The provision of professional, independent consulting services to assist Eskom in compiling applications for renewed postponement of the Minimum Emission Standards:

Component 4: Health impact focused cost benefit analyses

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PREAMBLE

This study forms part of a range of studies conducted for Eskom as part of their application for postponement of the Minimum Emission Standards compliance timeframes. The study investigates the costs and benefits of mitigating air pollution emissions from 13 Eskom coal-fired power stations. The methodology is based on World Health Organisation guidelines.

In addition to the authors, the contributors to this analysis include:

- Sean O'Beirne from SE Solutions facilitated the interactions between the different research teams and provided key insights on abatement technologies.
- Mark Zunckel and Atham Raghunandan from uMoya-NILU Consulting provided the dispersion modelling results.
- Caradee Wright from the South African Medical Research Commission and Rietha Oosthuizen provided the epidemiological evidence used in the study.
- Lorraine Ndala, Bryan McCourt, Bianca Wernecke, Tobile Bokwe, and Ebrahim Patel from Eskom provided important details on scenarios and abatement technology costs.

EXECUTIVE SUMMARY

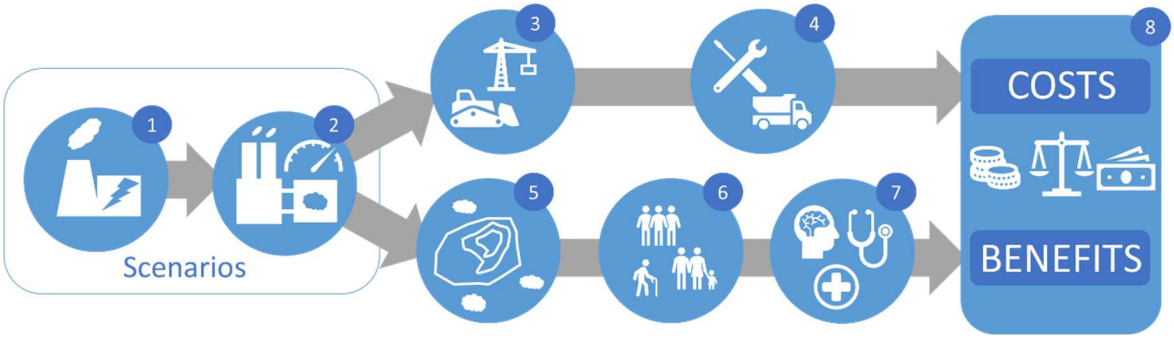
The combustion of fossil fuels results in the emission of numerous atmospheric pollutants, that include but are not limited to Particulate Matter (PM), Nitrogen dioxide (NO_2), and Sulphur dioxide (SO_2). Atmospheric pollutants have numerous negative effects on human health and have been demonstrated to increase the risk of premature mortality.

Technologies exist to reduce these emissions and therefore the health effects. Abatement technologies include Flue Gas Desulphurisation (FGD) for SO_2 reduction, Electrostatic Precipitators (ESP) and Fabric Filter Plants (FFP) for PM reduction, Low NO_X Burners (LNB) for NO_2 reduction. A cost-benefit analysis (CBA) allows for trade-offs between different scenarios to be compared to support decision making.

The aim of this study was to estimate the incremental health benefits associated with abatement technology options that achieves compliance with the new Minimum Emission Standards (MES) of the Department of Environmental Affairs (DEA).

Methodology

An integrated Health CBA Model was developed, to model the impacts of four different scenarios. The Health CBA Model followed the General Principles of the World Health Organisation (WHO, 2016) for performing air pollution health risk assessment (AP-HRA). The detailed methodology and assumptions are set out in section 2 below. In summary, the methodology proceeded through several steps, as set out in the schematic and proceeding text below:



1. Plant lifetimes were described for 13 coal-fired power plants and included commissioning and decommissioning dates (provided by Eskom).
2. Abatement technologies required for each scenario were defined, by type and likely implementation schedule (refer to section 2.3.3).
3. Capital expenditure required for abatement in each scenario was attributed per plant and per year using Eskom's internal estimates.
4. Operational expenditure required for abatement in each scenario was attributed per plant and per year using Eskom's internal estimates.
5. Dispersion modelling results were obtained as part of the broader Eskom investigation (Zunckel and Raghunandan, 2018). This data was segregated spatially, by ward and municipal boundaries to align with population data. Two sets of dispersion modelling data were obtained. The first set modelled predicted ambient concentrations of PM, NO_2 and SO_2 around individual power stations. The second set modelled cumulative predicted ambient concentrations of PM, NO_2 and SO_2 from all power stations on the Highveld. The dispersion modelling results were unique

because in addition to primary PM, the modelling predicted secondary PM effects, resulting from NO_2 and SO_2 reactions in the atmosphere (refer to Section 2.2).

6. Population exposure was estimated at a spatial resolution of municipality and municipal wards. At each municipality or ward, the number of people exposed to different concentration ranges were determined per scenario per year, based on Stats SA population estimates and United Nations population growth forecasts (refer to Section 2.2).
7. Health impacts were determined by using the AP-HRA methodology. Epidemiological evidence, in the form of Exposure-response functions (ERFs) and baseline incidence rates were provided by the SA Medical Research Council (SAMRC) (Wright and Oosthuizen, 2018) (refer to Section 2.3). The ERFs were limited to mortality incidence. The Cost of Illness (COI) methodology used was the value of statistical life (VSL). This method estimates the willingness to pay (WTP) of an individual for reducing their health risk. The VSL should not be interpreted as the intrinsic value of a life. Refer to Section 2.3.3 for a more detailed discussion.
8. The CBA compares the overall scenario benefits and costs. The outputs of the AP-HRA, the health cost savings of each scenario, was used as the benefit. The capital and operational cost estimates were used as the costs in the CBA. The analysis timeline spans 2015 – 2045. This timeframe allows for 5-year interval analysis, aligning to the 2020 MES. It also captures mitigation activities implemented since 2016. The base year was 2018, due to dispersion modelling timeframe. The CBA was performed in an Excel spreadsheet, which consolidated all data sources, which contains all calculations, and was macro-enabled to run the large spatial exposure estimates for each scenario for the review period (refer to Section 2.4). Finally, an assessment of uncertainty of the results was done (refer to Section 2.5).

Health benefits resulting from air pollution abatement

The WHO (2016) recommends that the health risk in a population, associated with air pollution, is to be estimated using exposure-response functions (ERFs). ERFs are based on Relative Risk (RR) estimates derived from primary epidemiological studies. These RR functions estimate the likelihood of health outcomes occurring in a population exposed to a higher level of air pollution relative to that in a population with a lower exposure level. RR is usually expressed as the proportional increase in the assessed health outcome risk incidence associated with a given increase in pollutant concentrations, measured in $\mu g/m^3$. The WHO (2016) notes that "*the RR estimate cannot be assigned to a specific person; it describes risk in a defined population, not individual risk.*"

Ideally, ERF studies and their RRs should be determined based on primary epidemiological studies focussing on the exposed population. In the absence of such studies, as in the case of South Africa, the WHO (2016) recommends using ERFs from other countries. The SAMRC provided a number of ERF options for South Africa (refer to section 2.3.1). These were all mortality related RRs, and thus mobility related RRs were not assessed in this study. It is to be noted however that there are inherently significant limitations in transferring ERF studies from other countries. Pollution levels, chemical composition and health care systems are typically very different in other settings, and this would affect the accuracy of the ERFs. It is important to understand at what level interval the ERFs would result in significant differences in health outcome incidences. As a result, the WHO (2016) advises performing an assessment of the uncertainty of the analysis; in this case therefore this requires an assessment

related to a lack of knowledge about one or more components of the integrated Health CBA Model. Section 2.5 discusses each source of uncertainty and related limitations. Variation in health outcome incidences between the various ERFs provided, in some cases exceed 80%. This variation was dealt with through performing sensitivity analysis in the CBA (refer to section 2.4).

Interpretation of premature mortality or attributable deaths has to be done with care. It is to be noted firstly that these numbers are indicators of health risk at a population level. The relative risk estimate inherent in the ERF is a metric of the likelihood of an adverse health outcome, and it cannot be attributed to an individual person. It can thus be used to quantify risk to a defined population (and not to an individual), (WHO 2016) and how this risk would vary between various policy options of scenarios.

The ERFs provided by the SAMRC focussed exclusively on mortality and thus a monetary measure of mortality was required in order to perform cost-benefit analyses. In air pollution cost-benefit analyses, the concept of value per statistical life (VSL) is commonly used to monetise mortality related benefits of air pollution reduction. The concept of a VSL is frequently misunderstood. It does not measure the intrinsic value of a human life, and neither does it value the economic productivity of a human. Rather, VSL is estimated by dividing an individual's willingness to pay (WTP) to reduce health risk, by the likelihood of risk reduction. Robinson and Hammitt (2009) defines VSL to represent the rate at which an individual is willing to exchange their own income for a small reduction in their own mortality risk over a particular time period. Primary WTP studies for mortality risk reductions have not been done in South Africa. However, three studies have made VSL estimates for South Africa, all based on extrapolation of United States studies. These values, when adjusted to 2018, vary between R21 million and R48 million (Table 4). The most conservative of these estimates (i.e. the highest VSL) of R 53 Million (in 2020) was adjusted to 2018 and was used in the CBA.

In spite of the various sources of uncertainty discussed above, the analysis still provides valuable insights into the comparison of scenarios tested in the CBA. This is because the uncertainty inherent in the analysis remains constant across all scenarios.

Scenario assessment

The scenarios evaluated in this study (against the baseline) included:

1. Full compliance with new plant standards (FC) (Scenario 1 (Sc1))
2. Eskom Emission Reduction Plan (ERP) (Scenario 2 (S2))
3. ERP + FGD at Kendal and Matimba (Scenario 3 (S3))
4. ERP + Early decommissioning (ED) of Komati, Hendrina and Grootvlei (Scenario 4 (S4))

Approximately 20.3 million people are exposed to air pollution from the 13 power plants modelled, that fall within the modelling domain. The mean additional annual average exposure to air pollution of the population within this domain, resulting from coal-fired power station emissions, was estimated by averaging dispersion modelling results over municipal boundaries. Approximately 17.7 million people were exposed to more than an additional 1µg.m^3 (mean annual average) of $PM_{2.5}$. Similarly, 15.3 and 19.0 million people, respectively, were exposed to more than an additional 1µg.m^3 of NO_2 and SO_2 (see Section 2.2.4).

Health benefits associated with each scenario were calculated against the baseline that assumed no new abatement technologies would be installed, and all plants would continue to emit air pollution at their current rates until decommissioning. The scenario with the highest health benefits was ERP+ED (S4), highlighting the immediate results achievable if early decommissioning of power plants can be

achieved. The ERP+ED (S4) is estimated to result in health benefits with a NPV that varied between R 3.4 billion and R 30.1 billion. The FC (S1) had the next highest health benefits with a NPV that varied between R 2.5 billion and R 22.1 billion. The ERP+FGD (S3) had marginally higher health benefits than ERP (S2) due to the additional FGD at Kendal. Figure 1 demonstrates the averaged flow of benefits for the four scenarios.

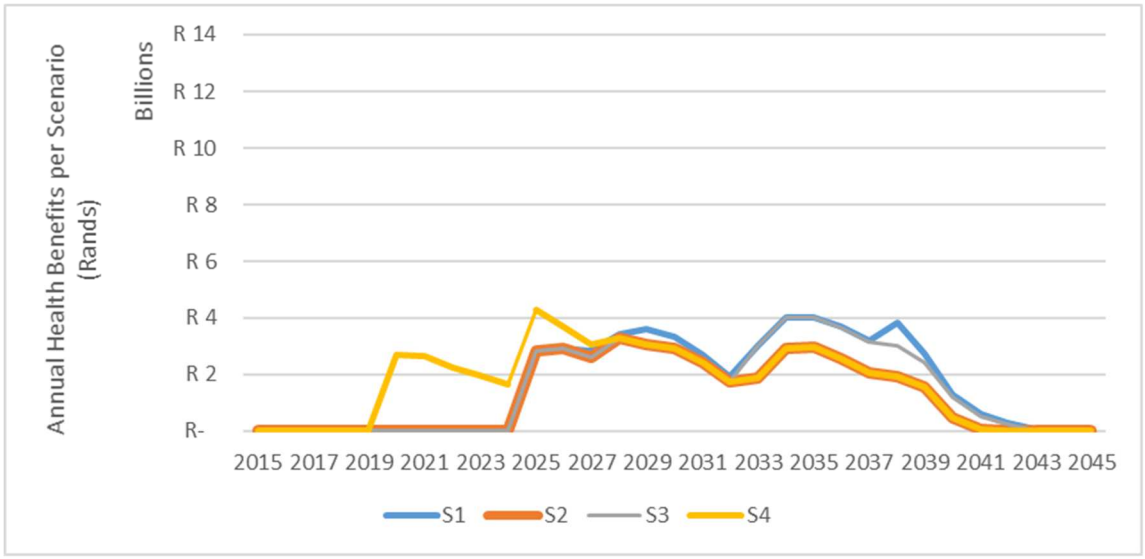


Figure 1 Average annual health benefits per scenario

Scenario costs were calculated using Eskom's estimates of abatement technology capital and operational spending requirements. As expected, the FC (S1) had the highest costs due to having the most abatement technologies installed, with the NPV between -R43.4 billion to -R65.1 billion. The ERP (S2) and ERP+ED (S4) had the same costs as they both had the same abatement technology additions with a NPV between -R16.9 billion to -R25.3 billion. The ERP+FGD (S3) had a higher cost with a NPV of -R21.2billion to -R31.8 billion due to the additional FGD at Kendal. Figure 2 demonstrates the averaged flow of costs for the four scenarios.

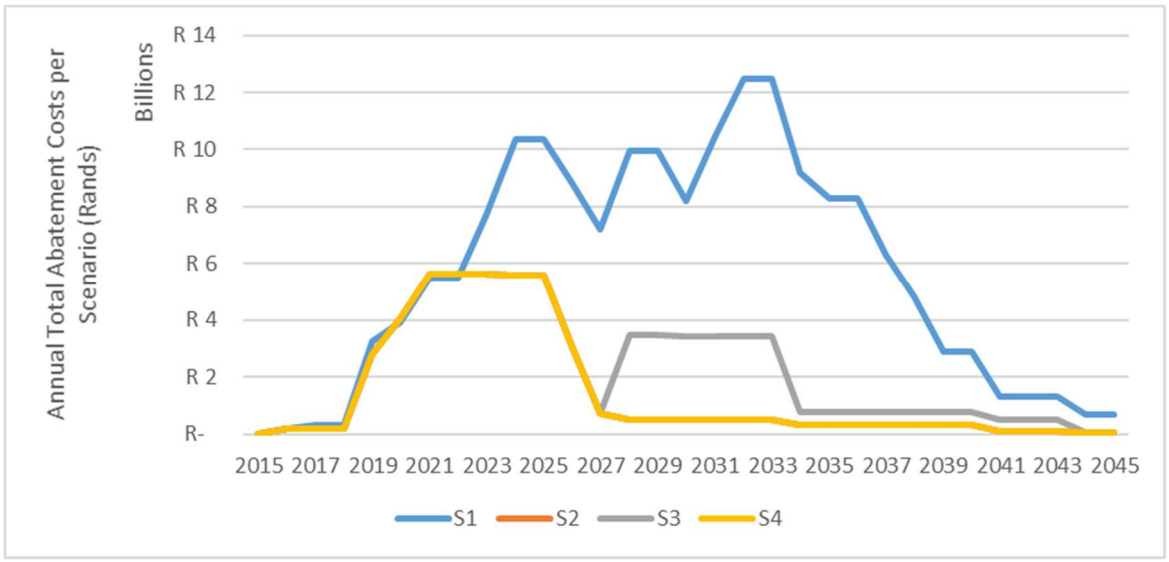


Figure 2 Total abatement costs (CAPEX and OPEX) associated with each scenario's abatement retrofits

Scenarios were compared in a cost-benefit analysis. The cost-benefit analysis apportioned costs (capital and operation expenditure on abatement technologies) and benefits (health benefits) to the years in which they would be realised. Because costs and benefits are accrued in different years according to the intervention schedules, the net present values of costs and benefits, using Eskom's weighted average cost of capital (WACC) rate of 8.4% as the discount rate allows an objective comparison of scenarios. Dividing the NPV of costs by the NPV of benefits provides a cost:benefit ratio, which when greater than 1 indicates that the costs outweigh the benefits, and when less than 1 indicate that the benefits outweigh the costs.

The CBA ratios need to be interpreted with care. They are meant only to provide a perspective on and inform the decision-making process underlying the scenarios. They are not meant to be interpreted as a definitive answer to making abatement decisions. Decisions involving human health have to be informed by non-economic criteria as well. In addition, uncertainty inherent in the analysis, the cost benefit ratio should thus not be viewed as absolute, but rather as a relative value from which to compare scenarios.

Table 1 Costs and benefits NPV estimates (lower and upper ranges) for each scenario, and cost:benefit ratios

	FC (S1)		**ERP (S2)**		**ERP+FGD (S3)**		**ERP+ED (S4)**	
Million Rands	*lower*	*upper*	*lower*	*upper*	*lower*	*upper*	*lower*	*upper*
NPV of Costs	-43 369	-65 053	-16 923	-25 385	-21 205	-31 808	-16 923	-25 385
NPV of Benefits	2 403	21 625	1 962	17 661	2 252	20 264	3 374	30 367
NPV of Benefits minus Costs	-40 966	-43 428	-14 961	-7 724	-18 954	-11 544	-13 549	4 982
Cost: Benefit Ratio (*range*)	18.0	3.0	8.6	1.4	9.4	1.6	5.0	0.8
Cost: Benefit Ratio (*central*)	4.5		2.2		2.4		1.3	

Refer to section 3.2 for sensitivity analysis using alternative discount rates.

In spite of the uncertainties that are inherent in the current assessment process, the assessment provides valuable insights into the effects of air pollution and abatement. The larger investigation has made significant progress on improving the accuracy of dispersion modelling, through modelling secondary PM emissions and through performing cumulative emissions analysis. This work has increased our understanding of the exposed population. It is recommended that the analysis performed here be continuously improved to address prioritised sources of uncertainty. Improving the accuracy of the ERFs needs priority attention as AP-HRA applications continue to be improved.

In spite of the level of uncertainty associated with ERFs, epidemiological evidence is sufficient to confirm the hypothesis that abatement technologies would have positive impacts on human health.

With the testing of different discount rates, all scenarios become more favourable (i.e. future health benefits received a higher weighting than upfront costs). What is important to note however is that the order of the scenarios, as assessed using cost benefit ratios, do not change.

Thus, most significantly, early decommissioning of the coal-fired power stations assessed in ERP+ED (S4), would have a significantly larger beneficial effect on health costs than abatement technologies alone. This holds for all discount rates tested. This plays a large role in positioning Scenario 4 as the most beneficial scenario, both in terms of largest health cost benefits, lowest cost of abatement, as well as relative cost:benefit ratio. The FC (S1) would eventually have the second most benefits (see Tables 5, 6 and 7).

It is also noted that the abatement technologies are expensive, and would place a significant financial burden on Eskom.

ACRONYMS AND ABBREVIATIONS

AP-HRA	Air Pollution Health Risk Assessment
CBA	Cost-Benefit Analysis
COI	Cost of Illness
COPD	Chronic Obstructive Pulmonary Disease
DEA	Department of Environmental Affairs
ERF	Exposure Response Function
ESP	Electrostatic Precipitators
FGD	Flue Gas Desulphurisation and Fabric Filter Plants (FFP).
HPA	Highveld Priority Area
ICD	International Classification of Diseases
IRP	Integrated Resource Plan
kW	Kilowatt
LNB	Low NOX Burners
MES	Minimum Emissions Standards
NAAQS	National Ambient Air Quality Standard
NO_2	Nitrogen Oxide
NPV	Net Present Value
PM	Particulate Matter
RR	Relative Risk
SAMRC	South African Medical Research Council
SO_2	Sulphur Dioxide
TB	Tuberculosis
VSL	Value of Statistical Life
WHO	World Health Organisation
WACC	Weighted Average Cost of Capital
WTP	Willingness to Pay

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1. INTRODUCTION

The Earth Summit[1] in Rio de Janeiro in 1991 raised the awareness of the linkages between environmental health and human wellbeing to a global agenda. In the two and half decades since the Summit, significant effort has gone into methods for quantifying these linkages, in all environmental spheres, and informing policy development. During the same period, we have seen an information technology revolution, which has radically improved our ability to collect and analyse large data sets. In the field of air quality health risk assessment specifically, there has been a rapid and continuously improving set of methodologies through which to analyse the linkages between air pollution and health risk.

