YES . . . . . 1 NO . . . . . 2 NO ANSWER 3	
712	Has anyone ever forced you to have sexual intercourse against your will by threatening, holding you down or hurting you in some way?	YES . . . . . 1 NO 2	→715
713	Has anyone ever persuaded you to have sexual intercourse when you did not want to?	YES . . . . . 1 NO 2	→718
715	Did this happen before you were 15 years old?	YES . . . . . 1 NO . . . . . 2 NO ANSWER 3	→718
716	How old were you when this first happened?	AGE . . . . . ▒▒	
717	Who did this to you?	FATHER . . . . . 01 OTHER MALE RELATIVE . . . . . 02 BROTHER . . . . . 03 FAMILY FRIEND/LODGER . . . . . 04 LANDLORD/FARMER . . . . . 05 SCHOOL TEACHER/PRINCIPAL . . . . . 06 MAN/BOY FROM NEIGHBOURHOOD/ SCHOOL/CHURCH . . . . . 07 MANAGER/FOREMAN/EMPLOYER . . . . 08 STEPFATHER/MOTHER'S BOYFRIEND 09 BOYFRIEND/HUSBAND . . . . . 10 STRANGER/RECENT ACQUAINTANCE 11 OTHER _____ 96 (SPECIFY)	→724
718	Before you were 15 years old, did any man touch you against your will in a sexual way, such as unwanted touching, kissing, grabbing or fondling?	YES . . . . . 1 NO . . . . . 2 NO ANSWER 3	→721
719	How old were you when this first happened?	AGE . . . . . ▒▒	
720	Who did this to you?	FATHER . . . . . 01 OTHER MALE RELATIVE . . . . . 02 BROTHER . . . . . 03 FAMILY FRIEND/LODGER . . . . . 04 LANDLORD/FARMER . . . . . 05 SCHOOL TEACHER/PRINCIPAL . . . . . 06 MAN/BOY FROM NEIGHBOURHOOD/ SCHOOL/CHURCH . . . . . 07 MANAGER/FOREMAN/EMPLOYER . . . . 08 STEPFATHER/MOTHER'S BOYFRIEND 09 BOYFRIEND/HUSBAND . . . . . 10 STRANGER/RECENT ACQUAINTANCE 11 OTHER _____ 96 (SPECIFY)	

721	Before you were 15 years old, did any man force you to touch his private parts against your will?	YES ..... 1 NO ..... 2 NO ANSWER 8	→724
722	How old were you when this first happened	AGE ..... [ ][ ]	
723	Who did this to you?	FATHER ..... 01 OTHER MALE RELATIVE ..... 02 BROTHER ..... 03 FAMILY FRIEND/LODGER ..... 04 LANDLORD/FARMER ..... 05 SCHOOL TEACHER/PRINCIPAL ..... 06 MAN/BOY FROM NEIGHBOURHOOD/ SCHOOL/CHURCH ..... 07 MANAGER/FOREMAN/EMPLOYER .... 08 STEPFATHER/MOTHER'S BOYFRIEND 09 BOYFRIEND/HUSBAND ..... 10 STRANGER/RECENT ACQUAINTANCE 11 OTHER _____ 96 (SPECIFY)	
724	Have you tried to get help from services of any kind because of beatings or other bad treatment?	YES ..... 1 NO 2	→726
725	What do or did you use?	SHELTER ..... A COUNSELLING ..... B WOMEN'S CENTRE ..... C SOCIAL WORKER ..... D POLICE ..... E CLINIC/HOSPITAL ..... F OTHER _____ X (SPECIFY)	
726	Would you have liked to have had help from a service that was not available?	YES ..... 1 NO 2	→801
727	What service would have been helpful to you?	SHELTER ..... A COUNSELLING ..... B WOMEN'S CENTRE ..... C SOCIAL WORKER ..... D POLICE ..... E CLINIC/HOSPITAL ..... F OTHER _____ X (SPECIFY)	

SECTION 8: AIDS

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES				SKIP
801	Have you ever heard of an illness called AIDS?	YES ..... 1 NO 2				→901
802	How much information about HIV/AIDS did you obtain from each of the following sources: Answer each question with a lot, some or none		A LOT	SOME	NONE	
	a) TV?	TV	1	2	3	
	b) Radio?	RADIO	1	2	3	
	c) Newspaper?	NEWSPAPER	1	2	3	
	d) Pamphlets?	PAMPHLETS	1	2	3	
	e) Health Workers?	HEALTH WORKERS	1	2	3	
	f) Friends?	FRIENDS	1	2	3	
	g) Partner(s)?	PARTNER(S)	1	2	3	
	h) Relatives?	RELATIVES	1	2	3	
803	I am going to read out some statements about protection against HIV/AIDS. For each statement, please tell me whether you think it is true or not. People can protect themselves from HIV/AIDS by:		TRUE	NOT TRUE	DON'T KNOW	
	a) having a good diet	DIET	1	2	8	
	b) staying with one faithful partner	FAITHFUL	1	2	8	
	c) avoiding public toilets	AVOID TOILETS	1	2	8	
	d) using condoms during sexual intercourse	CONDOMS	1	2	8	
	e) avoiding touching a person who has AIDS	AVOID TOUCH	1	2	8	
	f) avoiding sharing food with a person who has AIDS	AVOID SHARED FOOD	1	2	8	
	g) avoiding being bitten by mosquitos or similar insects	AVOID MOSQUITOS	1	2	8	
	h) making sure any injection they have is done with a clean needle	CLEAN INJECTION	1	2	8	
	I) avoid sharing razor blades	AVOID RAZOR	1	2	8	
804	Do you think that a person infected with the AIDS virus always shows symptoms or can such a person look perfectly healthy?	ALWAYS SHOWS SYMPTOMS ..... 1 CAN LOOK HEALTHY ..... 2 DON'T KNOW 8				
804A	I am going to ask you some questions about the need for people to be informed about their HIV/AIDS status:		TRUE	NOT TRUE	DON'T KNOW	
	a) should people with AIDS be told about their status?	TOLD ABOUT AIDS	1	2	8	
	b) should people diagnosed HIV positive be told about their status?	TOLD ABOUT HIV	1	2	8	
	c) should HIV/AIDS patients tell their partner(s) about their status?	TELL PARTNERS	1	2	8	
	d) should the reporting of AIDS status to health authorities be made mandatory by law?	REPORT AIDS	1	2	8	
	e) should the reporting of HIV status to health authorities be made mandatory by law?	REPORT HIV	1	2	8	
805	Do you personally know someone who has been diagnosed with HIV/AIDS or who has died of AIDS?	YES ..... 1 NO 2				→901
805A	How much assistance and support do you think AIDS patients receive from each of the following: Answer the questions with a lot, some or none.		ALOT	SOME	NONE	
	a) employers?	A) EMPLOYERS	1	2	3	
	b) co-workers?	B) CO-WORKERS	1	2	3	
	c) insurance companies?	C) INSURANCE C.	1	2	3	
	d) health workers?	D) HEALTH WORKERS	1	2	3	
	e) friends?	E) FRIENDS	1	2	3	
	f) partner(s)?	F) PARTNER(S)	1	2	3	
	g) relatives?	G) RELATIVES	1		23	