The World Health Organisation (WHO) has been leading the development of health risk assessment methodology. Formally, air pollution health risk assessments (AP-HRA) are performed to provide quantifiable information for informing public policy decisions. The general principles for AP-HRAs have been published by the WHO (WHO, 2016). An AP-HRA proceeds through three steps.

Firstly, it assesses the exposure of the target population to specific air pollutants. This requires a quantification of constituents in the atmosphere that are associated with human health risks. The atmosphere we breathe contains various such constituents, both from natural sources (e.g. sea salt and bio-aerosols) and anthropogenic sources (e.g. fuel combustion, suspension of fine particles, and industrial emissions) (refer to FRIDGE (2004) for a comprehensive discussion of pollution sources). When a particular policy option is analysed, specific indicator constituents need to be selected, and the incremental effect of the policy option needs to be estimated in terms of population exposure. In this study, incremental population exposure resulting from Eskom's coal-fired power plant emissions (from 13 plants), was estimated through the use of dispersion modelling (refer to Zunckel and Raghunandan, 2018).

Secondly, the AP-HRA estimates the resultant incremental change in health risk. This requires the application of exposure-response functions (ERFs). ERFs quantify the incremental change in health outcomes (compared to the baseline incidence), based on changes in exposure to pollutants. ERFs are derived from epidemiological studies, which are large scale population health studies that compare health outcome incidence between populations exposed to different concentrations of pollution. In this study, ERFs from studies in the USA and India were used, as no local ERFs exist (Wright and Oosthuizen, 2018). AP-HRA results can be reported in terms of morbidity indicators (e.g. cost of medical treatment and lost economic productivity) or mortality indicators (e.g. premature mortality). These indicators can be converted to monetary impacts by applying cost of illness (COI) methodologies. In this study, premature mortality was evaluated, using a value of a statistical life (VSL) COI methodology.

Thirdly, the AP-HRA process requires the quantification and expression of the uncertainty of the estimated health effects. The WHO states that this step is "*an important and integral component of the results, and … vital to ensure both that the main message is not lost and that the results produced are understandable by policy-makers and others who do not necessarily have a technical background or expertise in AP-HRA.*" This step requires "*the use of expert judgement (consensus) on the level of confidence of the results*".

This study investigates the health effects of air pollution resulting from coal-fired powered plants and applies the AP-HRA methodology described above.

[1] http://www.un.org/geninfo/bp/enviro.html

The indicator pollutants used included particulate matter (PM), nitrogen dioxide (NO_2) and sulphur dioxide (SO_2). These pollutants have several negative impacts, of most concern are the health impacts that include for instance heart disease, lung cancer, stroke and chronic obstructive pulmonary disease (WHO, 2016a).

The Department of Environmental Affairs under the National Environmental Management Act (NEMA: AQA, 2004) sets ambient air quality standards. The Highveld, containing most of South Africa's coal-fired power stations, often exceeds the National Ambient Air Quality Standards (NAAQS) (DEA, 2009 and 2012). As a result, the Minister of Environmental Affairs declared the Highveld Priority Area (HPA) in November 2007 in terms of Chapter 18 of the National Environment Management: Air Quality Act, 2004 (Act No. 39 of 2004) (NEMA: AQA). Where ambient air quality standards are exceeded, specific air quality mitigation actions would be required. Power generation is a Listed Activity in terms of Section 21 of the NEMA: AQA and Minimum Emission Standards (MES) are prescribed for existing and new plants. Existing plants must comply with new plant standards by 2020.

Technologies exist to reduce these emissions and therefore the health effects. Such abatement technologies include Flue Gas Desulphurisation (FGD) to reduce SO_2, Electrostatic Precipitators (ESP) to reduce PM, Low NO_X Burners (LNB) to reduce NO_2 and Fabric Filter Plants (FFP) to reduce PM.

This study investigated four air pollution mitigation scenarios for Eskom, through a cost-benefit analysis (CBA). The CBA uses the AP-HRA methodology to estimate the likely changes in health costs resulting from each scenario. The CBA compares these benefits against the capital costs and operational costs of the mitigation options for each scenario (refer to section 2.3).

Other studies have previously been conducted to estimate the health impacts of either fossil fuel power plants, air pollution in general or specific sources in South Africa. They estimated morbidity and mortality, and in some instances attributed costs to these health impacts. Studies of this nature can take either bottom up (deterministic) approaches or top down (stochastic) approaches to modelling pollution exposure with the latter usually preferable in data poor environments or large spatial domains (Dios et al., 2012). These studies also varied in geographic scale, ranging from selected areas to the national scale. Some of the most recent and relevant include:

The World Health Organisation estimated that, in South Africa, in 2009, approximately 1,100 deaths could be attributed to poor outdoor air quality (WHO, 2009).

- Scale: National (All Air Pollution)
- Resolution: Course
- Health Outcomes: Mortality
- Modelling Approach: Top-down

The Institute for Health Metrics and Evaluation listed air pollution as the 9th largest risk factor driving death and disability combined in 2016 in South Africa (IHME, 2016).

- Scale: National (All Air Pollution)
- Resolution: Medium
- Health Outcomes: Morbidity and Mortality
- Modelling Approach: Bottom-up

A study commissioned by Greenpeace in 2014 estimated air pollution emissions from Eskom's coal-fired power plants could cause as much as 2,200 premature deaths per year (Myllyvirta, 2014). The study also estimated the impacts of mercury pollution.

- Scale: National (Air Pollution from Coal-fired Power Plants)
- Resolution: Medium
- Health Outcomes: Morbidity and Mortality
- Modelling Approach: Bottom-up

A 2017 study commissioned by Groundwork, estimated the total impact of air pollution resulting from the coal-fired power plants at $2.4 billion of health costs annually in South Africa (Holland, 2017).

- Scale: National (Air Pollution from Coal-fired Power Plants)
- Resolution: Medium
- Health Outcomes: Morbidity and Mortality
- Modelling Approach: Bottom-up

Van Horen (1996) evaluated the health costs associated with Eskom's power stations as part of understanding the true costs of electricity generation. The valuation of morbidity outcomes was found to be small in terms of costs per kWh generated.

- Scale: National (Air Pollution from Coal-fired Power Plants)
- Resolution: Medium
- Health Outcomes: Morbidity and Mortality
- Modelling Approach: Bottom-up

The Fund for Research into Industrial Development Growth and Equity, in 2004, assessed the economic impact of air pollution in selected areas in South Africa. The study found that power generation was responsible for 51% of the 8,700 respiratory cases in Mpumalanga (FRIDGE, 2004).

- Scale: Selected Areas (All Air Pollution and Air Pollution from Power Plants)
- Resolution: Medium
- Health Outcomes: Morbidity and Mortality
- Modelling Approach: Bottom-up

A review by Spalding-Fecher and Matibe in 2003 aimed to calculate the external costs of electric power generation in South Africa. They estimated the health costs to be R1.1 billion per year (Spalding-Fecher and Matibe, 2003).

- Scale: National (Air Pollution from Power Plants)
- Resolution: Low
- Health Outcomes: Morbidity and Mortality
- Modelling Approach: Top-down.

The methodology used in this investigation is discussed in detail in Section 2 below.

2. METHODOLOGY AND INPUTS

2.1. Overview

An integrated Health CBA Model was developed that combined an AP-HRA with a CBA to assess four air pollution mitigation scenarios for 13 Eskom coal-fired power stations.

Figure 3 below provides an overview of the methodology, and Sections 2.2 - 2.5 provide a more detailed discussion of each component.

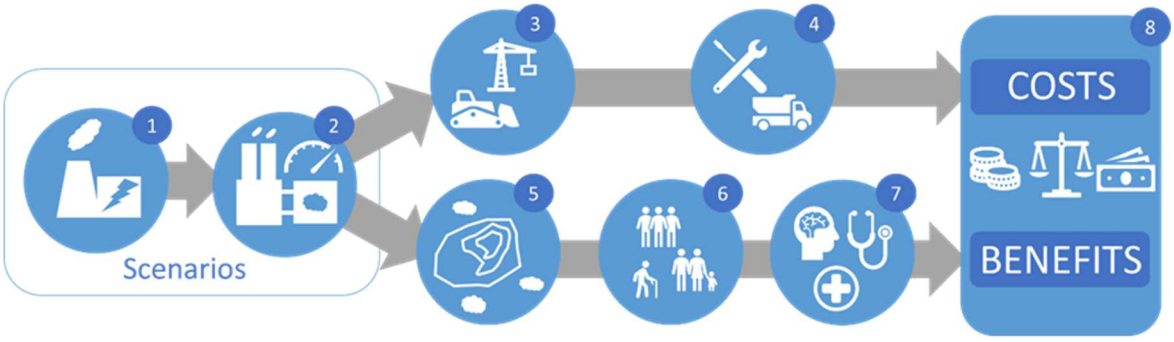

Figure 3 Overview of methodology and model architecture

With reference to Figure 3, the integrated Health CBA Model includes the following components:

1. Plant lifetimes were described for 13 coal-fired power plants and included commissioning and decommissioning dates (provided by Eskom) (Table 5).
2. Abatement technologies required for each scenario were defined, by type and likely implementation schedule (Figure 23 - Figure 26).
3. Capital expenditure required for abatement in each scenario was attributed per plant and per year using Eskom's internal estimates (Section 2.3.3).
4. Operational expenditure required for abatement in each scenario was attributed per plant and per year using Eskom's internal estimates (Section 2.3.3).
5. Dispersion modelling results were obtained as part of the broader Eskom investigation (Zunckel and Raghunandan, 2018). This data was segregated spatially, by ward and municipal boundaries to align with population data. Two sets of dispersion modelling data were obtained. The first set modelled predicted ambient concentrations of PM, NO_2 and SO_2 around individual power stations. The second set modelled cumulative predicted ambient concentrations of PM, NO_2 and SO_2 from all power stations on the Highveld. The dispersion modelling results were unique because in addition to primary PM, the modelling predicted secondary PM effects, resulting from NO_2 and SO_2 reactions in the atmosphere (refer to Section 2.2).
6. Population exposure was estimated at a spatial resolution of municipality and municipal wards. At each municipality or ward, the number of people exposed to different concentration ranges were determined per scenario per year, based on Stats SA population estimates and United Nations population growth forecasts (refer to Section 2.2).
7. Health impacts were determined by using the AP-HRA methodology. Epidemiological evidence, in the form of ERFs and baseline incidence rates were provided by the SA Medical Research Council (SAMRC) (Wright and Oosthuizen, 2018) (refer to Section

2.3). The ERFs were limited to mortality incidence. The COI methodology used was the value of statistical life (VSL). This method estimates the willingness to pay (WTP) of an individual for reducing their health risk. The VSL should not be interpreted as the intrinsic value of a life. Refer to Section 2.3.3 for a more detailed discussion.

8. The CBA compares the overall scenario benefits and costs. The outputs of the AP-HRA, the health cost savings of each scenario, was used as the benefit. The capital and operational cost estimates were used as the costs in the CBA. The analysis timeline spans 2015 – 2045. This timeframe allows for 5-year interval analysis, aligning to the 2020 MES. It also captures mitigation activities implemented since 2016. The base year was 2018, due to dispersion modelling timeframe. The CBA was performed in an Excel spreadsheet, which consolidated all data sources, which contains all calculations, and was macro-enabled to run the large spatial exposure estimates for each scenario for the review period (refer to Section 2.4). Finally, an assessment of uncertainty of the results was done (refer to Section 2.5).

2.2. Exposure of the target population to specific air pollutants

2.2.1. Overview

This section comprises the first step of the AP-HRA. It assesses the exposure of the target population to specific air pollutants.

This requires an incremental effects quantification of constituents in the atmosphere that are associated with human health risks. These pollutants include PM, NO_2 and SO_2 emitted by the 13 coal-fired power plants investigated. The emissions from these plants impact the Highveld Priority Area (HPA).

Dispersion modelling combined with population distribution provided an estimate of exposed population.

2.2.2. Pollutants analysed

A network of five ambient monitoring stations was established by the Department of Environmental Affairs (DEA) in the HPA in 2008. Currently a 9-year data record of SO_2, NO_2 and PM exists. The ambient monitoring stations are located at Ermelo, Hendrina, Middelburg, Secunda and Witbank. An overview of the state of air quality in the HPA follows, per pollutant (Naidoo et al., 2018).

Sulphur dioxide (SO_2)

Industrial processes and power generation are the main source of SO_2 in the atmosphere through the combustion or refining of sulphur containing fuels. In the HPA the annual average ambient SO_2 concentrations are relatively high, and the annual average concentration is consistently more than 50% of the NAAQS of 19 ppb. The highest concentrations occur in Witbank where the NAAQS was exceeded in 2010 and 2011, and the annual average concentration is 90% of the NAAQS in most other years (Figure 4).

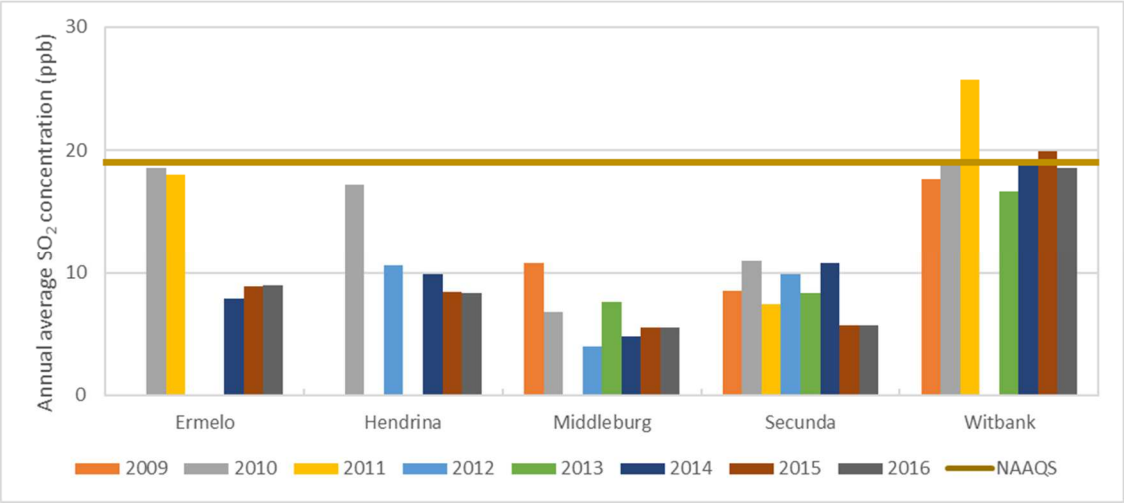

Figure 4 Annual average SO_2 concentrations at the DEA-owned monitoring stations in the HPA in ppb

Nitrogen dioxide (NO_2)

Industrial processes and power generation are the main source of NO_2 in the atmosphere through the combustion or refining of fossil fuels, with some contribution from motor vehicle emissions, residential fuel burning and biomass burning. In the HPA the annual average ambient NO_2

concentrations are relatively low compared to the NAAQS, except at Secunda in 2009 and Witbank in 2016 where exceedances occurred (Figure 5).

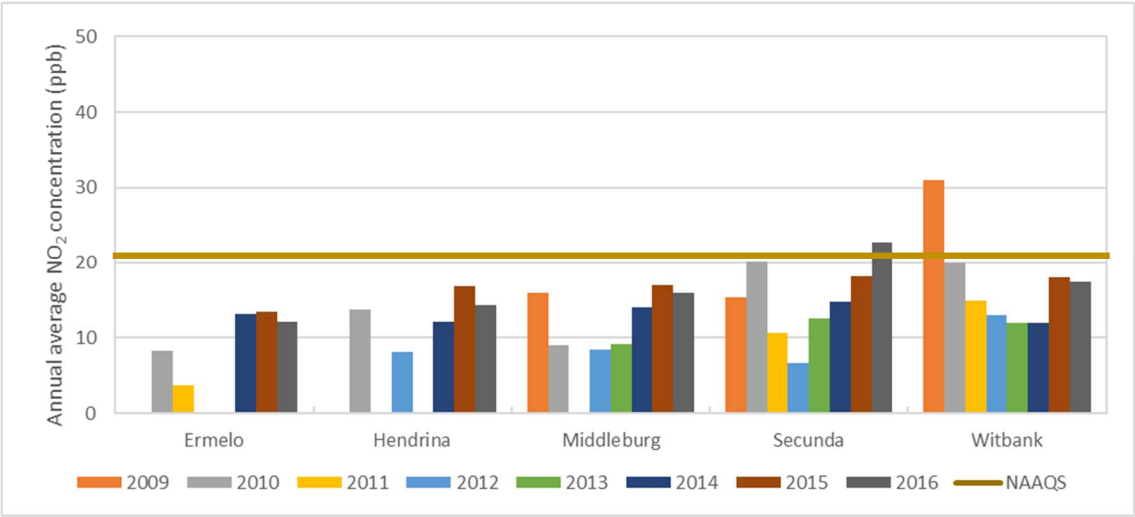

Figure 5 Annual average NO_2 concentrations at the HPA monitoring stations in ppb

Particulate matter (PM)

There are numerous sources of primary particulate matter, including power generation, industry, mining, biomass burning and agricultural, as well as natural sources such as wind entrainment. In addition, secondary PM is produced by NO_2 and SO_2 reactions in the atmosphere. In this study all PM assessed was below 2.5 microns, i.e. $PM_{2.5}$.

The annual average $PM_{2.5}$ concentrations are high at the HPA monitoring stations relative to the NAAQS (25 µg.m^{-3} prior to 2016 and 20 µg.m^{-3} thereafter). The exception is Hendrina and the Secunda stations. The 9-year monitoring record for the HPA monitoring stations also shows the consistent exceedances of the NAAQS, although there is evidence of a decreasing trend at the monitoring stations since 2012 (Figure 6).

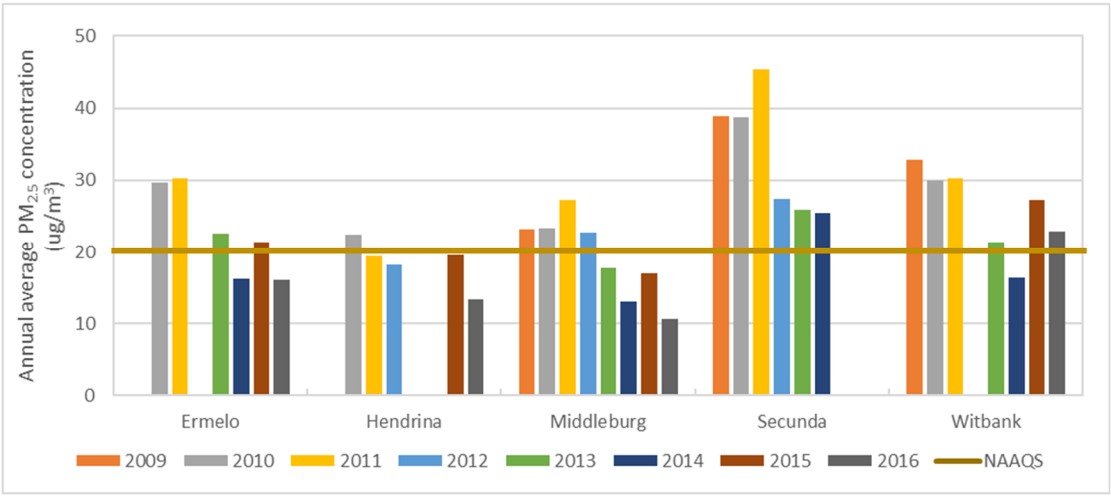

Figure 6 Annual average $PM_{2.5}$ concentrations for the HPA in µg.m^{-3}

2.2.3. Description of power plants

Eskom has indicated that the MES cannot be achieved at 13 coal-fired power stations and plans to apply for an extension of the conditions (Zunckel and Raghunandan, 2018). These power stations have a combined installed capacity of 38 510 MW and are listed in Table 2.

Table 2 Eskom coal-fired power stations, used in this study, and their installed capacity (Zunckel and Raghunandan, 2018).