SECTION 9 - MATERNAL MORTALITY

No.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
901	Now I would like to ask some questions about your brothers and sisters, that is, all of the children born to your natural mother, including those who are living with you, those living elsewhere and those who have died. How many children did your mother give birth to, including you?		
	NUMBER OF BIRTHS TO NATURAL MOTHER	☐☐	
902	CHECK 901: TWO OR MORE BIRTHS ☐ ↓ ONLY ONE BIRTH (RESPONDENT ONLY) ☐ → 1001		
903	How many of these births did your mother have before you were born?	NUMBER OF PRECEDING BIRTHS ☐☐	

904 What was the name given to your eldest (next oldest) brother or sister?	905 Is (NAME) male or female?	906 Is (NAME) still alive?	907 How old is (NAME)?	908 In what year did (NAME) die?	909 How many years ago did (NAME) die?	910 How old was (NAME) when she/he died?
[1] _____	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 ↵ GO TO 908 DK . . . . . 8 ↵ GO TO [2]	☐☐ GO TO [2]	☐☐☐☐ GO TO 910 ↵ DK 9998	☐☐	☐☐ IF MALE OR DIED BEFORE AGE 12 GO TO [2]
[2] _____	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 ↵ GO TO 908 DK . . . . . 8 ↵ GO TO [3]	☐☐ GO TO [3]	☐☐☐☐ GO TO 910 ↵ DK 9998	☐☐	☐☐ IF MALE OR DIED BEFORE AGE 12 GO TO [3]
[3] _____	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 ↵ GO TO 908 DK . . . . . 8 ↵ GO TO [4]	☐☐ GO TO [4]	☐☐☐☐ GO TO 910 ↵ DK 9998	☐☐	☐☐ IF MALE OR DIED BEFORE AGE 12 GO TO [4]
[4] _____	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 ↵ GO TO 908 DK . . . . . 8 ↵ GO TO [5]	☐☐ GO TO [5]	☐☐☐☐ GO TO 910 ↵ DK 9998	☐☐	☐☐ IF MALE OR DIED BEFORE AGE 12 GO TO [5]
[5] _____	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 ↵ GO TO 908 DK . . . . . 8 ↵ GO TO [6]	☐☐ GO TO [6]	☐☐☐☐ GO TO 910 ↵ DK 9998	☐☐	☐☐ IF MALE OR DIED BEFORE AGE 12 GO TO [6]
[6] _____	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 ↵ GO TO 908 DK . . . . . 8 ↵ GO TO [7]	☐☐ GO TO [7]	☐☐☐☐ GO TO 910 ↵ DK 9998	☐☐	☐☐ IF MALE OR DIED BEFORE AGE 12 GO TO [7]

911 Was (NAME) pregnant when she died?	912 Did (NAME) die during childbirth?	913 Did (NAME) die within two months after the end of a pregnancy or childbirth?	914 Was her death due to complications of pregnancy or childbirth?	915 How many children did (NAME) give birth to during her lifetime?
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [2]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [3]
YES 1 GOT TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [4]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [5]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [6]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [7]

904 What was the name given to your eldest (next oldest) brother or sister?	905 Is (NAME) male or female?	906 Is (NAME) still alive?	907 How old is (NAME)?	908 In what year did (NAME) die?	909 How many years ago did (NAME) die?	910 How old was (NAME) when she/he died?
[7]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [8]	GO TO [8]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [8]
[8]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [9]	GO TO [9]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [9]
[9]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [10]	GO TO [10]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [10]
[10]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [11]	GO TO [11]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [11]
[11]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [12]	GO TO [12]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [12]
[12]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [13]	GO TO [13]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [13]
[13]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [14]	GO TO [14]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [14]
[14]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [15]	GO TO [15]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [15]
[15]	MALE . . . . . 1 FEMALE . . . 2	YES . . . . . 1 NO . . . . . 2 GO TO 908 DK . . . . . 8 GO TO [16]	GO TO [16]	GO TO 910 DK 9998		IF MALE OR DIED BEFORE AGE 12 GO TO [16]

911 Was (NAME) pregnant when she died?	912 Did (NAME) die during childbirth?	913 Did (NAME) die within two months after the end of a pregnancy or childbirth?	914 Was her death due to complications of pregnancy or childbirth?	915 How many children did (NAME) give birth to during her lifetime?
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [8]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [9]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [10]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [11]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [12]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [13]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [14]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [15]
YES 1 GO TO 914 NO 2	YES . . . . . 1 GO TO 915 NO 2	YES . . . . . 1 NO . . . . . 2 GO TO 915	YES . . . . . 1 NO 2	GO TO [16]

SECTION 10. HUSBAND'S BACKGROUND, WOMAN'S WORK AND RESIDENCE

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
1001	CHECK 502 AND 504: CURRENTLY MARRIED/ LIVING WITH A MAN ▪ FORMERLY MARRIED/ LIVED WITH A MAN ▪ NEVER MARRIED AND NEVER IN UNION ▪		→1003 →1009
1002	How old was your husband/partner on his last birthday?	AGE	
1003	Did your (last) husband/partner ever attend school?	YES ..... 1 NO 2	→1005
1004	What was the highest (standard/year) he completed at school?	LESS THAN 1 YEAR COMPLETED . 00 SUB A/CLASS 1 ..... 71 SUB B/CLASS 2 ..... 72 STANDARD 1 ..... 01 STANDARD 2 ..... 02 STANDARD 3 ..... 03 STANDARD 4 ..... 04 STANDARD 5 ..... 05 STANDARD 6 ..... 06 STANDARD 7 ..... 07 STANDARD 8 ..... 08 STANDARD 9 ..... 09 STANDARD 10 ..... 10 FURTHER STUDIES INCOMPLETE . 11 DIPLOMA/OTHER POSTSCHOOL COMPLETE ..... 12 FURTHER DEGREE COMPLETE .. 13 DON'T KNOW 98	
1005	Does your husband/partner currently work?	YES ..... 1 NO ..... 2 DON'T KNOW 8	
1006	What (is/was) your (last) husband/partner's occupation? That is, what kind of work (does/did) he mainly do?		
1009	Aside from your own housework, are you currently working for money?	YES ..... 1 NO 2	→101 2
1010	As you know, some women take up jobs for which they are paid in cash or kind. Others sell things, have a small business or work on the family farm or in the family business. Are you currently doing any of these things or any other work?	YES ..... 1 NO 2	→101 2
1011	Have you done any work in the last 12 months?	YES ..... 1 NO 2	→102 6
1012	What is your occupation, that is, what kind of work do you mainly do?		
1015	Do you do this work for a family business, are you employed by someone outside the family or are you self-employed?	FOR FAMILY MEMBER ..... 1 FOR SOMEONE ELSE ..... 2 SELF-EMPLOYED 3	
1016	Do you usually work throughout the year, or do you work seasonally, or only once in a while?	THROUGHOUT THE YEAR ..... 1 SEASONALLY/PART OF THE YEAR . 2 ONCE IN A WHILE 3	→1018 →1019

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP
1017	During the last 12 months, how many months did you work?	NUMBER OF MONTHS ... [ ][ ]	
1018	During the last 12 months, how many days a week did you usually work (in the months that you worked)?	NUMBER OF DAYS ..... [ ]	→1020
1019	During the last 12 months, approximately how many days did you work?	NUMBER OF DAYS .. [ ][ ][ ]	
1020	Do you earn cash for your work? PROBE: Do you make money for working?	YES ..... 1 NO 2	→1023
1021	How much do you usually earn for this work? PROBE: Is this by the day, by the week, or by the month?	PER HOUR 1 [ ][ ][ ][ ][ ][ ] PER DAY . 2 [ ][ ][ ][ ][ ][ ] PER WEEK 3 [ ][ ][ ][ ][ ][ ] PER MONTH4 [ ][ ][ ][ ][ ][ ] PER YEAR 5 [ ][ ][ ][ ][ ][ ] OTHER _____ 999996 (SPECIFY)	
1022	CHECK 502: YES, CURRENTLY MARRIED YES, LIVING WITH A MAN Who mainly decides how the money you earn will be used: you, your husband/partner, you and your husband/partner jointly, or someone else? O, NOT IN UNION Who mainly decides how the money you earn will be used: you, someone else, or you and someone else jointly?	RESPONDENT DECIDES ..... 1 HUSBAND/PARTNER DECIDES .... 2 JOINTLY WITH HUSBAND/PARTNER 3 SOMEONE ELSE DECIDES ..... 4 JOINTLY WITH SOMEONE ELSE ... 5	
1023	Do you usually work at home or away from home?	HOME ..... 1 AWAY 2	
1024	CHECK 222 AND 223: IS A CHILD LIVING AT HOME WHO IS AGE 5 OR LESS? YES [ ] NO [ ]		→1026
1025	Who usually takes care of (NAME OF YOUNGEST CHILD AT HOME) while you are working?	RESPONDENT ..... 01 HUSBAND/PARTNER ..... 02 OLDER FEMALE CHILD ..... 03 OLDER MALE CHILD ..... 04 OTHER RELATIVES ..... 05 NEIGHBORS ..... 06 FRIENDS ..... 07 SERVANTS/HIRED HELP ..... 08 CHILD IS IN SCHOOL ..... 09 INSTITUTIONAL CHILD CARE ..... 10 HAS NOT WORKED SINCE LAST BIRTH ..... 95 OTHER _____ 96 (SPECIFY)	
1026	RECORD THE TIME	HOURS [ ][ ] MINUTES [ ][ ]	

INTERVIEWERS OBSERVATION

Comments about the respondent/s: _____

_____

_____

_____

Comments on Specific Questions: _____

_____

_____

_____

Any other comments:

_____

_____

_____

_____

SUPERVISOR'S OBSERVATION

_____

_____

_____

_____

Name of Supervisor: _____ Date:_____

EDITOR'S OBSERVATIONS

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_____

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Name of Editor:_____ Date:_____

W-ENG41

Appendix 4

UNIVERSITY OF PRETORIA ETHICS COMMITTEE APPROVAL LETTER

FWA Nr 0000 2567
IRB Nr. 0000 2235

University of Pretoria

Sculpansberg Road
MRC-Building
Room 2 - 19

Private Bag x 323
Pretoria
0001

Faculty of Health Sciences Research Ethics Committee
University of Pretoria
Date: 4/07/2005

Number	:	77/2005
Title	:	Probing secondary exposure and health data as a tool to improve public health in South Africa.
Investigators	:	Janine Wichman, School of Health Systems and Public Health, University of Pretoria jwichmann@postmed.up.ac.za
Sponsor	:	NRF

This Protocol and Informed Consent have been considered by the Faculty of Health Sciences Research Ethics Committee, University of Pretoria on 29/06/2005 and found to be acceptable.