Power Station	Province	Installed capacity (MW)
Arnot	Mpumalanga	2 352
Camden	Mpumalanga	1 561
Duvha	Mpumalanga	3 600
Grootvlei	Mpumalanga	1 180
Hendrina	Mpumalanga	1 893
Kendal	Mpumalanga	4 116
Komati	Mpumalanga	990
Kriel	Mpumalanga	3 000
Kusile	Mpumalanga	4 800
Lethabo	Free State	3 708
Majuba	Mpumalanga	4 110
Matla	Mpumalanga	3 600
Tutuka	Mpumalanga	3 600

2.2.4. Dispersion modelling

Dispersion modelling is required to estimate the effects of stack emissions on ambient concentrations of pollutants and describe them spatially.

uMoya-Nilu Consulting conducted the dispersion modelling and followed the requirements of the DEA guideline for dispersion modelling (DEA, 2014).

The work modelled the dispersion of primary and secondary particulate matter ($PM_{2.5}$), nitrogen dioxide (NO_2) and Sulphur dioxide (SO_2), for each power plant, as well as cumulatively. (Zunckel and Raghunandan, 2018). Dispersion modelling was performed using the CALPUFF suite of models. CALPUFF is a multi-layer, multi-species non-steady-state puff dispersion model that simulates the effects of time and space-varying meteorological conditions on pollution transport, transformation and removal. It includes algorithms for sub-grid scale effects, such as terrain effect, as well as longer range effects, such as pollutant removal due to wet scavenging and dry deposition, chemical transformation, and the formation of secondary particulate matter. The Air Pollution Model (TAPM) was used to model surface and upper air metrological data for the study domain.

Two types of analysis were performed, individual and cumulative models. Individual plant dispersion modelling results had smaller modelling domains (4 356 km^2; 66 km by 66 km), while the cumulative modelling had large domains (97 200 km_2; 360 km by 270 km). Because of the much larger modelling domain of the cumulative model, it was used to calculate the status quo health impacts. The cumulative assessment however only offers a static "snapshot" of predicted ambient concentrations and does not allow the effects of different scenarios to be modelled. The individual dispersion results are less useful for calculating health effects because of their smaller modelling domains (covering smaller populations), however individual models are useful for assessing the impacts of changes in scenarios, as pollution from individual plants can be evaluated. As such, integrated Health CBA Model used the individual models to estimate relative changes between scenarios and years, applied to the health costs derived from the cumulative models.

Individual Models: Primary and secondary $PM_{2.5}$ was combined to estimate total $PM_{2.5}$. Two of the emission scenarios were used; (i) the "Current"; and (ii) the "Compliance" scenario, that described predicted ambient pollution concentrations in the region attributable to the 13 plants that were modelled. For the health benefits analysis, it was assumed that all power plants will emit "Current" emissions until abatement technologies are installed, from which time they will emit "Compliance" emissions. Examples of the "Current" predicted annual average concentrations based on actual emissions and assuming new plant MES are presented in the Figure 7 to Figure 14.

Cumulative Models: Primary and secondary $PM_{2.5}$ was combined to estimate total $PM_{2.5}$. This analysis only used (i) the "Current"; scenario, that described cumulative predicted ambient pollution concentrations in the region attributable to the 13 plants that were modelled. These models were used to estimate the status quo health costs. The "Current" predicted annual average concentrations based on actual emissions are presented in the Figure 15 to Figure 18.

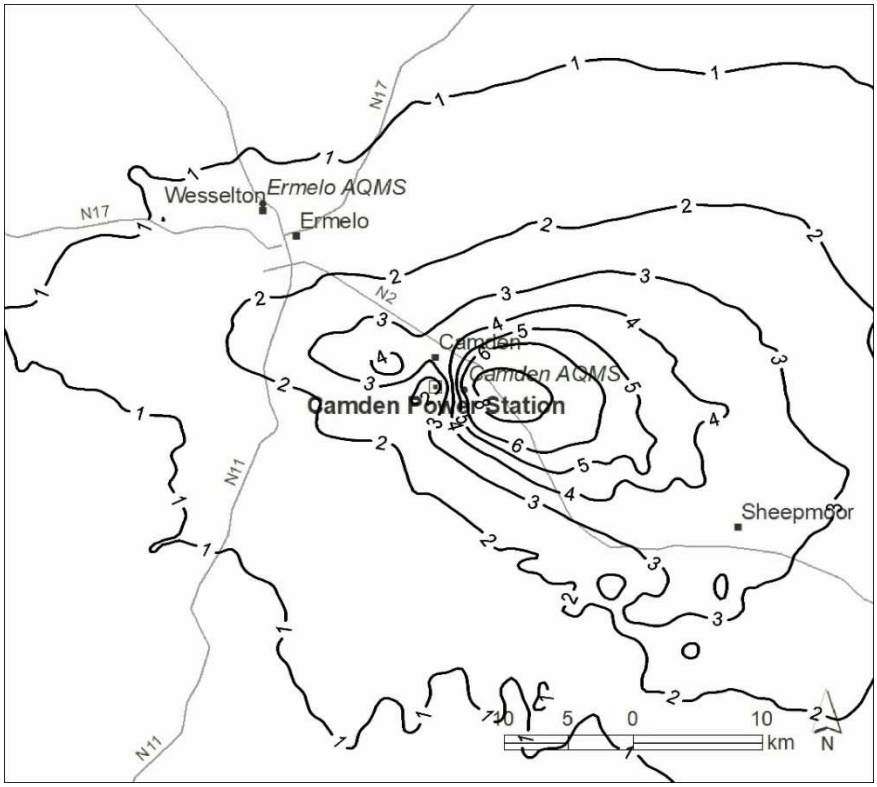

Figure 7 Predicted annual average SO_2 concentrations (µg/m³) resulting from actual emissions for Camden Power Station (Current)

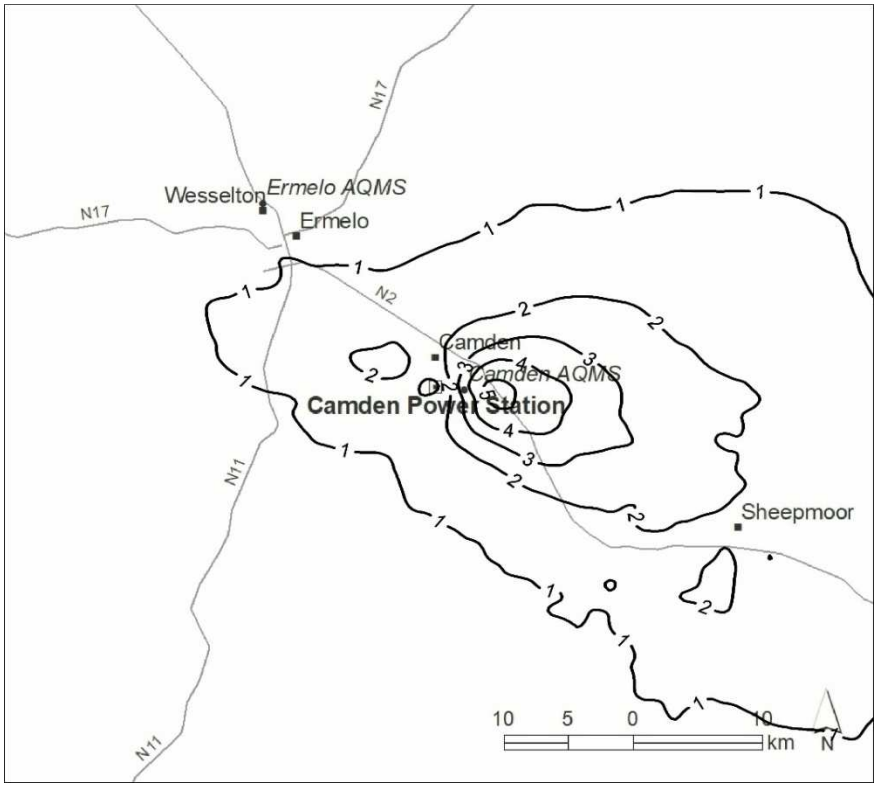

Figure 8 Predicted annual average SO_2 concentrations (µg/m³) assuming new plant MES from Camden Power Station (Compliance)

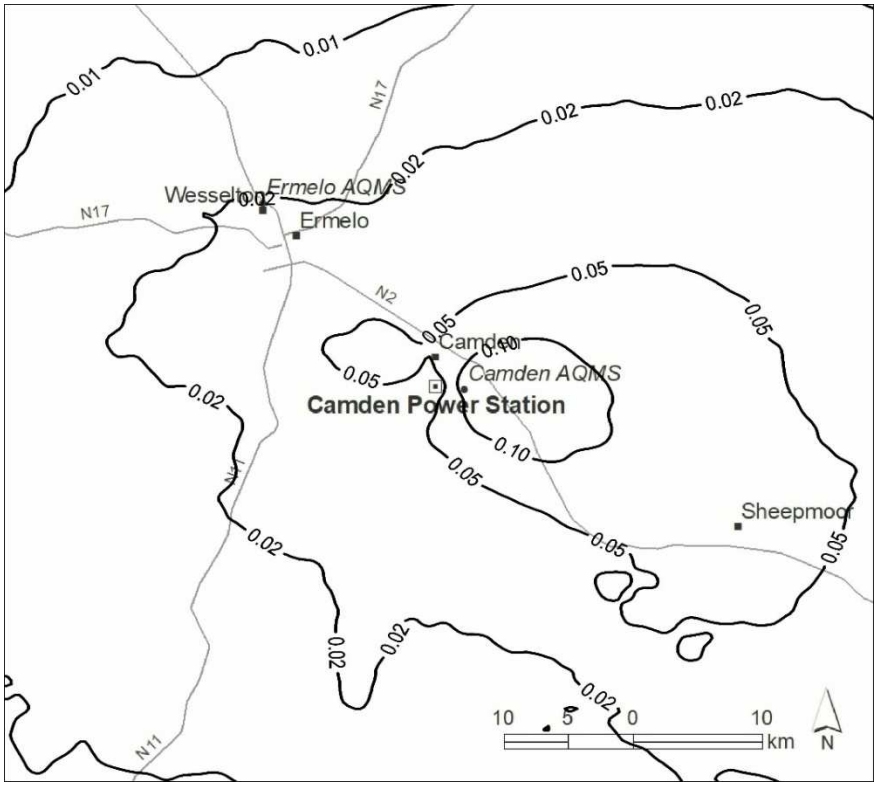

Figure 9 Predicted annual average $PM_{2.5}$ concentrations (µg/m³) resulting from actual emissions for Camden Power Station (Current)

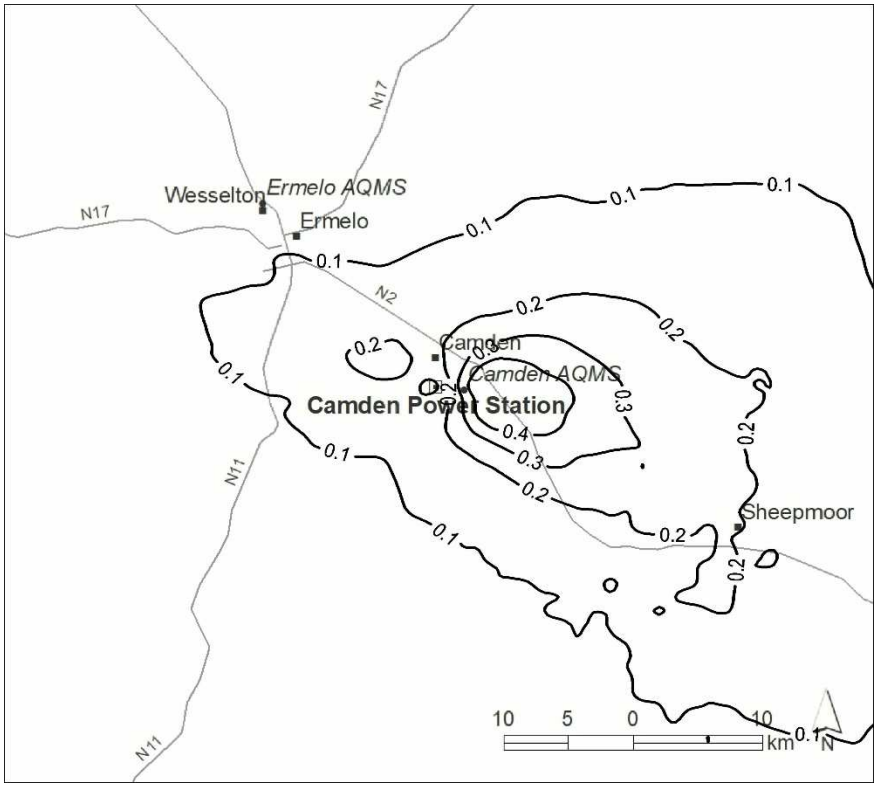

Figure 10 Predicted annual average $PM_{2.5}$ concentrations (µg/m³) assuming new plant MES from Camden Power Station (Compliance)

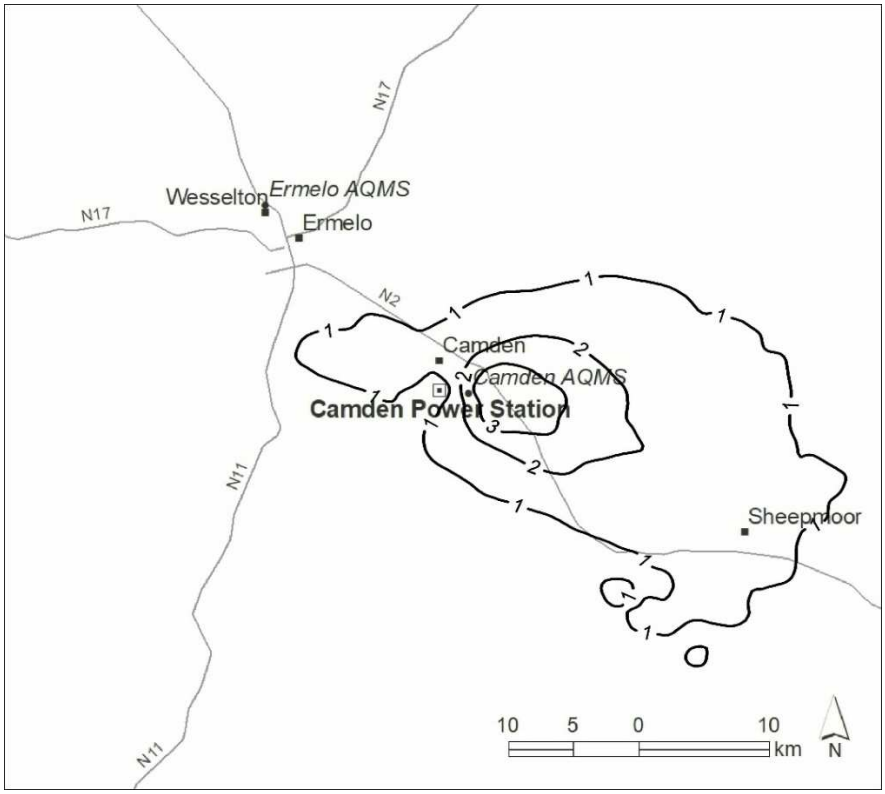

Figure 11 Predicted annual average NO_2 concentrations (µg/m^3) resulting from actual emissions for Camden Power Station (Current)

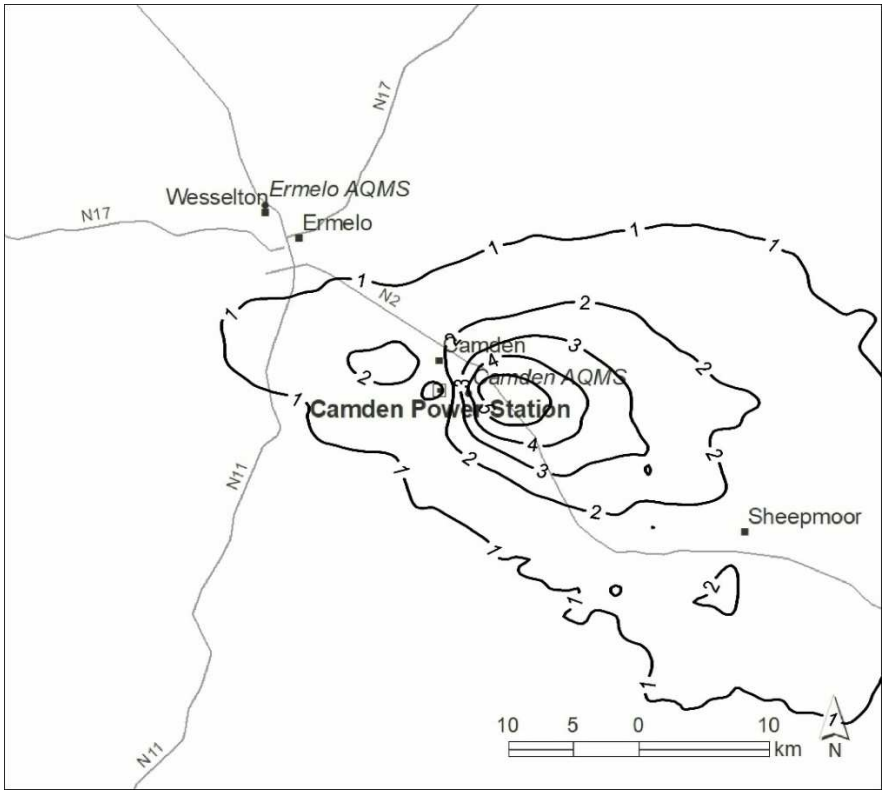

Figure 12 Predicted annual average NO_2 concentrations resulting assuming new plant MES from Camden Power Station (Compliance)

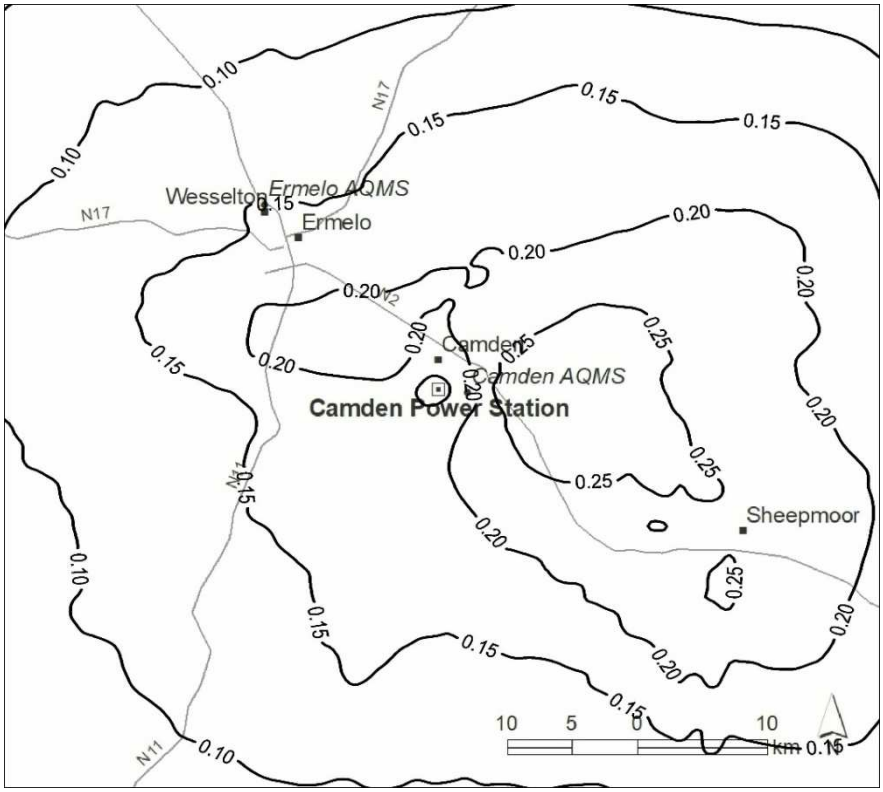

Figure 13 Predicted annual average secondary particulate concentrations (µg/m³) resulting from actual emissions for Camden Power Station (Current)