Advocate AG Nienaber	(female)BA(Hons) (Wits); LLB, LLM (UP): Dipl Datametrics (UNISA)
Prof S V Grey	(female) BSc (Hons); MSc; DSc: Deputy Dean
*Prof V O L Karusseit	MBChB; MFGP (SA); M Med (Chir); FCS (SA): Surgeon
Dr M F Kenoshi	MB,CHB; DTM & H (Wits); C E.O of the Pretoria Academic Hospital
*Prof M Kruger	(female) MB ChB (Pret); Mmed Paed.(Pret); PhDd (Leuven)
*Dr N K Likibi	MB BCh ; Med Adviser (Gauteng Dept of Health)
Dr F M Mulaudzi	(female) Department of Nursing,
Snr Sr J. Phatoli	(female) BCur (Et.Al) Senior Nursing-Sister
*Dr L Schoeman	(female) Bpharm, BA Hons (Psy), PhD
Prof I I W. Pretorius	MBChB; M Med (Psych) MD: Psychiatrist
Prof J R Snyman	MBChB, M Pharm Med; MD: Pharmacologist
*Dr R Sommers	(female) MBChB; M Med (Int); MPhar.Med;
Prof TJP Swart	BChD, MSc (Odont), MChD (Oral Path) Senior Specialist: Oral Pathology
Prof C W van Staden	MBChB; Mmed (Psych); MD; FTCL; UPLM: Dept of Psychiatry

DR R SOMMERS; MBChB; M Med (Int); MPhar Med
SECRETARIAT of the Faculty of Health Sciences Research Ethics Committee - University of Pretoria

* Members attended the meeting on 29/06/2005

ETHICAL APPROVAL OF 1998 SOUTH AFRICAN DEMOGRAPHIC AND HEALTH SURVEY BY MEDICAL RESEARCH COUNCIL

Office of the President

PO Box 19070, TYGERBERG 7505, Republic of South Africa • Francie van Zijl Drive, Parowvalley, Cape Town
Tel: +27 21 938-0211/938-0911 • Fax: +27 21 938-0301
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MRC

'AL RESEARCH COUNCIL

MEDIESE)RSINGSRAAD

MKHANDLU KUCWANINGA ZOKWELAPHA

KGOTLA LA 'HUPUTSO TSA KALAFO

15 December 1997

Dr D Bradshaw
SADHS Project Team Leader
CERSA
MRC

Dear Dr Bradshaw,

South African Demographic and Health Survey

The Committee, at its meeting on 1 December 1997, approved the study, with the following comments:

> The length of the questionnaire would be closer to 1,5 or 2 hours rather than 30 minutes. This should be indicated on the consent form; and it could also impact on the cost. Furthermore, interviews must preferably be done privately.

Wishing you well with your research.

Yours sincerely,

PROF PC BELONJE
CHAIRPERSON: MRC ETHICS COMMITTEE

DATA USE AGREEMENT LETTER FROM NATIONAL DEPARTMENT OF HEALTH

GW 1/16

Ref.

TO: School of Health Systems and Public Health University of Pretoria
CSIR Campus, Building 22, Room A126
Meiring Naude Road
Pretoria
0001

FROM RIKA DU PLESSIS, CLUSTER: HEALTH INFORMATION, EVALUATION AND RESEARCH/ HEALTH MONITORING AND EVALUATION

Enquiries: RIKA DU PLESSIS

Your ref.

Telephone (012) 312-0776

Fax (012) 312-0503

RE: Use of 1998 SADHS data

Ms. J Wichmann

As per your emails.

I would hereby like to confirm that you may use the data as per the data user's agreement, please could you provide the Department with a copy of the documentation when the research is completed.

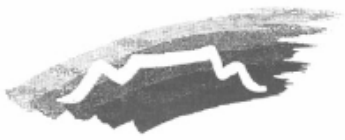
The data for the 1998 SADHS was provided without any cost to Ms. J Wichmann

Regards

Ms. H L du Plessis
HEALTH INFORMATION, EVALUATION AND RESEARCH/HEALTH MONITORING AND EVALUATION
DATE: 6 June 2005

AIDS HELPLINE
0800-01-23-22

DATA USE AGREEMENT LETTER FROM CITY OF CAPE TOWN



CITY OF CAPE TOWN | ISIXEKO SASEKAPA | STAD KAAPSTAD

Vasco Clinic
246 Voortrekker Road
Vasco 7460
P O Box 16548, Cape Town 8000
Ask for: Mr H Linde
Tel: 021 590-1419
Cell: 084 222 1486
Fax: 021 590-1621
E-mail: hans.linde@capetown.gov.za
Webmail:
Ref: kc
Filename: G:\APollution-Shared\STAFF DOCUMENTS\Hans Linde\Letters and Notices\Letters - 2005\Janine Wichmann'.doc

Vasco Clinic
246 Voortrekker Road
Vasco 7460
P O Box 16548, Cape Town 8000
Cela: Mnu H Linde
Umxeba: 021 590-1419
Cell: 084 222 1486
Iteksi: 021 590-1621

Vasco Kliniek
Voortrekkerweg 246
Vasco 7460
Posbus 16548, Kaapstad 8000
Vra vir: Mnr H Linde
Tel: 021 590-1419
Sel: 084 222 1486
Faks: 021 590-1621

COMMUNITY SERVICES — City Health — Air Pollution Control

2005-06-06

To Whom It May Concern:

The City of Cape Town Air Pollution Control Section has agreed that the student, Janine Wichmann, PhD Student: Environmental Health, School of Health Systems and Public Health, University of Pretoria, CSIR Campus, Building 22, Room A126, Meiring Naude Road, Pretoria, 0001, South Africa may use the air quality data of Cape Town for the period 1 August 1998 – 31 July 2003 for the project "Probing secondary exposure and health data as a tool to improve public health in South Africa".

for DIRECTOR: CITY HEALTH

Appendix 5

PROOF OF ARTICLE SUBMISSIONS AND/OR ACCEPTANCE

Chapter 2

This chapter has been submitted as an article titled "Air pollution epidemiological studies in South Africa: Need for freshening up" *South African Medical Journal* on 25 January 2005. Feedback was received on 23 March 2005. The article was found to be unsuitable for *South African Medical Journal*. The article was then submitted to *Reviews on Environmental Health* on 12 July 2005. Feedback was provided on 20 September 2005. The article was accepted for publication after slight modification on 19 October 2005.