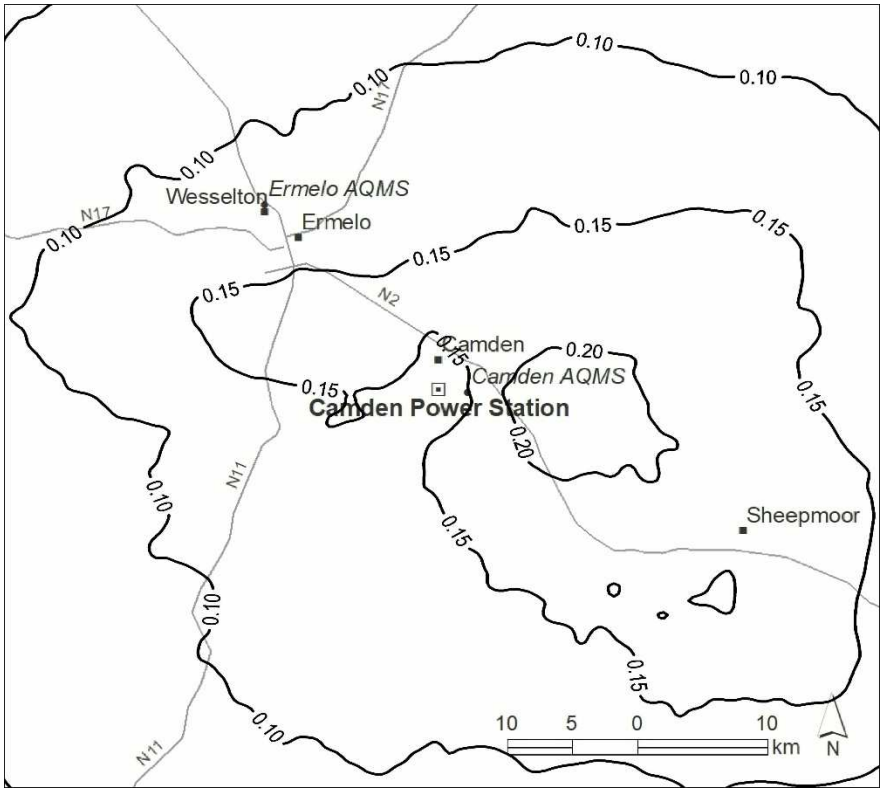

Figure 14 Predicted annual average secondary particulate concentrations (µg/m³) resulting, assuming new plant MES from Camden Power Station (Compliance)

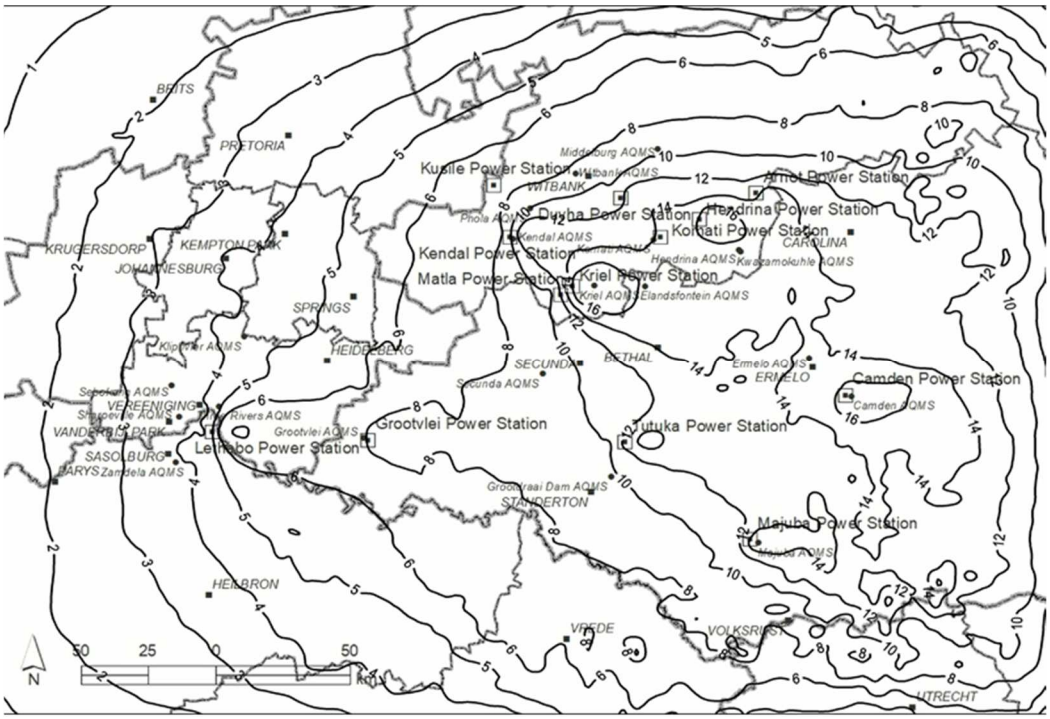

Figure 15 Predicted annual average SO_2 concentrations (µg/m^3) resulting from actual emissions from the 13 coal-fired power stations (Zunckel and Raghunandan, 2018)

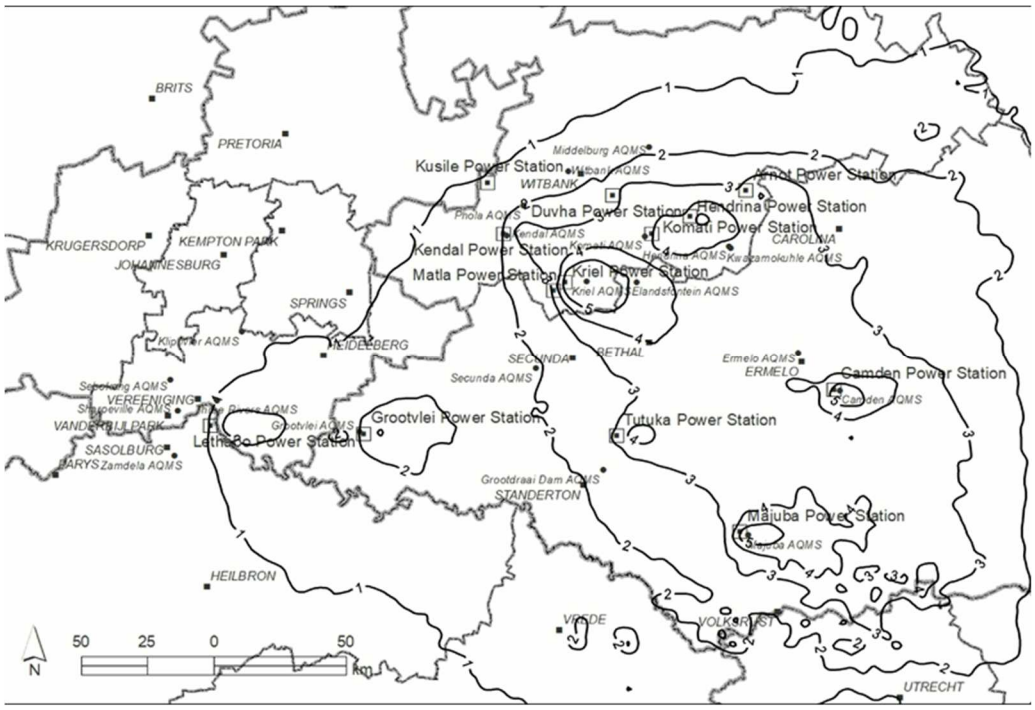

Figure 16 Predicted annual average NO_2 concentrations (µg/m^3) resulting from actual emissions from the 13 coal-fired power stations (Zunckel and Raghunandan, 2018)

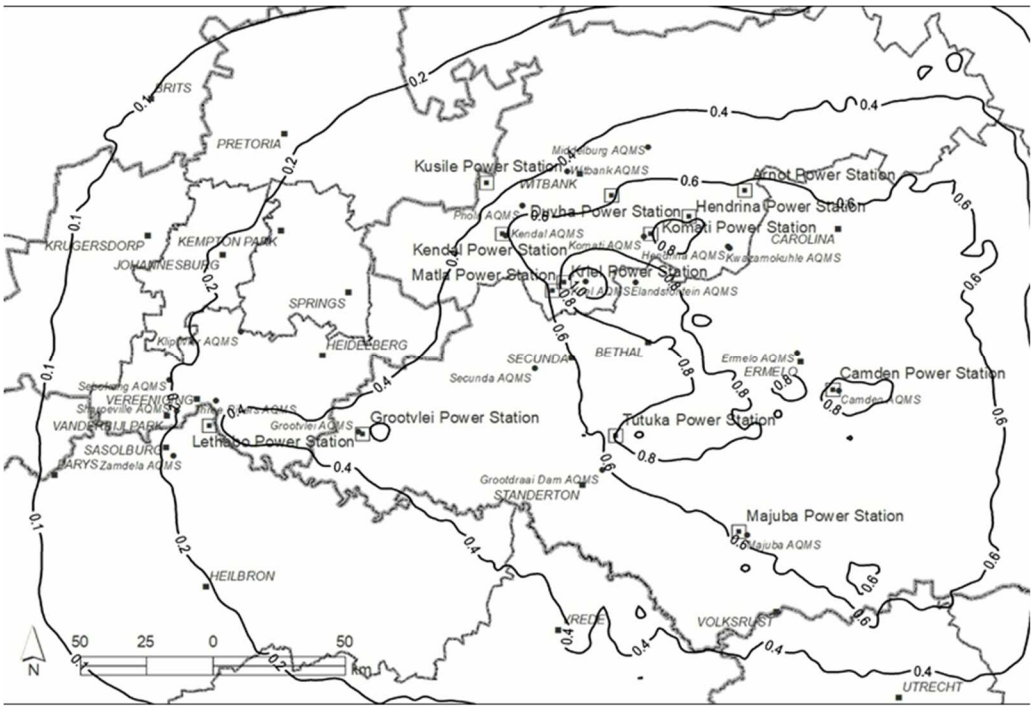

Figure 17 Predicted annual average $PM_{2.5}$ concentrations (µg/m³) resulting from actual emissions from the 13 coal-fired power stations (Zunckel and Raghunandan, 2018)

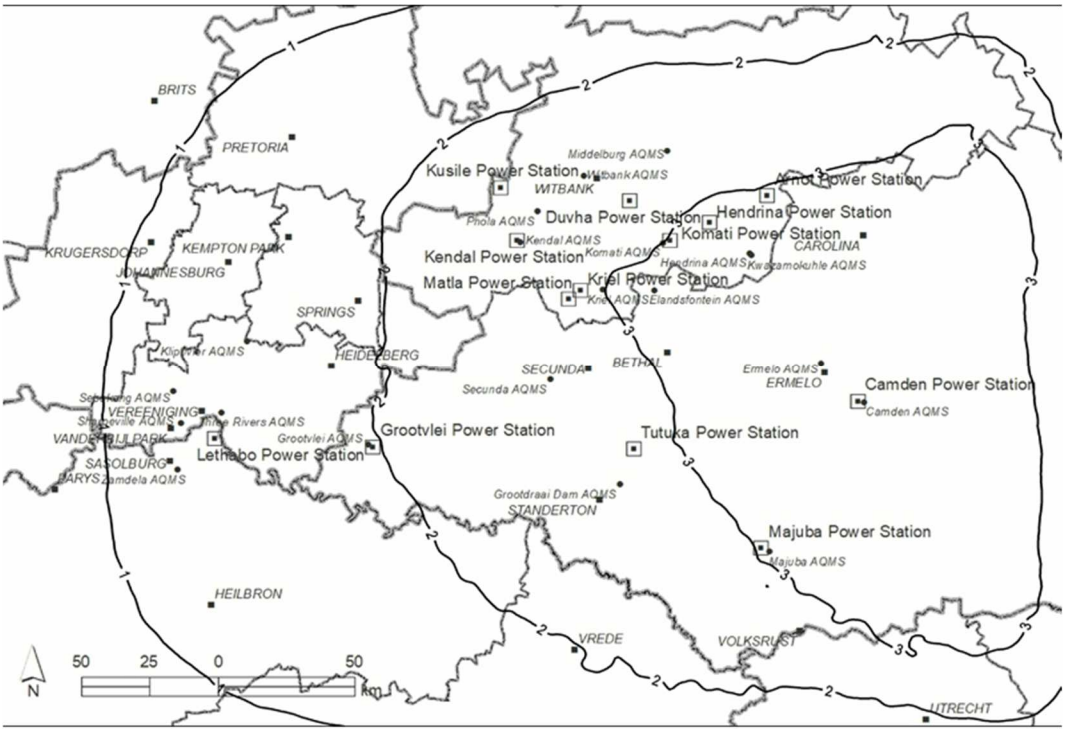

Figure 18 Predicted annual average secondary particulate concentrations (µg/m³) resulting from actual emissions from the 13 coal-fired power stations (Zunckel and Raghunandan, 2018)

2.2.5. Population exposure

Population exposure was estimated at a spatial resolution of municipality and municipal wards. At each municipality or ward, the number of people exposed to different concentration ranges were determined per scenario per year, based on Stats SA population estimates (Stats SA, 2012; Stats SA, 2018) and United Nations population growth forecasts (United Nations, 2017).

Population exposure was estimated at a spatial resolution of municipality (cumulative model) and municipal wards (individual models). At each ward, the number of people exposed to different concentration ranges for each pollutant were determined per scenario per year. Primary and secondary particulate matter was summed to estimate total exposure at each spatial unit.

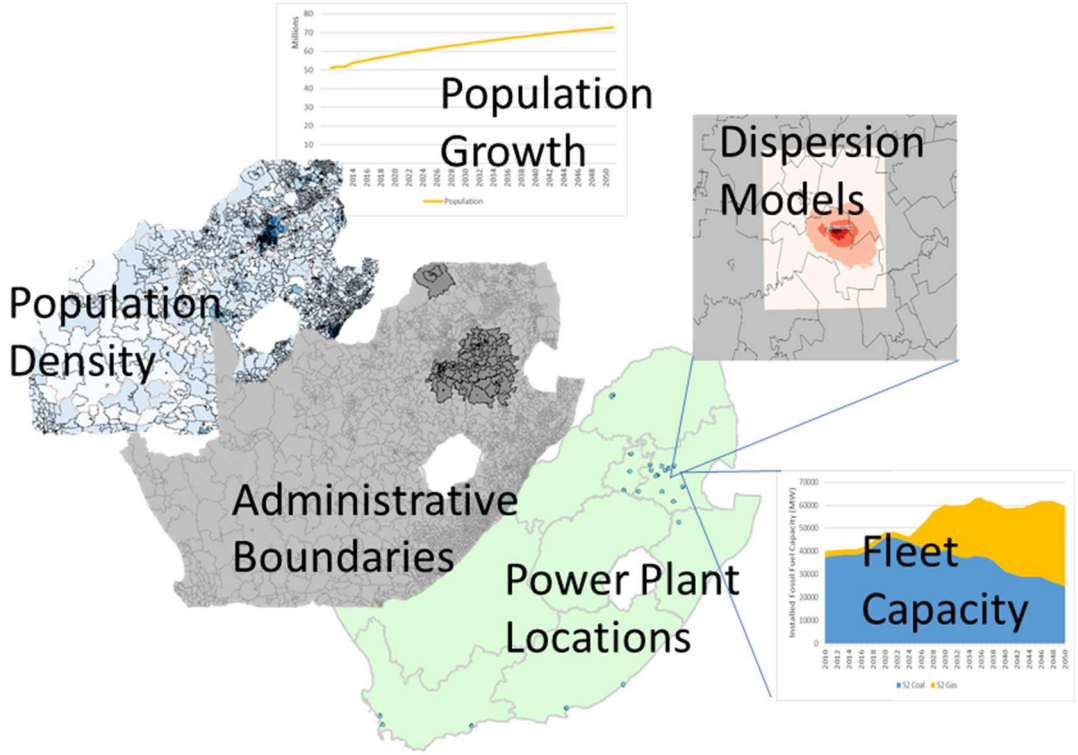

Figure 19 Overview of population exposure calculation

The integrated Health CBA model calculated pollution exposure as follows:

- Dispersion Model outputs were used to spatially apportion pollution concentrations. The co-ordinates (x;y) of receptors from the output files were attributed to specific administrative boundaries.
- Administrative boundaries used were municipalities and municipal wards. The predicted ambient concentrations for each pollutant was averaged for the entire spatial unit.
- Population density (population per ward) was obtained from the Census 2011 (Stats SA, 2018).
- Total population was obtained from mid-year population estimates (Stats SA, 2018).

- Power plant locations were used to determine the wards which were affected by each plant, to estimate relative impacts of each power plant to the cumulative impact modelled.
- Fleet capacity (Table 5) was used to determine whether power plants were operational in each year. If abatement technologies were installed, they had "Compliance" ambient pollution concentrations.
- Population growth forecasts were used to determine the growth in population exposure over time (United Nations, 2017).

Approximately 17.7 million people were population exposed to more than an additional 1µg.m^3 (mean annual average) of $PM_{2.5}$ due to the 13 power plants. Similarly, 15.3 and 19.0 million people were exposed to more than an additional 1µg.m^3 of NO_2 and SO_2, respectively.

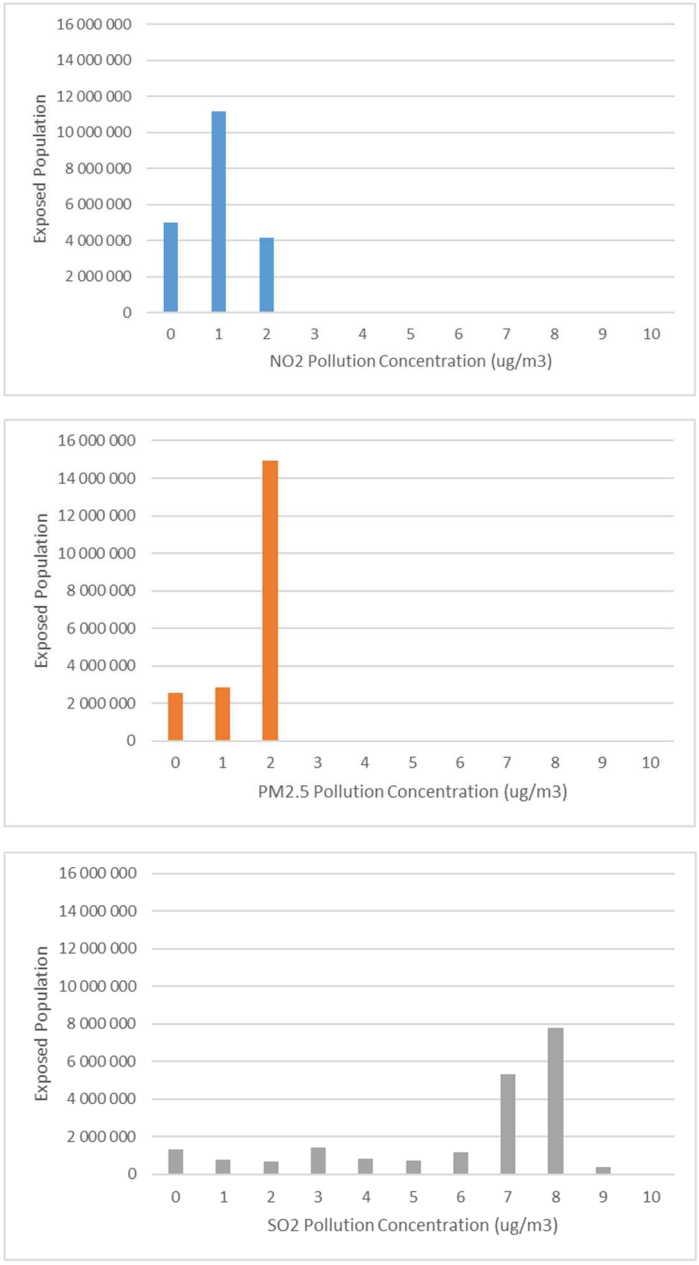

Figure 20 Population exposure to NO_2, $PM_{2.5}$ and SO_2 mean annual average concentration ranges.

2.3. Incremental change in health risk

2.3.1. Health impacts

The WHO (2016) recommends that the health risk in a population, associated with air pollution, is to be estimated using exposure-response functions (ERFs). ERFs are based on Relative Risk (RR) estimates derived from primary epidemiological studies.