Authors' Roles

Janine Wichmann: Conception and design of the analysis, interpretation of data and writing the paper.

Kuku V. V. Voyi: Revising it critically for substantial intellectual content.

Acknowledgements

Gratitude is expressed to Dr. Petro Terblanche for her useful comments, Ms. Liz Wolvaardt for proof reading this article, and the Institute for Risk Assessment Sciences, Utrecht University, the Netherlands for using its facilities whilst writing part of this article. The first author's PhD studies were funded by scholarships from the South African National Research Foundation (NRF) (2001-2003) and the University of Pretoria (2001) along with a High-level University Year to Gain Excellence in the Netherlands (HUYGENS) Scholarship (2002-2003) and a Dutch Education: Learning at Top Level Abroad (DELTA) Scholarship (2004).

Proof of submission and acceptance of Chapter 2 as journal article in *Reviews on Environmental Health*

YAHOO! MAIL UK&IRELAND

Print - Close Window

From: "J Wichmann" <janine.wichmann@up.ac.za>

To: "Janine Yahoo" <jwichmannza@yahoo.co.uk>

Subject: Fw: Final version of proof for REH article

Date: Tue, 1 Nov 2005 13:22:32 +0200

----- Original Message -----
From: J Wichmann
To: Virginia Buchner
Cc: K Voyi ; Samantha Hodgson
Sent: Tuesday, November 01, 2005 12:45 PM
Subject: Final version of proof for REH article

Dear Virginia
Please find attached our final version of the proof.

Regards
Janine Wichmann & Kuku V.V. Voyi

School of Health Systems and Public Health
University of Pretoria
CSIR Campus, Building 22, Room A126
Meiring Naude Road
Pretoria
0001
South Africa

Tel: +27 12 841 2236
Fax: +27 12 841 3328
Mobile: +27 84 712 1506
Website: http://shsph.up.ac.za

Attachments

Files:

REH_20_4_Wichmann_PROOF_final_version.doc (251k)

Chapter 3

This chapter has been submitted as an article titled "Seasonal spatial correlation of air pollution in Cape Town, South Africa" to *Atmospheric Environment* on 10 February 2005. Feedback was received on 4 June 2005. A modified version was submitted on 10 January 2006 to *Atmospheric Environment*. The article is currently under review.

The internal examiner and the two external examiners of this thesis did not provide the same feedback as the reviewers from the journal *Atmospheric Environment*. Therefore the chapter contents may differ substantially from that of the journal article version submitted to *Atmospheric Environment*. The reader of this thesis is advised to monitor *Atmospheric Environment* for publication of the article. Otherwise, please contact the PhD candidate to obtain information on the article.

Authors' Roles

Janine Wichmann: Conception and design of the analysis, interpretation of data and writing the paper.

Kuku V. V. Voyi: Revising it critically for substantial intellectual content.

Zeleke Worku: Providing recommendations for statistical analyses.

Grant Ravenscroft: Supplying the data in the correct electronic format. Involved in design of Cape Town air quality network.

Hans Linde: Giving permission to use the data. Involved in design of Cape Town air quality network.

Acknowledgements

Gratitude is expressed towards Dr Petro Terblanche for her useful comments and the Institute for Risk Assessment Sciences, Utrecht University, the Netherlands for using its facilities whilst writing part of this article. The corresponding author received PhD scholarships from the South African National Research Foundation (NRF) (2001-2003) and the University of Pretoria (2001) along with a High-level University Year to Gain Excellence in the Netherlands (HUYGENS) Scholarship (2002-2003) and a Dutch Education: Learning at Top Level Abroad (DELTA) Scholarship (2004).

Proof of submission of Chapter 3 as journal article to *Atmospheric Environment*

YAHOO! MAIL UK&IRELAND

Print - Close Window

From: "J Wichmann" <janine.wichmann@up.ac.za>
To: "Janine Yahoo" <jwichmannza@yahoo.co.uk>
Subject: Fw: Manuscript # 25107
Date: Tue, 10 Jan 2006 11:50:28 +0200

----- Original Message -----
From: Atmos_Env
To: J Wichmann
Sent: Tuesday, January 10, 2006 11:44 AM
Subject: Re: Manuscript # 25107

Dear Dr. Wichmann,
Thank you for your email and revised manuscript etc. I will get back to you as soon as possible with further news.

Yours Sincerely,
Michele Raychaudhuri.

Karen Sturges and Michele Raychaudhuri
Senior Editorial Assistants
Atmospheric Environment
School of Environmental Sciences
University of East Anglia
Norwich NR4 7TJ
UK

email: atmos_env@uea.ac.uk
Tel: +44 (0)1603 592543
Fax: +44 (0)1603 591327
WWW: http://www.uea.ac.uk/~e044/ae.htm

----- Original Message -----
From: J Wichmann
To: Atmos_Env
Cc: K Voyi ; Zeleke Worku ; Hans Linde ; Grant Ravenscroft ; J Wichmann
Sent: Monday, January 09, 2006 4:47 PM
Subject: Re: Manuscript # 25107

Dear Michele Raychaudhuri

Please find attached my detailed reply regarding the reviewers' comments of manuscript 25107 as requested in a letter dated 22 December 2005.

I have also attached the final version of the document. All additions or changes are indicated in red.

Regards
Janine Wichmann

Lecturer: Environmental Health
School of Health Systems and Public Health
University of Pretoria
CSIR Campus, Building 22, Room A126
Meiring Naude Road
Pretoria

Chapter 4

Not submitted.

Authors' Roles

Janine Wichmann: Conception and design of the analysis, interpretation of data and writing the paper.

Kuku V. V. Voyi: Revising it critically for substantial intellectual content.