These RR functions estimate the likelihood of health outcomes occurring in a population exposed to a higher level of air pollution relative to that in a population with a lower exposure level (WHO, 2016). RR is usually expressed as the proportional increase in the assessed health outcome associated with a given increase in pollutant concentrations, measured in $\mu g/m^3$. The WHO (2016) notes that "*the RR estimate cannot be assigned to a specific person; it describes risk in a defined population, not individual risk.*"

The RRs are derived from epidemiological studies. These are mostly based on evidence from population health studies that compare health outcome incidences of populations exposed to higher levels of air pollution to populations exposed to lower levels of air pollution. Most of these studies have been done in Europe and North America.

The health outcomes used in this study were specified by the SAMRC based on an international benchmarking exercise, and included commonly associated diseases such as cerebrovascular, respiratory and cardiovascular mortality (Wright and Oosthuizen, 2018). In addition, the MRS report also identified an emerging health outcome linked to air pollution, in the form of diabetes mellitus mortality.

Respiratory Mortality: The lung is the internal organ most vulnerable to infection and injury from the external environment because of its constant exposure to ambient air. Respiratory mortality includes deaths due to chronic obstructive pulmonary disease (COPD), asthma, acute lower respiratory tract infections, tuberculosis (TB), and lung cancer (FIRS, 2017). Diseases of the respiratory system make up 9.4% of total deaths in South Africa (StatsSA, 2018).

Cardiovascular Mortality: Cardiovascular mortality includes death attributable to myocardial ischemia and infarction, heart failure, ischaemic heart disease and cardiac arrest. Cardiovascular diseases fall within International Classification of Diseases (ICD) codes I00-I152. Long-term exposure to nitrogen dioxide is associated with increased cardiovascular mortality (Maji et al., 2017). In South Africa, diseases of the cardiovascular system make up 18.5% of total deaths (SAMRC, 2016).

Cerebrovascular Mortality: Stroke, transient ischemic attack, aneurysms, and vascular malformations are all types of cerebrovascular disease. Cerebrovascular diseases have ICD codes I60-I69. Exposure to particulate matter is associated with increased cerebrovascular mortality (Gutiérrez-Avila et al., 2018). Cerebrovascular diseases are responsible for 5.7% of natural deaths in South Africa (StatsSA, 2018).

Diabetes Mellitus Mortality: Diabetes mellitus is a group of diseases where a person has high blood glucose (blood sugar), either because insulin production is inadequate, or because the body's cells do not respond properly to insulin, or both. Diabetes diseases have a classification of E10 (Type 1) and E11 (Type 2) in the International Statistical Classification of Diseases (ICD) (WHO, 2016b). Long-term exposure to particulate matter is associated with a significant increase in diabetes-related mortality (Brook et al., 2013). Diabetes Mellitus is estimated to be responsible for 6.2% of natural deaths in South Africa (StatsSA, 2018).

The baseline incidence rates of these health outcomes, as well as the ERFs describing the change in incidence in relation to changes in exposure (RRs) were also provided by the Medical Research Council. Figure 22 demonstrates how the relative risks for each of these health outcomes are related to pollution concentrations.

The baseline incidences were estimated by the SAMRC based for either 2012 or 2016, based on Stats SA data.

Ideally, ERF studies and their RRs should be determined based on primary epidemiological studies focussing on the exposed population. In the absence of such studies, as in the case of South Africa, the WHO (2016) recommends using ERFs from other countries. The SAMRC provided a number of ERF options for South Africa, as set out in Table 3.

It is to be noted that there are inherently significant limitations in transferring ERF studies from other countries. Pollution levels, chemical composition and health care systems are typically very different in other settings, and this would affect the accuracy of the ERFs.

It is important to understand at what level interval the ERFs would result in significant differences in health outcome incidences. It is also to be noted that the ERFs proposed by the SAMRC were all specified for relatively coarse intervals in ambient concentrations of 10 μg/m^3. These are relative coarse bands and have to be applied to the changes in annual average ambient concentration estimated by the dispersion modelling, which are in the order of 1-3 μg/m^3. Figure 21 provides a sensitivity analysis that demonstrates how the choice of finer pollution concentration interval affects the health incidence estimates. At 10 μg/m^3 intervals, at which the ERFs are specified, no health effects are visible. This is because the changes in ambient concentrations modelled are smaller than 10 μg/m^3. At a finer interval of 0.75 μg/m^3, much larger health effects are visible. This study adopted a conservative approach favouring higher health costs per incidence by assuming ERFs are significant at 0.2 μg/m^3 intervals

Figure 21 can also be used to demonstrate an example of how an ERF is applied. In 2018, 4.17 million people were exposed to an additional 2 μg/m^3 from the 13 power stations modelled. Cerebrovascular mortality has a baseline incidence rate of 0.0413%, meaning that we would expect 2792 mortalities out of the 4.17 million people in that year. However, the incidence of cerebrovascular mortality increases by 11% (from baseline incidence) for every 10 μg/m^3 increase in $PM_{2.5}$ exposure, so a 2 μg/m^3 increase results in a new incidence rate of 0.0422% (0.0413%×1.11^(2/10)). This extra 0.00087% incidence or 36.4 cases of cerebrovascular mortality is then attributable to $PM_{2.5}$ from the 13 power plants modelled. Similarly, this method was applied to all pollutants and exposed population ranges and the increased exposure is estimated to result in an additional 334 cases of premature respiratory, cardiovascular and cerebrovascular mortality attributed to air pollution from the 13 power stations in 2018.

In the AP-HRA, each health outcome must be attributed to an individual indicator pollutant. While health outcomes can be attributed to many different indicator pollutants, using all would result in double accounting of health impacts as these pollutants are associated with each other. For instance, there are three ERFs for respiratory mortality health outcome, respectively for $PM_{2.5}$ (WHO, 2014), for SO_2 (based on a study from India (Maji et al., 2017)), and for NO_2 (based on a study from Holland (Fischer et al., 2015)). These three ERFs give widely varying results. For the purpose of this study, the Indian example, which gave mid-range incidence was selected. Ischaemic heart disease mortality was excluded from the analysis as it is a component of cardiovascular mortality. Variation in health

outcome incidences between the various ERFs provided in some cases exceed 80%. This variation was dealt with through performing sensitivity analysis in the CBA (refer to section 2.4).

For this reason, the following four ERFs were selected for evaluation in the AP-HRA:

- Respiratory mortality using SO_2 as an indicator pollutant
- Cardiovascular mortality using NO_2 as an indicator pollutant
- Cerebrovascular mortality using $PM_{2.5}$ as an indicator pollutant
- Diabetes mellitus mortality using $PM_{2.5}$ as an indicator pollutant.

Figure 22 demonstrates how the relative risks for each of these health outcomes are related to pollution concentrations.

Table 3 Indicator pollutants, baseline incidence, relative risks, and costs of each health outcome (Source: SAMRC, Table meeting 15 August 2018)

Indicator Pollutant	Health Outcome	Baseline data	Relative Risk or Hazard Ratio per 10µg/m^3	Reference
$PM_{2.5}$	Diabetes Mellitus Mortality	5.5% of total deaths	Hazard ratio 1.13	Pope III et al., 2015
	Cerebrovascular Mortality	5.10% of total deaths	Hazard ratio 1.11	Pope III et al., 2015
	Respiratory Mortality	9.40% of total deaths	RR 1.10	WHO, 2014
	Ischaemic Heart Disease Mortality	2.80% of total deaths	RR 1.05	Burnett et al., 2014
	Cardiovascular Mortality	18.5% of total deaths	Hazard ratio 1.12	Pope III et al., 2015
SO_2	Cardiovascular mortality	18.5% of total deaths In 2012	RR: 1.0103	Maji et al., 2017
	Respiratory Mortality	9.40% of total deaths	RR:1.0106	Maji et al., 2017
NO_2	Respiratory Mortality	9.40% of total deaths	Hazard Ratio 1.02	Fischer et al., 2015
	Cardiovascular mortality	18.5% of total deaths In 2012	RR: 1.0206	Maji et al., 2017

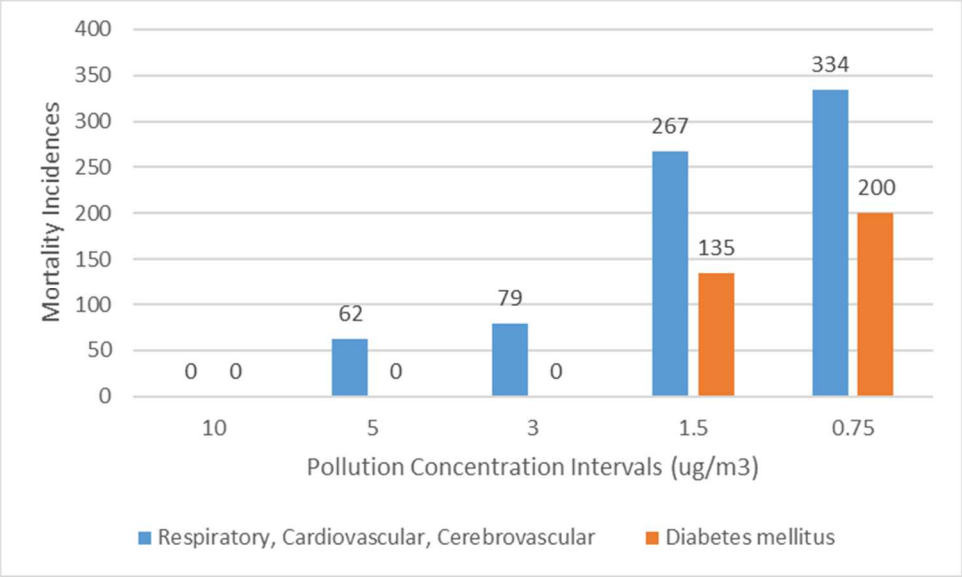

Figure 21 Sensitivity analysis of the effect of pollution concentration intervals on predicted mortality incidence.

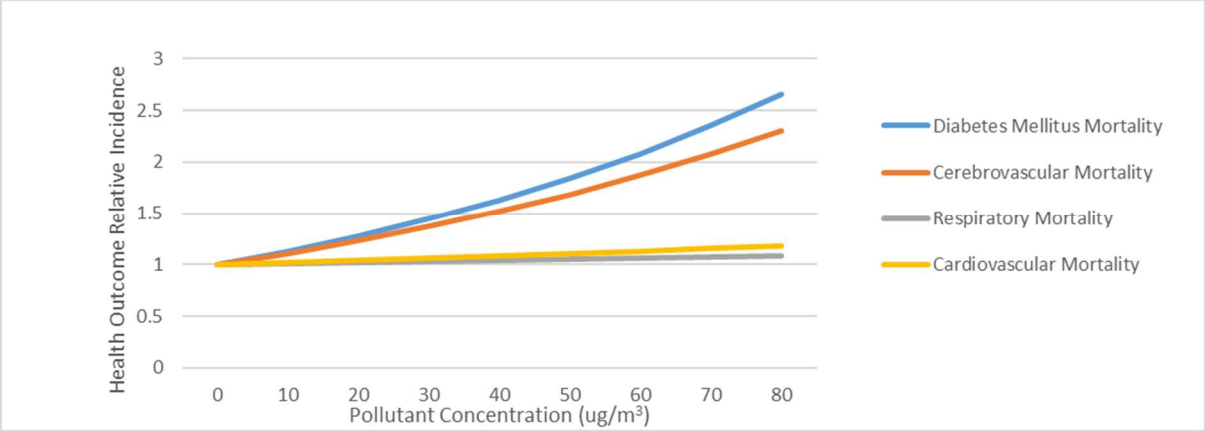

Figure 22 Exposure-response functions for health outcomes related to air pollution

2.3.2. Health costs

The detrimental effects of air pollution on human health is borne in the economy by households, insurance companies, employers and public health programs (Romley et al., 2010).

The fundamental goal of health cost or cost of illness (COI) studies is to evaluate the economic burden that illness imposes on society as a whole (Jo, 2014). Rice (1967) and Rice et. al (1985), were instrumental in standardising methodologies for estimating COI, and these methodologies continue to be used internationally, and periodically updated (Rice, 1996; Rice, 2000).

COI studies contextualise adverse diseases effects into monetary terms, with the purpose of informing decision-making. Such decisions could include (a) to simply present the magnitude of disease in monetary terms; (b) to comparatively evaluate intervention programs; (c) to assist in the allocation of research funding on specific diseases; (d) to provide a basis for policy and planning relative to mitigation initiatives; and (e) to provide an economic framework for program evaluation (Rice, 2000).

The COI studies traditionally stratify costs into two categories: direct costs and indirect costs. Direct costs relate to the cost of medical treatment. This would include costs of visiting health care facilities, medicine and hospitalisation. Indirect costs comprise morbidity costs (the cost of lost economic productivity due to absenteeism or temporary or permanent disability) and mortality costs. With respect to mortality costs, valuing human life is contentious, as it can be seen as a judgement on the intrinsic value of life and involves complex ethical considerations. Often, cost-effectiveness analysis is used as an alternative (Muchapondwa, 2009). This side-steps the complexity of life valuation and uses disease or fatality incidence indicators to compare effectiveness of different policy or spending options.

In this study, the ERFs provided by the SAMRC, focussed exclusively on mortality and thus a monetary measure of mortality was required in order to perform cost-benefit analyses. In air pollution cost-benefit analyses, the concept of value per statistical life (VSL) is commonly used to monetise mortality related benefits of air pollution reduction. The concept of a VSL is frequently misunderstood. It does not measure the intrinsic value of a human life, and neither does it value the economic productivity of a human. Rather, VSL is estimated by dividing an individual's willingness to pay (WTP) to reduce health risk, by the likelihood of risk reduction. Robinson and Hammitt (2009) defines VSL to represent the rate at which an individual is willing to exchange their own income for a small reduction in their own mortality risk over a particular time period. By example, if an individual is willing to pay R5 to reduce her/his annual mortality risks by 1 in 10,000, the VSL is R50,000 (R5 ÷ 1/10,000). Primary WTP studies for mortality risk reductions has not been done in South Africa. However, three studies have made VSL estimates for South Africa, all based on extrapolation of United States studies. These values, when adjusted to 2018, vary between R21 million and R48 million (Table 4).

A thesis on evaluating the costs and benefits associated with the reduction in SO_2 emissions from industrial activities on the Highveld of South Africa (Grobler, 2016), provided the most conservative of these estimates (i.e. the highest VSL) of R 53 Million (in 2020). This value adjusted to 2018 was used in the CBA.

Table 4 VSL estimates for South Africa from literature

Study	Method	VSL (US$)	VSL Adjusted to 2018
World Bank; Robinson and Hammitt (2009)	Transfer of USA studies	$1,313,600 (2007)	R 21,000,000
Thesis; Grobler (2016)	Transfer of USA studies	2005 values extrapolated to 2020 at R53 million	R 48,000,000
Harvard; Robinson et al (2018)	Transfer of USA studies	2015 values	R 30,000,000

2.3.3. Pollution abatement options

Abatement options include early decommissioning and the installation of technologies to reduce emissions. Technologies include Flue Gas Desulphurisation (FGD), Electrostatic Precipitators (ESP), Low NOx Burners (LNB) and Fabric Filter Plants (FFP). ESP and FFP are used to reduce particulate matter (PM) emissions, LNB to reduce nitrogen dioxide (NO_2) emissions and FGD to reduce sulphur dioxide (SO_2) emissions. The model required that each abatement technology applied in each plant in each scenario was described in terms of commissioning periods. The model assumed that upon commissioning, each plant's emissions would shift from current emissions to the MES for each of their respective pollutants.

Early decommissioning

Plant lifetimes were described for the 13 power plants modelled, and included commissioning and decommissioning dates, to evaluate pollution emissions per year for each scenario. All scenarios had the same commissioning and decommissioning dates, except for ERP+ED (S4), that specified the early decommissioning of Komati, Hendrina and Grootvlei (Table 5). The decommission dates are based on the present IRP and any decommissioning would be subject to the necessary legal and consultative processes.

Table 5 Commissioning and decommissioning periods for different scenarios

Plant	Commissioning Period		Decommissioning Period			
	All Scenarios		Baseline		S4: ERP +ED	
	Start	End	Start	End	Start	End
Arnot	1971	1975	2021	2029	2021	2029
Camden	2005	2008	2020	2023	2020	2023
Duvha	1980	1984	2030	2034	2030	2034
Grootvlei	2008	2011	2025	2028	**2019**	**2019**
Hendrina	1970	1976	2020	2026	**2019**	**2019**
Kendal	1988	1992	2038	2043	2038	2043
Komati	2009	2013	2024	2028	**2019**	**2019**
Kriel	1976	1979	2026	2029	2026	2029
Kusile	2017	2022	2051	2051	2051	2051
Lethabo	1985	1990	2035	2040	2035	2040
Majuba	1996	2001	2046	2050	2046	2050
Matla	1979	1983	2029	2033	2029	2033
Tutuka	1985	1990	2035	2040	2035	2040

These plant lifetimes were used to describe the base emission rates for each scenario in the integrated model. Plants being decommissioned after 2050, were given a default value of 2051.

PM reduction

Abatement technologies that reduce the emission of PM include Fabric Filter Plants (FFP) and Electrostatic Precipitators (ESP).

An ESP removes particulate matter, from the flue gas using the force of an induced electrostatic charge. ESP upgrades or refurbishments can reduce particulate matter between 95-97%. Eskom estimates that the capital expenditure for ESP upgrades and refurbishments to have overnight costs of between R432 and R537 per kW, based on international benchmarks. It is assumed that these capital costs would be equally distributed throughout the commissioning period. Operational costs for ESPs include increases in auxiliary power, and operations and maintenance costs, which is estimated to be between R10 and R14 per kW per year.

FFPs remove particulate matter by passing flue gas through fabric bags that physically remove the particulate matter. FFPs are much more effective than ESPs and can reduce particulate matter emissions by 99%. Eskom estimates that the capital expenditure for FFP retrofits to have an overnight cost of R1 697 per kW. It is assumed that these capital costs would be equally distributed throughout the commissioning period. Operational costs for FFPs include increases in auxiliary power, and operations and maintenance costs, which is estimated to be approximately R44 per kW per year.

NO_2 reduction

LNBs are an abatement technology to reduce NO_2 emissions. LNBs are designed to control fuel and air mixing to reduce peak flame temperature and thereby reduce NO_2 formation. LNBs can reduce NO_2 emissions by approximately 30%. Eskom estimates that the capital expenditure for LNB retrofits to have an overnight cost of R587 per kW, based on international benchmarks. It is assumed that these capital costs would be equally distributed throughout the commissioning period. LNBs result in increased coal consumption due to an increase in unburnt coal. At R400 per tonne, the annual operational expenditure on coal can range from R 1.7 to R26 per kW per year.

SO_2 reduction

FGD is a set of technologies used to reduce SO_2 emissions. FGD systems typically include a fly ash removal and SO_2 removal. SO_2 (an acid gas) removal is facilitated by alkaline sorbents such as limestone to react with the gas. FGDs are typically separated into two types, semi-dry and wet, dependent on their water requirements, and can reduce SO_2 emissions by 90%. Eskom estimates capital expenditure to range from R 4 211 (semi-dry) to R 5 560 (wet) per kW (overnight costs), based on international benchmarks. It is assumed that these capital costs would be equally distributed throughout the commissioning period. Operational costs include increased auxiliary power requirements, water consumption, reagents and operations and maintenance to range from R 147 (wet) to R 227 (dry) per kW per year.

2.4. Cost-Benefit Analysis

A CBA is a widely used approach employed for decision-making support. This approach was formalized in the United States in 1958 with the purpose of justifying public expenditures on alternative investment options competing public funds such as water, roads, and other public utilities' networks construction projects. CBA methodology broadly advises on the treatment of income benefits and costs; externality costs; how to measure them conceptually; how future prices should be treated; the importance of using a discount rate; the proper period of analysis; and cost allocation procedures for projects.

The World Bank[2] defines a Social CBA as an extension of a financial analysis. Ideally, in extending the financial analysis, all relevant economic costs and benefits are quantified and analysed. The CBA pulls together the component analyses of the study to assess the overall impact for a set of scenario options (emission reduction measures).

The objective of the CBA is to comparatively analyse investments or scenarios (in this case interventions in air quality management). The CBA achieves this end by identifying and monetizing the costs and benefits and predicting the timing thereof over the same horizon as the projects' economic lifetime (National Treasury, 2017).

A CBA allows scenarios to be objectively compared according to the cost:benefit relationship to analyse the relative efficiency of various interventions and the magnitude of the benefits to identify the interventions that will have the largest impacts.