Acknowledgements

The author would like to thank Mr Hans Linde from the City of Cape Town for giving permission to use the data and for reviewing this article and Mr Grant Ravescroft from the City of Cape Town for supplying the data in the correct electronic format and reviewing this article. Gratitude is also expressed towards the Institute for Risk Assessment Sciences, Utrecht University, the Netherlands for using its facilities whilst writing part of this article. The first author received PhD scholarships from the South African National Research Foundation (NRF) (2001-2003) and the University of Pretoria (2001) along with a Netherlands Organisation for International Cooperation in Higher Education (NUFFIC) Scholarship (2002-2003) and a Dutch Education: Learning at Top Level Abroad (DELTA) Scholarship (2004).

Chapter 5

This chapter has been submitted as an article titled "Determinants of respiratory diseases and symptoms amongst adults in South Africa" to *Tropical Medicine and International Health* on 1 February 2005. Feedback was received on 13 September 2005. The reviewers raised major issues. The article was found to be unsuitable for the journal. A substantially shortened modified version was submitted to *Occupational Health Southern Africa* on 10 October 2005 with a slightly changed title "Association between risk factors and asthma prevalence amongst South African adults". The article is currently under review.

The internal examiner and the two external examiners of this thesis did not provide the same feedback as the reviewers from the journal *Tropical Medicine and International Health*. Therefore the chapter contents may differ substantially from that of the journal article version submitted to *Occupational Health Southern*

Africa. The reader of this thesis is advised to monitor *Occupational Health Southern Africa* for publication of the article. Otherwise, please contact the PhD candidate to obtain information on the article.

Authors' Roles

Janine Wichmann: Conception and design of the analysis, interpretation of data and writing the paper.

Kuku V. V. Voyi: Revising it critically for substantial intellectual content.

Acknowledgements

The authors would like to thank the South African National Department of Health for supplying the SADHS 1998 data freely. Gratitude is also expressed towards Dr Petro Terblanche for her useful comments and the Institute for Risk Assessment Sciences, Utrecht University, the Netherlands for using its facilities whilst writing part of this article. The first author received PhD scholarships from the South African National Research Foundation (2001-2003) and the University of Pretoria (2001) along with a High-level University Year to Gain Excellence in the Netherlands (HUYGENS) Scholarship (2002-2003) and a Dutch Education: Learning at Top Level Abroad (DELTA) Scholarship (2004).

Proof of submission of Chapter 5 as journal article to *Occupational Health Southern Africa*

Refer to next page

YAHOO! MAIL UK&IRELAND

Print - Close Window

From: "J Wichmann" <janine.wichmann@up.ac.za>

To: "Janine Yahoo" <jwichmannza@yahoo.co.uk>

Subject: Fw: Publication submission to Occupational Health Southern Africa

Date: Mon, 10 Oct 2005 15:23:01 +0200

----- Original Message -----
From: J Wichmann
To: fiona.robinson@up.ac.za
Cc: K Voyi ; Samantha Hodgson
Sent: Monday, October 10, 2005 3:22 PM
Subject: Publication submission to Occupational Health Southern Africa

Dear Dr Robinson

We would like to submit our publication titled *Association between risk factors and asthma prevalence amongst South African adults* to *Occupational Health Southern Africa*.

The authors hereby declare that the publication submitted to *Occupational Health Southern Africa* has neither been published elsewhere nor is being considered elsewhere for publication. No competing interests are declared. Both authors hereby transfer copyright of the submitted publication to *Occupational Health Southern Africa*.

Yours sincerely,
Miss Janine Wichmann and Prof Kuku V.V. Voyi

School of Health Systems and Public Health
University of Pretoria
CSIR Campus, Building 22, Room A126
Meiring Naude Road
Pretoria
0001
South Africa

Tel: +27 12 841 2236
Fax: +27 12 841 3328
Mobile: +27 84 712 1506
Website: http://shsph.up.ac.za

Attachments

Files:

Asthma_SA_adults.doc (82k)

Chapter 6

This chapter has been submitted as an article titled "Acute respiratory health implications amongst preschool children and indoor fossil and biomass fuel use in South Africa" to *Environmental Research* on 18 January 2005. Feedback was received on 15 July 2005. The reviewers raised major issues. The article was found to be not suitable for the journal. A substantially shortened modified version was submitted to *Southern African Journal of Epidemiology and Infection* on 5 October 2005 with a slightly different title "Potential impact of cooking and heating fuel use on acute respiratory health of preschool children in South Africa". The article is currently under review.

The internal examiner and the two external examiners of this thesis did not provide the same feedback as the reviewers from the journal *Environmental Research*. Therefore the chapter contents may differ substantially from that of the journal article version submitted to *Southern African Journal of Epidemiology and Infection*. The reader of this thesis is advised to monitor *Southern African Journal of Epidemiology and Infection* for publication of the article. Otherwise, please contact the PhD candidate to obtain information on the article.

Authors' Roles

Janine Wichmann: Conception and design of the analysis, interpretation of data and writing the paper.

Kuku V. V. Voyi: Revising it critically for substantial intellectual content.

Acknowledgements

The authors would like to thank the South African National Department of Health for supplying the SADHS 1998 data freely. Gratitude is also expressed towards the Institute for Risk Assessment Sciences, Utrecht University, the Netherlands for making use of its facilities whilst writing part of this article. The first author received PhD scholarships from the South African National Research Foundation (2001-2003) and the University of Pretoria (2001) along with a High-level University Year to Gain Excellence in the Netherlands (HUYGENS) Scholarship (2002-2003) and a Dutch Education: Learning at Top Level Abroad (DELTA) Scholarship (2004).

Proof of submission of Chapter 6 as journal article to *Southern African Journal of Epidemiology and Infection*

INBOX: 245 of 590 Move | Copy this message to:

Delete | Reply | Reply to all | Forward | Bounce | Resume | Save as Back to

Date Mon, 30 Jan 2006 10:51:33 +0200

From Hendrik Koornhof <Hendrik.Koornhof@nhls.ac.za>

To "'jwichmann@med.up.ac.za'" <jwichmann@med.up.ac.za>

Cc "'feldmanc@medicine.wits.ac.za'" <feldmanc@medicine.wits.ac.za>, Priscilla May <priscilla.may@nhls.ac.za>

Subject RE: Submission of paper to SA Journal of Epidemiology and Infecti on

Parts Message Source

Dear authors,

Your paper is in the process of being reviewed and we are awaiting the referees' reports. We will let you know the outcome as soon as our journal's editorial committee has perused their response.

Thank you for supporting our journal.

Regards,

Professor H Koornhof
Emeritus Editor

-----Original Message-----
From: jwichmann@med.up.ac.za [mailto:jwichmann@med.up.ac.za]
Sent: 27 January 2006 06:30 PM
To: Hendrik Koornhof
Cc: Priscilla May; feldmanc@medecine.wits.ac.za; kvoyi@med.up.ac.za; shsph@up.ac.za
Subject: Submission of paper to SA Journal of Epidemiology and Infection

Dear Dr Koornhof

We have submitted a paper titled "Impact of cooking and heating fuel use on acute respiratory health of preschool children in South Africa" to Southern African Journal of Epidemiology on 10 October 2005.

Can you please provide us with an update regarding its review process?

Regards
Dr Janine Wichmann and Prof Kuku V.V. Voyi

School of Health Systems and Public Health
University of Pretoria
CSIR Campus, Building 22, Room A126
Meiring Naude Road
Pretoria
0001
South Africa

Tel: +27 12 841 3240
Fax: +27 12 841 3308
Website: http://shsph.up.ac.za

Chapter 7

This chapter has been submitted as an article titled "Influence of cooking and heating fuel use on 1-59 month old mortality in South Africa" to *Annals of Tropical Paediatrics: International Child Health* on 31 March 2005. Feedback was received on 1 June 2005. The reviewers raised major issues. The article was found to be unsuitable for *Annals of Tropical Paediatrics: International Child Health*. A substantially shortened modified version was submitted to *Maternal and Child Health Journal* on 10 October 2005. The article is currently under review.

The internal examiner and the two external examiners of this thesis did not provide the same feedback as the reviewers from the journal *Annals of Tropical Paediatrics: International Child Health*. Therefore the chapter contents may differ substantially from that of the journal article version submitted to *Maternal and Child Health Journal*. The reader of this thesis is advised to monitor *Maternal and Child Health Journal* for publication of the article. Otherwise, please contact the PhD candidate to obtain information on the article.

Authors' Roles

Janine Wichmann: Conception and design of the analysis, interpretation of data and writing the paper.

Kuku V. V. Voyi: Revising it critically for substantial intellectual content.

Acknowledgements

The authors would like to thank the South African National Department of Health for supplying the 1998 SADHS data freely. The first author received PhD scholarships from the South African National Research Foundation (NRF) (2001-2003) and the University of Pretoria (2001) along with a High-level University Year to Gain Excellence in the Netherlands (HUYGENS) Scholarship (2002-2003) and a Dutch Education: Learning at Top Level Abroad (DELTA) Scholarship (2004).

Proof of submission and acceptance of Chapter 7 as journal article in *Maternal and Child Health Journal*

Refer to next page

INBOX: 559 of 590 Move | Copy this message to:

Delete | Reply | Reply to all | Forward | Bounce | Resume | Save as Back to

Date Sat, 18 Feb 2006 09:14:05 -0500

From Maternal and Child Health Journal <tsinger@hsc.usf.edu>

To jwichmann@med.up.ac.za

Subject Decision on your Manuscript #MACI197

Parts Message Source

Dear Dr Janine - Wichmann, PhD,

Thank you for submitting manuscript #MACI197 entitled "Influence of cooking and heating fuel use on 1-59 month old mortality in South Africa" to the Maternal and Child Health Journal. We have concluded our review of this manuscript and are pleased to inform you that this article has been accepted for publication in the MCH Journal provided the recommended revisions are made.

When compiling your response to reviewer comments, please be sure NOT to include author contact information as this is a doubleblind reviewing journal should this need to go back to the reviewers.

Below, please find copies of the reviewer comments. Please consider these comments in preparing your revision and include with your response an explanation of how you chose to incorporate their suggestions. We would like to receive your revised manuscript within the next 60 days. If this is not possible, or you elect not to revise the manuscript for us, please let us know immediately by contacting Terri Singer, Managing Editor, at 813-974-7273 or at tsinger@hsc.usf.edu.

In order to submit your revised manuscript electronically, please access the following web site:

http://maci.edmgr.com/

Your login is: [redacted]
Your password is: [redacted]

Click "Author Login" to submit your revision.

If you haven't already sent the signed Copyright Transfer Form, we will need to receive it. You can locate the form on the journal's Welcome Page at:

http://maci.edmgr.com/

Please print the form, sign it, and return it to us. Thank you.

We appreciate your interest in the MCH Journal and look forward to receiving your revision. Please let us know if you have any questions. We look forward to hearing from you within the next two months. Thank you.

Sincerely,

Donna J. Petersen, MHS, ScD
Co-Editor-in-Chief

Greg R. Alexander, MPH, ScD
Co-Editor-in-Chief

COMMENTS FOR THE AUTHOR:

Appendix 6

CURRICULUM VITAE

I was born on 7 May 1973 in Sisheng, Northern Cape Province. I matriculated at Montana Hoërskool, Pretoria in December 1991. I graduated in December 1994 and March 1995 with a B.Sc (Chemistry) and a B.Sc(Hons) (Analytical Chemistry) from the University of Pretoria. I continued with M.Sc(Med) (Chemical Pathology) studies at the University of Cape Town during 1996-1997 and graduated in June 1998. I worked at the Environmentek Divison, CSIR during 1998-2000 as an environmental scientist. In 2001 I graduated with an M.Sc (Environmental Ecology) from the University of Pretoria. My doctoral studies started in 2001 at the School of Health Systems and Public Health (SHSPH), University of Pretoria. I furthermore conducted two research projects on personal air pollution exposure assessment at the Institute for Risk Assessment Sciences, Utrecht University, the Netherlands during 2002-2004 under the supervision of Prof Bert Brunekreef. I was appointed on a temporary part-time basis and lectured postgraduate modules at the SHSPH during 2003 and 2005. My appointment with the SHSPH was extended on a temporary permanent basis during 1 March – 31 December 2005. I was awarded a National Research Foundation Postdoctoral Fellowship for Abroad during January 2006. I am conducting the postdoctoral project at the Institute of Environmental Medicine, Karolinska Institute in Sweden during January - December 2006 under the guidance of Prof Tom Bellander. The project investigates the relationship and contributing factors of personal, indoor and outdoor air pollution.