In this analysis, the CBA compares the scenario health benefits to capital and operational costs pf abatement. This CBA does not capture all potential costs and benefits, both direct and indirect. (Refer to section 2.5.1 below for a discussion of CBA limitations.)

The analysis timeline spans 2015 – 2045. This timeframe allows for 5-year interval analysis, aligning to the 2020 MES. It also captures mitigation activities implemented since 2016. The base year was 2018, due to dispersion modelling timeframe. The CBA was performed in an Excel spreadsheet, which consolidated all data sources, which contains all calculations, and was macro-enabled to run the large spatial exposure estimates for each scenario for the review period. Because costs and benefits are accrued in different years according to the intervention schedules, the net present values of costs and benefits, using Eskom's weighted average cost of capital (WACC) rate of 8.4% as the discount rate allows an objective comparison of scenarios.

The health cost benefits were estimated based on the outputs of the AP-HRA, and followed the steps below:

1. Each of the assessed Scenarios implemented an abatement schedule at the 13 power plants assessed (refer to section 3 for details)
2. It was assumed that the abatement schedule achieved MES compliance, and thus the dispersion effects modelled by uMoya-Nilu Consulting was used to estimate the change in population exposure from the "*Current*" to the "*Compliance*" levels.
3. The change in population exposure resulting from step 2 above was applied to the ERFs identified in section 2.3.1 to estimate health impact outcomes (sensitivity analysis was performed in the CBA to develop a view on the uncertainty inherent in the ERFs, also refer to section 2.5.1)

[2] http://documents.worldbank.org/curated/en/445971468767366310/pdf/multi-page.pdf

4. The VSL (refer to section 2.3.2) was applied to the health impact outcomes for each scenario, to estimate change in health cost benefits.
5. Capital and operational cost estimates were used as the costs in the CBA (refer to section 2.3.3).
6. Sensitivity analysis was performed on both the health benefit and abatement cost estimates. We assumed that capital and operations costs estimates is at a concept accuracy or 80%. This implies that costs could vary by +-20%. The health benefits variation was more difficult to judge, due to the extent of uncertainties (refer to section 2.5.1). Comparative analysis of various ERFs in literature shows that health outcome incidences could vary by up to 80%. For this reason, we used a +-80% variation for health benefits in the CBA.

Section 3.2 provides the CBA results.

2.5. Uncertainty of the estimated health effects

2.5.1. Sources of uncertainty and limitations

The WHO (2016) advises performing an assessment of the uncertainty of the analysis; in this case therefore this requires an assessment related to a lack of knowledge about one or more components of the integrated Health CBA Model. The sections below discuss each source of uncertainty and related limitations.

Air pollutants exist as a complex mixture: Despite improvements in the science underlying AP-HRAs, it is still not possible to estimate with complete certainty the effects of air pollution on health (WHO Regional Office for Europe, 2014. The observed adverse effects attributed to an individual air pollutant may actually be (partly) attributable to other pollutants in the mixture which are correlated with the assessed pollutant (WHO Regional Office for Europe, 2013). It is not possible to assess the uncertainty relating to this (WHO, 2016).

Pollutants modelled: The analysis was limited to SO_2, NO_2 and $PM_{2.5}$ pollutants. Other pollutants may also contribute to health risk and these were not modelled in the dispersion modelling. This may under-estimate health risks.

Population pollution exposure level: Dispersion modelling was conducted to estimate exposed population for the selected pollutants. In addition, secondary PM effects were estimated. Dispersion modelling has certainty attached to it as a result of input uncertainty and inherent errors in the model, discussed in Zunckel and Raghunandan (2018). Modelling for estimates of future exposure levels were based on the assumption that after an individual plant has been fitted with abatement technology, the resultant emissions would be equal to MES. This assumption increases the uncertainty of the assessment.

Modelling Domain: The cumulative dispersion modelling domain was 360 km by 270 km. This domain is large but does not offer full coverage of all predicted ambient concentration ranges from power plants. This leads to an underestimation of exposed population. Ideally a dispersion modelling exercise for a study of this nature should have full national coverage.

Baseline disease burden: The number of deaths or cases of disease were estimated by the SAMRC based for either 2012 or 2016, based on Stats SA data. The data for these years is therefore accurate. Uncertainty arises however because projections are made of population size growth in future, under the assumption that the relative ratio of number of deaths in the future remain constant.

Exposure response functions: ERFs are derived from epidemiological studies, in which the parameters of the epidemiological experiment and assumptions made during the experiment introduce some uncertainty into the results. More significantly, because primary epidemiological evidence on air pollution is not available for South Africa. This absence of direct epidemiological evidence is a key limitation. As a result, inference has to be drawn from studies in other parts of the world. It is to be noted that health response per unit change in air pollution high ambient levels (such as the HPA) may differ from that observed in countries with lower pollution levels. In summary, the WHO (2016) notes that extrapolated ERF information may not accurately describe the exposure-response relationship in the region to be assessed, leading to uncertainties in the results.

Resolution of ERFs: The ERFs used were all specified for stepped changes in ambient concentrations of 10µg/m^3. These are relative coarse bands and have to be applied to the changes in annual average ambient concentration estimated by the dispersion modelling, which are in the order of 1-3 µg/m^3.

Morbidity effects were not assessed: The costs of medical treatment (including visiting health care facilities, and costs of medicine and hospitalisation) and the loss of economic production due to sick-leave absenteeism or temporary or permanent disability, were not assessed. As a result, the CBA underestimates the health benefits of the various scenarios.

Value of statistical life: VSLs are accurate when estimated based on primary data collected through willingness to pay studies specific to the exposed population. All VSL estimates for South Africa are derived and transferred from studies done in the United States of America. This introduces uncertainty in the CBA results.

The CBA does not capture economic externalities. These include both benefits and costs. The benefits of reduced health risk on households, employers and the health care and insurance industries were not assessed. The costs of implementation of abatement technologies would put additional pressure on Eskom debt requirements, and further on electricity price escalations. These would result in additional economic costs, and these were not assessed. In Scenario 4, the reduced revenue to Eskom, as a result of earlier plant closure, and its multiplier effects, were not assessed.

The CBA does not capture coal-mining related environmental externalities. In Scenario 4, earlier closure of coal-fired power plants would reduce coal requirements. This scenario may be associated with benefits in the form of a reduction in coal-mining associated environmental externalities.

2.5.2. Dealing with the uncertainties and limitations in the assessment of results

Several important considerations exist when interpreting the results of the integrated Health CBA.

Interpretation of premature mortality or attributable deaths has to be done with care. It is to be noted firstly that these numbers are indicators of health risk at a population level. The relative risk estimate inherent in the ERF is a metric of the likelihood of an adverse health outcome, and it cannot be attributed to an individual person. It can thus be used to quantify risk to a defined population (and not to an individual), (WHO 2016) and how this risk would vary between various policy options of scenarios.

The various sources of uncertainty discussed above, affect the accuracy of the absolute values of the assessments. In the absence of primary ERF studies, it is not possible to judge the accuracy of the absolute values of the assessment with a high level of confidence. However, this report uses ranges to reflect uncertainty.

In spite of the various sources of uncertainty discussed above, the analysis still provides valuable insights into the comparison of scenarios tested in the CBA. This is because the uncertainty inherent in the analysis remain constant across all scenarios.

The description of uncertainty sources also serves as a basis for further work to be prioritised in improving future integrated Health CBAs.

3. RESULTS AND DICUSSION

3.1. Scenarios

The scenarios evaluated in this study (against a baseline) included:

1. Full compliance with new plant standards (FC) (S1)
2. Eskom Emission Reduction Plan (ERP) (S2)
3. ERP + FGD at Kendal (S3)
4. ERP + Early decommissioning (ED) of Komati, Hendrina and Grootvlei (S4)

3.1.1. Scenario 1: Full compliance with new plant standards (FC)

Scenario 1 (FC) assumes that all 13 plants investigated will be in full compliance with new plant standards. FC sees the retrofitting of power plants with LNB (6 plants), FFP (6 plants) and FGD (7 plants), in addition to those already installed. LNB installations begin in 2016 at Camden, and end in 2031 at Lethabo. FFP installations begin in 2017 at Duvha, and end in 2026 at Matla and Tutuka. FGD installations begin in 2019 at Kriel and end in 2038 at Lethabo.

Full compliance with all new plant standards for all stations

S1

	Plant Comissioning Period		Decomissioning Period		Abatement Technologies Installed (1-				Abatement Technology Comissioning Period							
Plant	COD Start	COD End	S1DS	S1DE	ESP	LNB	FFP	FGD	ESP-S	ESP-E	LNB-S	LNB-E	FFP-S	FFP-E	FGD-S	FGD-E
Arnot	1971	1975	2021	2029												
Camden	2005	2008	2020	2023		1					2016	2020				
Duvha	1980	1984	2030	2034			1	1					2017	2022	2024	2029
Grootvlei	2008	2011	2025	2028												
Hendrina	1970	1976	2020	2026												
Kendal	1988	1992	2038	2043			1	1					2019	2025	2028	2033
Komati	2009	2013	2024	2028												
Kriel	1976	1979	2026	2029		1	1	1			2019	2025	2019	2025	2019	2025
Lethabo	1985	1990	2035	2040		1	1	1			2026	2031	2019	2025	2032	2038
Majuba	1996	2001	2046	2050		1		1			2020	2026			2030	2036
Matla	1979	1983	2029	2033		1	1	1			2021	2027	2020	2026	2023	2029
Tutuka	1985	1990	2035	2040		1	1	1			2021	2026	2021	2026	2031	2037
Kusile	2017	2022	2051	2051												

Figure 23 Scenario 1 (FC) power plant commissioning and decommissioning periods, and abatement technology installation schedules. An S-suffix denotes the start of an activity, and the E-suffix denotes the end of the activity. Abatement technologies are assumed to run from the end of their commissioning date to the decommissioning date of the power plant.

3.1.2. Scenario 2: Emission reduction plan (ERP)

Scenario 2 (ERP) assumes that Eskom will continue with its planned emission reduction plan up to the decommissioning of each power plant. ERP sees the retrofitting of power plants with LNB (4 plants), FFP (1 plant), ESP (4 plants) and FGD at none of the 13 plants modelled, in addition to those abatement technologies already installed. ESP installations begin in 2019 at Kendal, Kriel and Lethabo, and end in 2026 at Matla. LNB installations begin in 2016 at Camden, and end in 2027 at Matla. FFP installations begin in 2017 at Duvha, and end in 2026 at Matla and Tutuka. FFP is only installed at Tutuka, beginning in 2021 and ending in 2026.

Planned emission reduction plan up to 50 year life for each station (with only FGD at Medupi)

S2

	Plant Comissioning Period		Decomissioning Period		Abatement Technologies Installed (1-				Abatement Technology Comissioning Period							
Plant	COD Start	COD End	S2DS	S2DE	ESP	LNB	FFP	FGD	ESP-S	ESP-E	LNB-S	LNB-E	FFP-S	FFP-E	FGD-S	FGD-E
Arnot	1971	1975	2021	2029												
Camden	2005	2008	2020	2023		1					2016	2020				
Duvha	1980	1984	2030	2034												
Grootvlei	2008	2011	2025	2028												
Hendrina	1970	1976	2020	2026												
Kendal	1988	1992	2038	2043	1				2019	2025						
Komati	2009	2013	2024	2028												
Kriel	1976	1979	2026	2029	1				2019	2025						
Lethabo	1985	1990	2035	2040	1				2019	2025						
Majuba	1996	2001	2046	2050		1					2020	2026				
Matla	1979	1983	2029	2033	1	1			2020	2026	2021	2027				
Tutuka	1985	1990	2035	2040		1	1				2021	2026	2021	2026		
Kusile	2017	2022	2051	2051												

Figure 24 Scenario 2 (ERP) power plant commissioning and decommissioning periods, and abatement technology installation schedules. An S-suffix denotes the start of an activity, and the E-suffix denotes the end of the activity. Abatement technologies are assumed to run from the end of their commissioning date to the decommissioning date of the power plant.

3.1.3. Scenario 3: ERP + Flue gas desulphurization (ERP+FGD)

Scenario 3 (ERP+FGD) assumes that Eskom will continue with its planned emission reduction plan up to the decommissioning of each power plant, as well as installs FGD at Kendal. ERP+FGD sees the retrofitting of power plants with LNB (4 plants), FFP (1 plant), ESP (4 plants) and FGD (1 plant), in addition to those abatement technologies already installed. ESP installations begin in 2019 at Kendal, Kriel and Lethabo, and end in 2026 at Matla. LNB installations begin in 2016 at Camden, and end in 2027 at Matla. FFP installations begin in 2017 at Duvha, and end in 2026 at Matla and Tutuka. FFP is only installed at Tutuka, beginning in 2021 and ending in 2026. FGD is only installed at Kendal with installation beginning in 2028 and ending in 2033.

Planned emission reduction plan, as above, and FGD at Kendal and Matimba

S3

	Plant Comissioning Period		Decomissioning Period		Abatement Technologies Installed (1-				Abatement Technology Comissioning Period							
Plant	COD Start	COD End	S3DS	S3DE	ESP	LNB	FFP	FGD	ESP-S	ESP-E	LNB-S	LNB-E	FFP-S	FFP-E	FGD-S	FGD-E
Arnot	1971	1975	2021	2029												
Camden	2005	2008	2020	2023		1					2016	2020				
Duvha	1980	1984	2030	2034												
Grootvlei	2008	2011	2025	2028												
Hendrina	1970	1976	2020	2026												
Kendal	1988	1992	2038	2043	1			1	2019	2025					2028	2033
Komati	2009	2013	2024	2028												
Kriel	1976	1979	2026	2029	1				2019	2025						
Lethabo	1985	1990	2035	2040	1				2019	2025						
Majuba	1996	2001	2046	2050		1					2020	2026				
Matla	1979	1983	2029	2033	1	1			2020	2026	2021	2027				
Tutuka	1985	1990	2035	2040		1	1				2021	2026	2021	2026		
Kusile	2017	2022	2051	2051												

Figure 25 Scenario 3 (ERP+FGD) power plant commissioning and decommissioning periods, and abatement technology installation schedules. An S-suffix denotes the start of an activity, and the E-suffix denotes the end of the activity. Abatement technologies are assumed to run from the end of their commissioning date to the decommissioning date of the power plant.

3.1.4. Scenario 4: ERP + Early decommissioning (ERP+ED)

Scenario 4 (ERP+ED) assumes that Eskom will continue with its planned emission reduction plan up to the decommissioning of each power plant, as well initiates early decommissioning at Grootvlei, Hendrina and Komati. ERP+ED sees the retrofitting of power plants with LNB (4 plants), FFP (1 plant), ESP (4 plants), in addition to those abatement technologies already installed. ESP installations begin in 2019 at Kendal, Kriel and Lethabo, and end in 2026 at Matla. LNB installations begin in 2016 at Camden, and end in 2027 at Matla. FFP installations begin in 2017 at Duvha, and end in 2026 at Matla and Tutuka. FFP is only installed at Tutuka, beginning in 2021 and ending in 2026.

Planned emission reduction plan and without Komati/ Hendrina/ Grootvlei from 2018/2019

S4

	Plant Comissioning Period		Decomissioning Period		Abatement Technologies Installed (1-				Abatement Technology Comissioning Period							
Plant	COD Start	COD End	S4DS	S4DE	ESP	LNB	FFP	FGD	ESP-S	ESP-E	LNB-S	LNB-E	FFP-S	FFP-E	FGD-S	FGD-E
Arnot	1971	1975	2021	2029												
Camden	2005	2008	2020	2023		1					2016	2020				
Duvha	1980	1984	2030	2034												
Grootvlei	2008	2011	2019	2019												
Hendrina	1970	1976	2019	2019												
Kendal	1988	1992	2038	2043	1				2019	2025						
Komati	2009	2013	2019	2019												
Kriel	1976	1979	2026	2029	1				2019	2025						
Lethabo	1985	1990	2035	2040	1				2019	2025						
Majuba	1996	2001	2046	2050		1					2020	2026				
Matla	1979	1983	2029	2033	1	1			2020	2026	2021	2027				
Tutuka	1985	1990	2035	2040		1	1				2021	2026	2021	2026		
Kusile	2017	2022	2051	2051												

Figure 26 Scenario 4 (ERP+ED) power plant commissioning and decommissioning periods, and abatement technology installation schedules. An S-suffix denotes the start of an activity, and the E-suffix denotes the end of the activity. Abatement technologies are assumed to run from the end of their commissioning date to the decommissioning date of the power plant.

3.2. Summary

Approximately 20.3 million people are exposed to air pollution from the 13 power plants modelled, that fall within the modelling domain. The mean additional annual average exposure to air pollution of the population within this domain, resulting from coal-fired power station emissions, was estimated by averaging dispersion modelling results over municipal boundaries. Approximately 17.7 million people were exposed to more than an additional 1µg.m^3 (mean annual average) of $PM_{2.5}$. Similarly, 15.3 and 19.0 million people, respectively, were exposed to more than an additional 1µg.m^3 of NO_2 and SO_2.

The health effects of this increased exposure were determined using an AP-HRA, that applied ERFs to the baseline incidence rates, and determined that air pollution from the 13 power plants do have a large health impact. There was extreme variability with the total health costs estimates, which varied by as much as 80%. Furthermore, the total health cost is extremely sensitive to the VSL used, and a conservative value of R48 million was used.

Health benefits associated with each scenario were calculated against the baseline that assumed no new abatement technologies would be installed, and all plants would continue to emit air pollution at their current rates until decommissioning. The scenario with the highest health benefits was ERP+ED (S4), highlighting the immediate results achievable if early decommissioning of power plants can be achieved. The ERP+ED (S4) is estimated to result in health benefits with a NPV that varied between R 3.4 billion and R 30.1 billion. The FC (S1) had the next highest health benefits with a NPV that varied between R 2.5 billion and R 22.1 billion. The ERP+FGD (S3) had marginally higher health benefits than ERP (S2) due to the additional FGD at Kendal. Figure 27 demonstrates the averaged flow of benefits for the four scenarios.

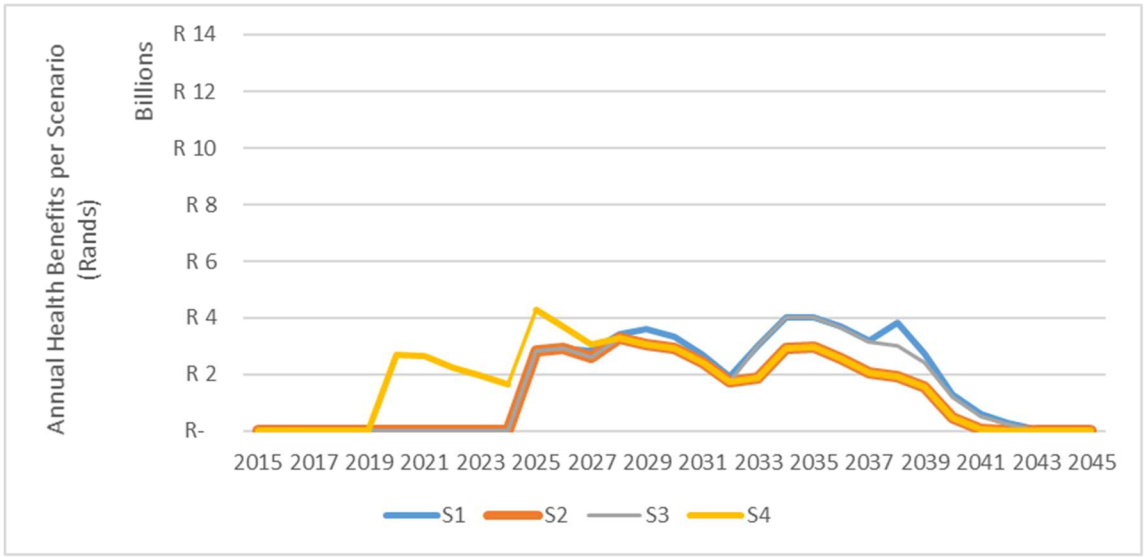

Figure 27 Annual health benefits per scenario

Scenario costs were calculated using Eskom's estimates of abatement technology capital and operational spending requirements. As expected, the FC (S1) had the highest costs due to having the most abatement technologies installed, with the NPV between -R43.4 billion to -R65.1 billion. The ERP (S2) and ERP+ED (S4) had the same costs as they both had the same abatement technology additions with a NPV between -R16.9 billion to -R25.3 billion. The ERP+FGD (S3) had a higher cost with a NPV of

-R21.2billion to -R31.8 billion due to the additional FGD at Kendal. Figure 28 demonstrates the averaged flow of costs for the four scenarios.

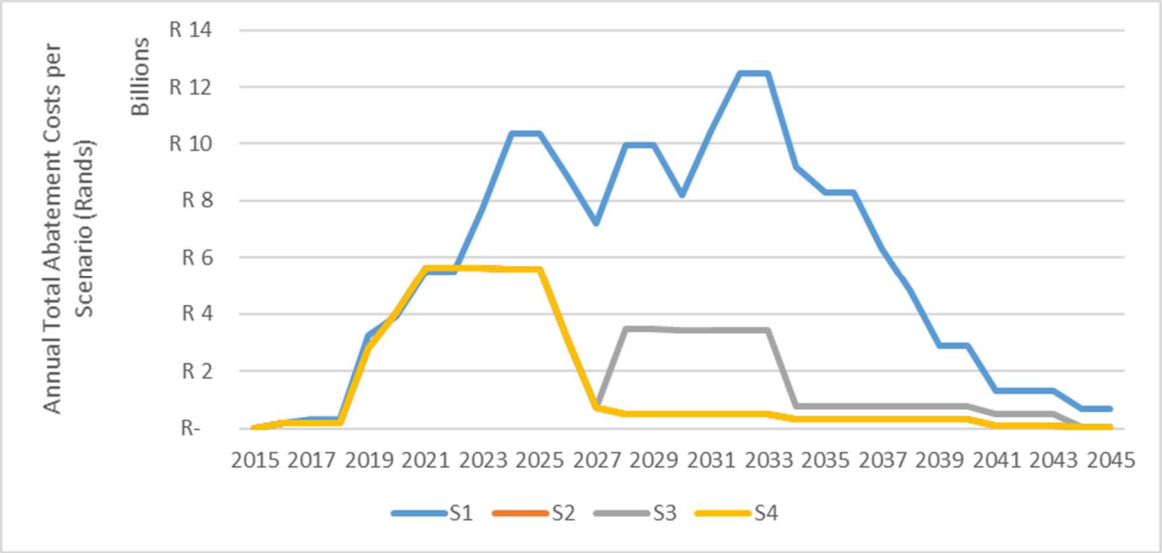

Figure 28 Total abatement costs (CAPEX and OPEX) associated with each scenario's abatement retrofits

Scenarios were compared in a cost-benefit analysis. The cost-benefit analysis apportioned costs (capital and operation expenditure on abatement technologies) and benefits (health benefits) to the years in which they would be realised. Because costs and benefits are accrued in different years according to the intervention schedules, the net present values of costs and benefits, using Eskom's weighted average cost of capital (WACC) rate of 8.4% as the discount rate, and additional sensitivity analysis testing using social discount rates of 1% and -1%, allowing for an objective comparison of scenarios. Dividing the NPV of costs by the NPV of benefits provides a cost:benefit ratio, which when greater than 1 indicates that the costs outweigh the benefits, and when less than 1 indicate that the benefits outweigh the costs.

The CBA ratios need to be interpreted with care. They are meant only to provide a perspective on and inform the decision-making process underlying the scenarios. They are not meant to be interpreted as a definitive answer to making abatement decisions. Decisions involving human health has to be informed by non-economic criteria as well. In addition, uncertainty inherent in the analysis, the cost benefit ratio should thus not be viewed as absolute, but rather as a relative value from which to compare scenarios.

Table 6 Costs and benefits NPV estimates (lower and upper ranges) for each scenario, and cost:benefit ratios using a discount rate of 8.4%.

	FC (S1)		**ERP (S2)**		**ERP+FGD (S3)**		**ERP+ED (S4)**	
Million Rands	*lower*	*upper*	*lower*	*upper*	*lower*	*upper*	*lower*	*upper*
NPV of Costs	-43 369	-65 053	-16 923	-25 385	-21 205	-31 808	-16 923	-25 385
NPV of Benefits	2 403	21 625	1 962	17 661	2 252	20 264	3 374	30 367
NPV of Benefits minus Costs	-40 966	-43 428	-14 961	-7 724	-18 954	-11 544	-13 549	4 982
Cost: Benefit Ratio (*range*)	18.0	3.0	8.6	1.4	9.4	1.6	5.0	0.8
Cost: Benefit Ratio (*central*)	4.5		2.2		2.4		1.3	

Table 7 Costs and benefits NPV estimates (lower and upper ranges) for each scenario, and cost:benefit ratios using a discount rate of 1.0%.

	FC (S1)		ERP (S2)		ERP+FGD (S3)		ERP+ED (S4)	
Million Rands	*lower*	*upper*	*lower*	*upper*	*lower*	*upper*	*lower*	*upper*
NPV of Costs	-119 810	-179,716	-32,713	-49,070	-47,478	-71,216	-32,713	-49,070
NPV of Benefits	8,391	75,517	6,408	57,675	7,819	70,370	8,952	80,565
NPV of Benefits minus Costs	-111,420	-104,198	-26,305	8,606	-39,659	-847	-23,761	-31,495
Cost: Benefit Ratio (*range*)	14.3	2.4	5.1	0.9	6.1	1.0	3.7	0.6
Cost: Benefit Ratio (*central*)	3.6		1.3		1.5		0.9	

Table 8 Costs and benefits NPV estimates (lower and upper ranges) for each scenario, and cost:benefit ratios using a discount rate of 1.0%.

	FC (S1)		ERP (S2)		ERP+FGD (S3)		ERP+ED (S4)	
Million Rands	*lower*	*upper*	*lower*	*upper*	*lower*	*upper*	*lower*	*upper*
NPV of Costs	-164,459	-246,688	-40,048	-60,072	-61,237	-91,856	-40,048	-60,072
NPV of Benefits	12,178	109,599	9,104	81,937	11,325	101,923	12,119	109,069
NPV of Benefits minus Costs	-152,281	-137,090	-30,944	21,865	-49,912	10,067	-27,929	48,997
Cost: Benefit Ratio (*range*)	13.5	2.3	4.4	0.7	5.4	0.9	3.3	0.6
Cost: Benefit Ratio (*central*)	3.4		1.1		1.4		0.8	

In spite of the uncertainties that are inherent in the current assessment process, the assessment provides valuable insights into the effects of air pollution and abatement. The larger investigation has made significant progress on improving the accuracy of dispersion modelling, through modelling secondary PM emissions and through performing cumulative emissions analysis. This work has increased our understanding of the exposed population.

It is recommended that the analysis performed here be continuously improved to address prioritised sources of uncertainty. Improving the accuracy of the ERFs needs priority attention as AP-HRA applications continue to be improved.

In spite of the level of uncertainty associated with ERFs, epidemiological evidence is sufficient to confirm the hypothesis that abatement technologies would have positive impacts on human health.

With the testing of different discount rates, all scenarios become more favourable (i.e. future health benefits received a higher weighting than upfront costs). What is important to note however is that the order of the scenarios, as assessed using cost benefit ratios, do not change.

Thus, most significantly, early decommissioning of the coal-fired power stations assessed in ERP+ED (S4), would have a significantly larger beneficial effect on health costs than abatement technologies alone. This holds for all discount rates tested. This plays a large role in positioning Scenario 4 as the

most beneficial scenario, both in terms of largest health cost benefits, lowest cost of abatement, as well as relative cost:benefit ratio. The FC (S1) would eventually have the second most benefits (see Tables 5, 6 and 7).

It is also noted that the abatement technologies are expensive and would place a significant financial burden on Eskom.

4. REFERENCES

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APPENDIX A: SAMRC REPORT

Concentration-response functions for human health effects of sulphur dioxide, nitrogen dioxide, and particulate matter

Report prepared by CY Wright and MA Oosthuizen

South African Medical Research Council

For

Naledzi Environmental Consultants

2 August 2018

Contents

1. Background

The South African Medical Research Council (SAMRC) Environment and Health Unit was contracted by Naledzi Environmental Consultants to compile a list of relevant concentration-response functions for human health effects of sulphur dioxide (SO_2), nitrogen dioxide (NO_2), and particulate matter (PM). These concentration-response functions will then be used as input in a human health risk assessment in a project with ESKOM.

We have prepared a Microsoft Excel spreadsheet of all of the information mentioned in this report and therefore this report should be read in conjunction with the appended spreadsheet.

2. Introduction

Environmental Health, according to the World Health Organization (WHO) (1992) can be defined as "….those aspects of human health, including quality of life, that are determined by physical, biological, social and psychosocial factors in the environment". Ambient air and specifically the quality of ambient air can therefore be considered as a factor that can determine the quality of an individual's life.

In addition, Target 3.9 of the Sustainable Development Goals, states: "By 2030, substantially reduce the number of deaths and illnesses from hazardous chemicals and air, water and soil pollution and contamination" and one of the indicators (3.9.1) for this target is "mortality due to air pollution (ambient and household) (WHO, 2016).

As far back as 1285, an air pollution commission in London recommended the banning of coal burning in urban areas to reduce air pollution (Smith and Akhar, 2003). In December 1952, severe air pollution in the form of a layer of smoke from coal burning (formed due to unfavourable meteorological conditions) caused a high mortality rate in London. This high mortality rate continued for a number of months after the air pollution episode. Evidence shows that what was previously thought to be mortalities due to influenza during and after the episode, could not be possible, which leaves 12 000 unexplained and

additional deaths (Brunekreef and Holgate, 2002; Davis et al, 2002). As recent as 2013, an air pollution episode in China was responsible for daily $PM_{2.5}$ (particulate matter with a diameter of equal to or less than 2.5 μm) concentrations that exceeded 500 $\mu g/m^3$ (West, 2016).

Since the London episode of 1952, results of numerous studies have established associations between air pollutants and acute and chronic human health effects, including cardiovascular effects, cerebrovascular effects, respiratory effects and cancer (Bowe et al., 2018, Maji et al., 2017, WHO, 2013, Wichmann and Voyi 2012). Associations with kidney effects and diabetes II have also been observed (Bowe et al., 2018, Pope III et al., 2015, Pearson et al, 2010).

In 2013, the International Agency for Research on Cancer (IARC) classified outdoor air pollution as a human carcinogen and associated with lung cancer. An association between outdoor air pollution and an increased risk for urinary tract/bladder cancer has also been observed (WHO, 2018).

According to estimations by the WHO, 58% of outdoor air pollution-related premature deaths in 2016 were due to ischaemic heart disease and strokes, while 18% were due to chronic obstructive pulmonary disease, another 18% due to acute lower respiratory infections and 6% of deaths were due to lung cancer (WHO, 2018).

Although the physiological mechanisms of action are not clear in all of the health effects associated with exposure to air pollution, biologically plausible mechanisms have been reported. These mechanisms include biomarkers of systemic inflammation, such as C-reactive protein and fibrinogen (Kelly and Fussel, 2015).

Despite a decrease in air pollution in some countries, such as countries in Europe and North America (West et al., 2016) due to legislation and engineering controls, air pollution is still a global issue. Ambient $PM_{2.5}$ was the fifth-ranking mortality risk factor in 2015 and was associated with more than 4 million deaths and 103.1 million disability-adjusted life-years (DALYs) globally (Cohen et al, 2017). In the same year (2015), household air pollution was responsible for 2.9 million deaths and 85.6 million DALYs (GBoD of 2015, 2016).

A concern is that air pollution in sub-Saharan Africa is on the increase, supposedly due to an increase in population, motor vehicles and domestic fuel use (Amegah and Agyei-Mensah, 2017). Air pollution is also on the increase in the Middle East, Asia and Latin America (West et al., 2016).

It must be kept in mind that air pollution has no boundaries and thus is not confined to the country where the sources are located. Of further concern is that research from epidemiological and toxicological studies has indicated that 'current' air pollution is even more detrimental to health, arguably due to the different components of air pollution (Hoek et al., 2013, Kelly and Fussel, 2015, Thurston et al., 2016, Wang et al, 2017). These different components are as a result of different sources and different technologies now in use, which in some developing countries, goes together with poor control of air pollution (West, et al., 2016).

As a result of the changing nature of air pollution, a current important research question is: "what specific characteristics and components of air pollution or specific mixtures of air pollutants are responsible for specific health effects?" (West et al., 2016).

3. Exposure-response functions

Exposure–response functions or concentration-response functions of air pollutants are used to determine the burden of disease from air pollution and therefore the impact air pollution may have on human health, which in turn, may be used to inform policy (WHO, 2014).

Accurate exposure of populations to air pollutants is of the utmost importance to develop these exposure-response functions and the policies they inform (Shaddick et al., 2018, WHO, 2014). Initial exposure-response functions were based on monitored data, however, not all countries have extensive monitoring networks. To overcome this problem, additional measures of air quality are being introduced lately, for example:

- Land-Use Regression models (looking at road type, traffic counts, topography, and land cover) (Fischer et al., 2015),
- Satellite remote sensing (of aerosol optical depth), and

- Chemical transport models which are then calibrated against ground measurements using linear regression to produce air quality data at high resolutions (Shaddick et al., 2018). Ideal resolution would be ~1 km, but ~12 km is acceptable (WHO, 2014).

Studies used to develop concentration-response functions are mainly epidemiology studies and they tend to focus on mortality (death) rather than morbidity (illnesses or conditions). The reason for this is often the lack of baseline data for morbidity, such as the number of hospital and clinic visits and the prevalence of illnesses, for example, asthma (WHO, 2014). This is also true for South Africa where national mortality data are more readily available than morbidity data (StatsSA, 2018).

There are uncertainties in concentration-response functions. For example, relating to the **shape of the concentration-response curve** (linear or threshold) at different concentrations, also the risk of **exposure to air pollution mixtures** (whether the risk will be: the same as the sum of the effects of the single pollutants evaluated (additive) or more (synergistic) or less (antagonistic)), and **how concentration-response relationships may differ from one population to another**.

The question is thus whether the concentration-response function can be extrapolated from one population to another if one population has a different lifestyle, age structure and medical service than the other, for example from a developed to a developing country? (WHO, 2014).

Another uncertainty is the health impacts of **specific sources** (WHO, 2014). In determining concentration-response functions by using mass per volume, for example, for PM, it is generally assumed that all particulates are equally toxic, although studies suggest a difference in health impacts of particulate matter from different sources or areas, most probably due to a difference in size and chemical composition (Hoek et al., 2013, Lelieveld et al., 2015, Wang et al., 2017).

3.1 Shape of exposure-response curve

The shape of the exposure-response graph may be different for different outcomes (diseases) as determined by Pope III et al. (2011). They found the concentration-response function for $PM_{2.5}$ and lung cancer to be “nearly linear”, whereas for cardio-vascular disease it was not the case; the response was much steeper at the very low levels of exposure compared with higher levels of exposure. One explanation

for the different shapes of the concentration-response graphs is that the cardiovascular effects may have been caused by the particulates themselves, whereas the carcinogenic compounds adsorbed onto the particulates, may be responsible for lung cancer (Pope III, 2011). The average daily ambient $PM_{2.5}$ concentrations were approximately 14 μg/m^3 (Pope III, 2011).

The team assessing the Global Burden of Disease (GBD) in 2010, incorporated information about exposure and the health risks posed by ambient and indoor particulate matter from studies done all over the world, into an integrated exposure model, in order to estimate the relative risk of dying from different diseases in relation to different concentrations of fine particulate matter ($PM_{2.5}$) (Arnold, 2014). Previous models used a linear exposure-response relationship between 7.5 and 50 μg/m^3 (the latter concentration to cater for smokers) based on the American Cancer society Cancer Prevention Study II (CPSII) where the exposure concentration of $PM_{2.5}$ was less than 22 μg/m^3 (Burnett et al., 2014). There was thus a gap in the knowledge of what the exposure–response curve would look like when average concentrations of $PM_{2.5}$ would be above 50 μg/m^3 (Lim et al., 2012) indicating the importance of health impact assessments in developing countries, where a range in annual concentration of $PM_{2.5}$ of 50-100 μg/m^3 may be possible (WHO, 2014). In their analysis of data from the 2015 Global Burden of Diseases Study, Cohen et al. (2017) found no risk (no response) in association with $PM_{2.5}$ concentrations below 2.4 μg/m³.

Yin et al., (2017) determined the shape of the concentration-response graphs in a study in China, where exposure was to higher concentrations (4.2 to 83.8 μg/m^3) of $PM_{2.5}$ than in the United States studies. However, they confirmed the non-linear shape (threshold or S-curve) for cardiovascular disease mortality that Pope III et al., (2011) described in their study, but for lung cancer they also found a threshold and thus did not confirm the linear shape of Pope III's study. These findings indicate that the concentrations of exposure (high or low) could have an influence on the shape of the concentration-response graphs for mortality due to $PM_{2.5.}$

There is a lack of evidence regarding the shape of $PM_{2.5}$ concentration-response graphs in terms of morbidity (disease) both for low and high concentrations of exposure (WHO, 2014). Studies of ambient PM pollution suggest a smaller effect on the incidence of cardiovascular and respiratory disease than on mortality (Lim et al, 2012).

3.2 Exposure to air pollution mixtures

As mentioned earlier in Section 2, the WHO (2014) considers the exposure-response functions of pollution mixtures as an area of work where there is still uncertainty. There is uncertainty whether the risk will be additive, synergistic or antagonistic when pollutant mixtures are evaluated for their association with diseases compared to when single pollutants are evaluated. In this regard a population cohort study involving about 2 million adults in Canada investigated the association between $PM_{2.5,}$ NO_2 and ozone (O_3) and the incidence of dementia. The results showed a hazard ratio of 1.04 for $PM_{2.5,}$ 1.10 for NO_2 and 0.98 for O_3 when evaluated as single pollutants and a hazard ratio of 1.02 for $PM_{2.5,}$ 1.09 for NO_2 and 0.99 for O_3 when evaluated in a three-pollutant model, thus a slight decrease in risk (Chen et al., 2017). Results may, however, differ in a different setting or with different pollutants.

3.3 Concentration-response relationships in different populations and from different sources.

Different sources of air pollution, such as coal combustion, metal production, traffic and biomass burning, have been associated with different types of health effects (Kelly and Fussel, 2015). Different populations may be exposed to different compositions of particulates depending on the sources, for example in London people are mostly exposed to diesel particulates, while in Ethiopia the main source of particulates is biomass burning, in Ghana it is the desert's wind-blown sand as well as traffic and in some parts of China, particulates are mostly from coal-fired power stations (Kelly and Fussel, 2015). Particulates from different sources may differ in chemical composition, size, shape and surface area, all of which are characteristics that may influence their toxicity. In addition, volatile organic compounds adsorbed to particulates may influence toxicity (Kelly and Fussel, 2015).

Requia et al. (2018) performed a meta-analysis on 70 studies from 28 countries on air pollution and human health related to cardiovascular and respiratory effects. In their analysis, they observed significant heterogeneity which could be as a result of differences in ambient air pollution, population characteristics and environmental exposure.

Requia et al. (2018) also investigated the effect that variables related to energy (clean or dirty electricity production), transportation (consumption of bio-fuel, distillate fuel oil and motor gasoline) socioeconomic

status (population density, Gross Domestic Product (GDP) and education) and a variable for smoking or using solid fuel for cooking, could have on the association between air pollutants and cardio-respiratory effects. These effects included both cardio-respiratory morbidity (hospital admissions) and cardio-respiratory mortality. An increased risk of cardio-respiratory effects was demonstrated for exposure to all the pollutants considered the current report (PM_{10}, $PM_{2.5}$, SO_2 and NO_2). The effect of air pollution on cardio-respiratory effects varied spatially across the 28 countries assessed. The highest risks identified were for Brazil (Requia et al., 2018).

Results from this meta-analysis showed that for hospital admissions related to $PM_{2.5}$ exposure, clean electricity production, consumption of biofuels and urban population accounted for about 69% of the heterogeneity, while for mortality related to $PM_{2.5}$ exposure, clean electricity production, consumption of motor gasoline, consumption of cooking fuel, population density and education, accounted for 64% of the heterogeneity (Requia et al., 2018).

Lelieveld et al. (2015) determined global pre-mature deaths related to air quality from different sources in different countries. They found $PM_{2.5}$ was associated with most pre-mature deaths and mainly in Asia. In addition, they found emissions from household fuel use (for heating and cooking) had the largest impact globally.