List of Dissertations

Wichmann J. *The role of TNP-nucleotides, LYS-492 and Ca^{2+} chelators in the skeletal muscle sarcoplasmic reticulum Ca^{2+}ATPase cycle*.M.Sc(Med) thesis; University of Cape Town; 1997.

Wichmann J, Kubheka L, Jones J, Howland J, Ashan N. *Colbyn Valley Case Study 2000: An Enquiry into Environmental Issues and Management*. M.Sc(Environmental Studies) Group Project Report; University of Pretoria; 2000.

List of Research Reports

Wichmann J, Henning NJ, Taljaard JF. *Exposures to pollutants from low-smoke fuels compared to ordinary coal, measured during a macro-scale experiment*. CSIR Report; ENV-P-CONF 98047; 1998.

Henning NJ, Wichmann J, McCormick SG. *Determination of chromium in ambient air at Chrome International, Newcastle*. CSIR Report; ENV-P-C 98101; 1998.

Wichmann J. *CALINE 4 model vs IVL passive sampler results for NO_2 concentrations in Pretoria*. CSIR Report; ENV-P-CONF 99001; 1999.

Voyi KVV, Wichmann J. *Material safety data sheet for p-hydroxybenzyl methyl ether solution, and data sheet for p-hydroxybenzyl methyl ether crystals*. CSIR Report, ENV-P-C 99052, 1999.

Wichmann J. *Human Health Risk Assessment Case Study – Value addition of passive sampler generated data*. CSIR Report, ENV-P-I-99008; 1999.

Wichmann J, Henning NJ. *Implementation of the IVL passive sampler technology in Durban and Pretoria*. CSIR Report; ENV-P-I-99009; 1999.

John J, Wichmann J, Matooane L, Oosthuizen R, Crookes D. *Economic evaluation of health risks resulting from air pollution: Health deliverable*. CSIR Report; ENV-P-I-2000-062; 2000.

List of Conference Presentations

Wichmann J, Henning NJ, Taljaard JF. *Exposures to pollutants from low-smoke fuels compared to ordinary coal, measured during a macro-scale experiment*. 1998 (Oral presentation at the 11th World Clean Air Congress, Durban, South Africa, 13-18 September 1998).

Wichmann J. *Utilising diffusive (passive) samplers - a modern cost-effective approach to air quality measurement and monitoring*. 1999 (Oral presentation at the Air Pollution Control Conference, Johannesburg, South Africa, August 1999).

Wichmann J. *CALINE 4 model vs IVL passive sampler results for NO_2 concentrations in Pretoria*. 1999 (Oral presentation at the National Association for Clean Air Conference, Cape Town, South Africa, 7-8 October 1999)

Boer A, Genthe B, Wichmann J. *Human Health Risk Assessment - A tool in the way forward*. 1999 (Oral presentation at the National Association for Clean Air Conference, Cape Town, South Africa, 7-8 October 1999).

Ferm M, Wichmann J. *Mapping SO_2 concentrations in Durban South*. 2001 (Poster presented at the International Conference Measuring Air Pollutants by Diffusive Sampling, Montpellier, France; p93; 2001).

Van Roosbroeck S, Wichmann J, Janssen N, Hoek G, Brunekreef B. *Personal exposure to traffic-related air pollution among school children in Amsterdam*. 2004 (Oral presentation at the XXIII Congress of the European Academy of Allergology and Clinical Immunology, Utrecht, the Netherlands, 16-18 June 2004).

Wichmann J, KVV Voyi. Determinants of respiratory diseases and symptoms amongst adults in South Africa. Epidemiology 2005;16(5):S19-S20. (Oral presentation at the 17th International Society of Environmental Epidemiology Conference, Johannesburg, South Africa, 13-16 September 2005).

Wichmann J, Janssen NAH, van der Zee S, Brunekreef B. Traffic-related differences in indoor and personal absorption coefficient measurements in Amsterdam, the Netherlands. *Epidemiology* 2005; 16(5):S27. (Poster presentation at the 17th International Society of Environmental Epidemiology Conference, Johannesburg, South Africa, 13-16 September 2005).

Wichmann J, KVV Voyi. Air Pollution Epidemiological Studies in South Africa: Need for freshening up. *Epidemiology* 2005; 16(5): S20. (Poster presentation at the 17th International Society of Environmental Epidemiology Conference, Johannesburg, South Africa, 13-16 September 2005).

Wichmann J, KVV Voyi. Acute respiratory health implications amongst preschool children and indoor fossil and biomass fuel use in South Africa. *Epidemiology* 2005; 16(5): S20. (Poster presentation at the 17th International Society of Environmental Epidemiology Conference, Johannesburg, South Africa, 13-16 September 2005).

List of Publications

Wichmann J. Particulate matter associated transition metals: Is there a possible link with childhood respiratory diseases in South Africa? December 2003, *Medical Research Council Urban Health and Development Bulletin* (http://www.mrc.ac.za/urbanbulletin/dec2003.pdf).

Wichmann J, Janssen NAH, van der Zee S, Brunekreef B. Traffic-related differences in indoor and personal absorption coefficient measurements in Amsterdam, The Netherlands. *Atmospheric Environment* 2005; 39:7384–7392.

Wichmann J, KVV Voyi. Air Pollution Epidemiological Studies in South Africa: Need for Freshening Up. *Reviews on Environmental Health* 2005;20(4): 265-301.

Wichmann J, Voyi KVV. Influence of cooking and heating fuel use on 1-59 month old mortality in South Africa. Accepted by *Maternal and Child Health Journal* on 20 February 2006.