4 Particulate matter (PM)

Particulate matter is defined as “a complex mixture of solid and liquid particulates of organic and inorganic substances suspended in the air” (WHO, 2018). Smaller particulates have a higher potential to enter deep into the lungs and are therefore considered to be a risk to human health (WHO, 2018).

As far back as 1935 PM was already implicated in respiratory health effects. An article was published in “Public Health Reports” (Brown et al., 1935) about a dust storm in Kansas in the United States of America, that caused instant darkness for 40 minutes and for three hours light only occasionally broke through. Apart from the livestock that was lost and crops that were destroyed, there was a marked increase in respiratory infections and hospital admissions for infections as well as deaths. Analysis of micro-organisms in samples from the dust storms did not identify pathogens and it was therefore concluded that the

irritation caused by exposure to PM (which had a relatively high silica content) contributed to susceptibility to infections.

4.1 Particulate matter with a diameter equal to or smaller than 2.5 µm ($PM_{2.5}$)

As mentioned in the introduction, globally ambient $PM_{2.5}$ was the fifth-ranking mortality risk factor in 2015 (Cohen et al, 2017). According to the Global Burden of Disease study of 2015 (published in 2016), ambient PM ranked 7th among the 10 largest contributors to death and disability in South Africa, while household air pollution was not amongst the ten largest contributors to death and disability in South Africa.

Many studies have found significant associations between $PM_{2.5}$ and many adverse health effects, including an increased risk of death, cardiovascular disease, stroke and even diabetes (Pope III et al., 2015, WHO 2013, Hoek et al., 2013, Pearson et al., 2010). A population cohort study involving about 2 million adults in Canada (where air pollution is amongst the lowest in the world) even showed a risk of dementia from exposure to $PM_{2.5,}$ although the five-year cumulative exposure was only 10.4 µg/m^3 (Chen et al., 2017).

The risk for cardiovascular mortality stimulated by air pollution, is increased, not only in individuals with cardio-metabolic disorders (such as insulin resistance/high blood glucose/diabetes mellitus, hypertension, obesity (especially central obesity), and elevated levels of lipids in the blood) but also in those without such disorders (Pope III et al., 2015). Deaths from hypertension or diabetes were more strongly associated with exposure to $PM_{2.5}$ compared to other disorders (Pope III et al., 2015). These findings were based on a 22-year prospective cohort study with improved exposure assessment (residential level) involving 669 046 participants from the American Cancer Society Cancer Prevention Cohort II Study (Pope III et al., 2015). It is believed that $PM_{2.5}$ is the biggest driver for pre-mature mortality in the world (Requia et al., 2018).

Studies on the association between $PM_{2.5}$ and effects on the kidney are few but a recent cohort study, involving more than 2 million people, found that a 10 µg/m^3 increase in the concentration of $PM_{2.5}$ was associated with an increased risk of having a low estimated glomerular filtration rate (eGFR) (hazard ratio 1.21) (which is an indication that the kidneys are not functioning optimally), chronic kidney disease

(hazard ratio 1.27) and end stage renal disease (chronic kidney failure) (hazard ratio 1.26) (Bowe et al., 2018).

According to Pope III et al. (2018) the indication from epidemiological studies is that the impact of $PM_{2.5}$ on the burden of disease is mainly due to cardiovascular morbidity and mortality. Long-term prospective cohort studies in the United States showed a Hazard Ratio of 1.34 for cardiovascular mortality for an increase of 10 µg/m^3 in the concentration of $PM_{2.5,}$ even after control for risk factors such as smoking.
A study by Thurston et al. (2016) involving more than 4 million adults in more than 100 metropolitan areas of the United States indicated that the risk of ischaemic heart disease mortality associated with $PM_{2.5}$ differ according to compounds and source. The risk was five times higher for $PM_{2.5}$ from coal combustion, than for the same mass in general ambient air. Similarly, diesel traffic-related elemental carbon soot produced a Hazard Ratio of 1.03, but $PM_{2.5}$ from wind-blown soil and biomass combustion was not associated with a risk of ischaemic heart disease mortality (Thurston et al., 2016).

4.2 Particulate matter with a diameter equal to or smaller than 10 µm (PM_{10})

We tried to locate studies in developing countries that may be comparable to the South African situation. A study in India by Maji et al. (2017) showed an increased risk of hospital admissions for respiratory and cardiovascular diseases per 10 µg/m^3 increase in exposure to PM_{10}. The same study in India (Maji et al 2017), as well as studies in South Korea (Kim et al, 2017), Hong Kong and Taiwan (Lu et al., 2015) and China (Shang et al., 2013) showed associations between PM_{10} exposure and respiratory and cardiovascular mortality.

Meta-analyses of 10 European cohort studies, involving about 16 000 children, indicated an increased odds of pneumonia when exposed to PM_{10} (Macintyre et al., 2014).

The Dutch Environmental Longitudinal Study (DUELS) was a study that involved about 7 million individuals above the age of 30 years, followed as a cohort for seven years, and reported associations between PM_{10} and non-accidental mortality, circulatory disease mortality, respiratory mortality and lung cancer mortality. The association they found between NO_2 and respiratory mortality disappeared when adjusted for PM_{10} but it remained for $PM_{10,}$ and the association with lung cancer mortality and NO_2 was reduced from a hazard ratio of 1.093 to 1.08 when adjusted for PM_{10}.

5 Nitrogen dioxide (NO_2)

The main man-made sources of NO_2 are combustion processes (i.e. heating, power generation, and engines in vehicles and ships) (WHO, 2018). Most atmospheric NO2 is emitted as NO, which is rapidly oxidized by ozone to NO_2 (WHO, 2006).

As far as short-term effects are concerned, the lowest observed adverse effect level on lung function in laboratory studies involving asthmatics, was a one hour concentration above 500 µg/m^3.

Epidemiological studies have shown a reduction in lung function growth associated with NO_2 (WHO, 2018). Hoek et al. (2013) performed meta-analyses on 14 cohort studies and found a Relative Risk (RR) of 1.05 for all-(natural) cause mortality for a 10 µg/m^3 increase in NO_2.

In the Dutch Environmental Longitudinal Study (DUELS), Fisher et al. (2015) found an association between NO_2 and a number of mortalities (non-accidental, respiratory and lung cancer) but not for circulatory disease and when they adjusted the association between the different mortalities and NO_2, for PM_{10}, the association of NO_2 with respiratory mortality disappeared (but it remained for PM_{10}) and the association with lung cancer mortality was reduced from a hazard ratio of 1.093 to 1.08. However, other studies did find an association between NO_2 and cardiovascular effects (see below).

In a South African case-cross-over study by Wichmann and Voyi (2012) it was found that cardiovascular mortality increased by 2.6% and cerebrovascular mortality by 6.6% with an increase of 10 µg/m^3 in the daily average of NO_2.

A population cohort study involving about 2 million adults in Canada (where air pollution is amongst the lowest in the world - 16.2 ppb) showed a risk of dementia from exposure to NO_2 (Chen et al., 2017). Another long-term (16 years) Canadian cohort study, involving 2.5 million people, showed associations between NO_2 exposure and mortality from: respiratory diseases, cardiovascular and cerebrovascular diseases, as well as diabetes and lung cancer (Crouse et al., 2015).

A study in India by Maji et al (2017) showed an increased risk of hospital admissions for respiratory and cardiovascular diseases per 10 µg/m^3 increase in NO_2 exposure. The same study in India (Maji et al 2017), as well as a study in China (Shang et al., 2013) showed associations between NO_2 exposure and respiratory and cardiovascular mortality.

Meta-analyses of 10 European cohort studies, involving about 16 000 children, indicated an increased odds of pneumonia and otitis media (middle ear infection) when exposed to NO_2 (Macintyre et al., 2014). Studies on co-exposure of NO_2 and SO_2 have found synergistic (worse than additive) effects on human health (WHO, 1997).

6 Sulphur dioxide (SO_2)

The main man-made sources of SO_2 are the burning of sulphur-containing fossil fuels, for example, coal-fired power stations, domestic coal burning for heating and motor vehicles (WHO, 2018).

Exposure to SO_2 may cause upper respiratory irritation, inflammation of the respiratory tract, aggravation of asthma and chronic bronchitis and have a detrimental effect on lung function. In addition, it was found that hospital admissions and cardiac disease increase on days when SO_2 levels are higher (WHO, 2018).

Exercising asthmatics who participated in controlled studies experienced changes in pulmonary function and respiratory symptoms after 10 minutes of exposure to SO_2 which formed the basis of the recommended 10 minute SO_2 guideline of 500 µg/m^3 (WHO, 2006). However, subsequent re-analysis of the effects of SO_2 post-2005 found evidence that the point of departure for setting the 10-minute guideline of 500 µg/m^3 needs an additional uncertainty factor, which indicates that the guideline may have to be lowered when it is re-evaluated (WHO, 2013a). The WHO is of the opinion that an annual guideline is not needed, since compliance with the 24-hour level will assure low annual average levels (WHO, 2006).

In a South African case-cross-over study by Wichmann and Voyi (2012) it was found that cardiovascular mortality increased by 3.3% and cerebrovascular mortality by 5.3% with an increase of 10 µg/m^3 in the daily average of SO_2.

A study in India by Maji et al. (2017) showed an increased risk of hospital admissions for respiratory and cardiovascular diseases per 10 µg/m^3 increase in SO_2 exposure. The same study in India (Maji et al 2017) as well as a study in China (Shang et al., 2013) showed associations between SO_2 exposure and respiratory and cardiovascular mortality.

Studies on co-exposure of SO_2 and NO_2 have found synergistic (worse than additive) effects on health (WHO, 1997).

7 Choice of concentration-response functions

The concentration-response functions per 10 µg/m^3 increase in the concentration of the specific pollutants, are presented in the attached spreadsheet and some recommended concentration-response functions are tabled in Appendix A. In compiling these functions to be used in the current study, the focus was on recent studies, large study populations, and long-term studies, meta-analyses of large studies, prospective cohort studies and studies from developing and developed countries, higher and lower concentrations of pollutants.

For long-term effects of $PM_{2.5}$, the following concentration-response functions are recommended:

For ***all-cause mortality*** – 1.06, and ***all natural cause*** mortality 1.066, as recommended by the WHO (2013 and 2014 respectively) from annual mean concentrations.

Similar concentration-response functions for all-cause mortality were found by Pope III et al. (2015) namely, 1.07, in the 22 year follow-up of the American Cancer Society study and 1.06 resulted from two long-term cohort studies published in 2018 by Pope III et al.

For ***cardiovascular mortality*** from long-term exposure, the concentration-response function of 1.12 from the American Cancer Society study (Pope III et al., 2015) is recommended.

This concentration-response function is in the same order as the 1.10 recommended by the WHO (2014) and the 1.09 found in the Chinese men prospective cohort (Yin et al., 2017), where exposure was to higher concentrations (range 4.2 to 83:8 µg/m^3).

For ***respiratory mortality*** from long-term exposure, the concentration-response function of 1.10 from studies in Europe, Canada and the US (WHO, 2014) is recommended. Crouse et al., (2015) found no increased risk per 5 µg/m^3 in the 16 year Canadian Census cohort study

For ***lung cancer mortality*** the exposure-response function of 1.09 determined by Burnett et al. (2014) is recommended.

Burnett et al. applied an Integrated Exposure model to data from the Global Burden of Disease 2010 study. This concentration-response function is the same as that determined by Hamra et al. (2014) in meta-analyses of 18 studies on particulate matter and lung cancer in the USA, Europe, China, Japan and New Zealand. The concentration-response function from the Chinese men study (Yin et al., 2017) was slightly higher (1.12). It must be noted that this study recorded higher exposure concentrations (range 4.2 to 83:8 µg/m^3).

For ***ischaemic heart disease mortality*** the concentration-response function by Burnett (2014) of 1.05 is recommended.

This function is in the same order as that found by Yin et al (2017) (1.09) in the Chinese men study. The function resulted from the American Cancer Society follow-up analyses is 1.14 (Pope III, 2015).

Thurston et al., 2016 found different concentration-response functions for the association with long-term exposure to $PM_{2.5}$ and ischaemic heart disease, depending on the type of particulates. For general ambient particulates the hazard ratio was 1.01 per 1 µg/m^3 increase. For $PM_{2.5}$ from coal combustion, which is relevant to this project, it was 1.05 per 1 µg/m^3 increase. These risks were based on data from the American Cancer Society II study.

<u>For short-term effects of $PM_{2.5}$,</u> the following concentration-response functions are recommended:

For ***daily all-cause mortality*** – two concentration-response functions are recommended for consideration. The first is 1.012, as recommended by the WHO (2013) from daily mean concentrations (14 µg/m^3). This concentration-response function, however, differs from the 0.40% increase in daily mortality

found in a meta-analyses of 59 epidemiology studies in 22 cities in mainland China, Hong Kong and Taiwan (Lu et al., 2015), where the exposure was to higher concentrations (39 to 177 μg/m^3). It is therefore possible that the difference could be due to exposure to different concentrations and we suggest using both.

For ***hospital admissions for cardiovascular diseases***, including stroke, based on daily mean concentrations, 1.0091 is recommended as per the WHO (2013) from the APED study.

For ***respiratory mortality*** from short-term exposure, the percentage increase was 0.75% (Lu et al., 2015), thus from exposure to relatively high concentrations.

For ***ischaemic heart disease mortality*** from short-term exposure (to approximately 14 μg/m^3), Pope et al., 2011, published a concentration-response factor of 1.18 based on data from the American Cancer Society II study.

For other health outcomes from short-term and long-term exposure to $PM_{2.5}$, including kidney failure, diabetes mortality and risk of dementia, concentration-response functions are presented in the attached spreadsheet.

<u>For long-term effects of PM_{10}</u>

For ***total mortality*** the following concentration –response functions are recommended for consideration:

The largest study investigating the association between PM_{10} and total mortality, was the Dutch Environmental Longitudinal study (DUELS) (involving about 7 million people), published by Fischer et al., (2015). They found a concentration-response function of 1.08 for an increase of 10 μg/m^3. The median exposure concentration was relatively low at 29 μg/m^3.

This exposure-response concentration was similar to the 1.05 published by Kim et al (2017) from a study in Korea involving about 300 000 individuals, where the mean exposure was 60 μg/m^3 (range between 39 and 72 μg/m^3). However, another study in India involving 1.69 million people exposed to concentrations

between 150 and 210 µg/m^3 found a concentration-response function of 1.0044, which is much lower (Maji et al., 2017).

When selecting a concentration-response function from the above, the concentration to which the specific community is exposed, should be taken into account.

For ***cardiovascular disease mortality*** the same situation was evident as for total mortality, namely the largest study investigating the association between PM_{10} and cardiovascular mortality, was the Dutch study (Fischer et al., 2015). They found a concentration-response function of 1.06 which was similar to the 1.05 of the Korean study (Kim et al., 2017), but higher than the Indian study's concentration-response function of 1.006, despite higher exposure concentrations in India (Maji et al., 2017).

For ***cerebrovascular mortality*** from long-term exposure to $PM_{10,}$ the concentration-response function of 1.14 from the Korean study (Kim et al., 2017) is recommended.

For ***respiratory mortality*** the concentration-response function of the Dutch study was found to be 1.13 (Fischer et al., 2015), which was in the same order as that of the Korean study (1.19) (Kim et al., 2017), with the Indian study again lower at 1.008 (Maji et al., 2017).

<u>For short-term effects of PM_{10}</u>

For ***total mortality*** a concentration-response function of 0.36 % increase for an increase of 10 µg/m^3 is recommended.

This function was determined by Lu et al. (2015) from meta-analyses of 59 epidemiology studies in 22 cities in China where the exposure concentrations ranged between 52 and 174 µg/m^3. This function is similar to the 0.32 excess risk found in another meta-analyses of 33 studies (also in China) published by Shang et al. (2013).

For ***cardiovascular mortality*** the concentration-response functions of the two Chinese studies were again similar showing increases of 0.36% (Lu et al., 2015) and 0.43% (Shang et al., 2013). However, in this case

data for South Africa is available from a study by Wichmann and Voyi (2012) that showed a 1.7% increase in daily cardiovascular mortality for an increase of 10 µg/m^3 in the daily concentration of PM_{10}.

For ***cerebrovascular mortality*** South African data are also available from the study by Wichmann and Voyi (2012), which showed an increase of 3.2% for an increase of 10 µg/m^3 in the daily concentration of PM_{10}.

For ***respiratory mortality*** the concentration-response functions of the two Chinese studies are in the same order of magnitude. The larger Chinese study showed a 0.42% increase in respiratory mortality (Lu et al., 2015) and the South African study (Wichmann and Voyi, 2012) a 1.1% increase for an increase of 10 µg/m^3 in the daily concentration of PM_{10}.

For other less frequently measured health outcomes from short-term and long-term exposure to PM_{10}, including pneumonia, otitis media (ear infection) and croup as well as incidence of asthma in asthmatic children and hospital admissions for respiratory and cardiovascular disease, concentration-response functions are presented in the attached spreadsheet.

<u>For short-term effects of SO_2</u>

Since the health effects of SO_2 are normally acute, more studies determining concentration-response functions focused on short-term exposure than long-term exposure.

For ***respiratory mortality***, the South African study (Wichmann and Voyi, 2012) did not find an increased risk, but the study by Shang (2013) (meta-analyses of 33 Chinese studies) reported excess risk of 1.18.

For ***cardiovascular mortality*** the South African study reported an increase of 3.3% per 10 µg/m^3 increase in the daily concentration, while the Chinese study reported an excess risk of 0.85 (Shang et al., 2013).

Only the Chinese study (Shang et al., 2013) reported on ***all-cause mortality*** (excess risk of 0.81), and only the South African study (Wichmann and Voyi, 2012) reported on ***cerebrovascular mortality*** (an increase of 5.3%).

Long-term effects of SO_2

For long-term effects of SO_2, only one study, the study by Maji et al (2017), was found in the literature surveyed. For ***total mortality***, the RR was 1.0068, for ***respiratory mortality***, 1.0106 and for ***cardiovascular mortality*** the RR was 1.0103.

For ***hospital admissions***, the RR for chronic obstructive pulmonary disease (COPD) was 1.007, for respiratory conditions 1.0014 and for cardiovascular conditions 1.0079.

For long-term effects of NO_2,

For ***all-cause mortality***, the concentration-response function of 1.03 is recommended, resulted from three large studies, namely the Californian study (Jerret et al 2013), the Dutch study (Fisher et al 2015) and the Indian study (Maji et al., 2017).

For ***cardiovascular disease mortality***, the results from these three studies differed slightly. Jerret et al. (2013) reported a RR of 1.048, Maji et al. (2017) a RR of 1.0206, while Fischer et al. (2015) found no risk.

The study by Maji et al. (2017) was the most recent, involved 1.69 million people and was in a country where the concentrations are relatively high. It was therefore decided to use the concentration –response function of 1.0206 from the Indian study.

For the same reason it was decided to use the concentration-response function of 1.0371 from the same study for ***respiratory mortality*** over the one of 1.02 from the Dutch study (Fischer et al., 2015).

For short-term effects of NO_2

For ***all-cause mortality*** there are two concentration-response functions that can be used. One from Europe (WHO, 2013) (RR 1.0027) and another from China (Shang et al., 2013) (excess risk 1.3).

For ***respiratory mortality***, the concentration-response function of the South African study by Wichmann and Voyi (2012) is recommended (percentage increase of 1.7). The function resulted from the Chinese study (Shang et al., 2013) showed an excess risk of 1.62.

For ***cardiovascular mortality***, the concentration-response function of the South African study by Wichmann and Voyi (2012) is again recommended (percentage increase of 2.6), while the function resulted from the Chinese study (Shang et al., 2013) showed an excess risk of 1.42.

For other less frequently measured health outcomes from short-term and long-term exposure to NO_2, including pneumonia, otitis media (ear infection) and croup as well as hospital admissions for respiratory and cardiovascular disease, concentration-response functions are presented in the attached spreadsheet.

8 Conclusions

It is evident that air pollution is an environmental risk factor for a number of health outcomes. These outcomes are not confined to respiratory effects only but include vascular diseases, diabetes, kidney effects and even dementia.

Of concern is the fact that air pollution is on the increase in mainly developing countries and since air pollution has no boundaries, the impact will not be confined to the country where the sources are. Also of concern is that currently air pollution is considered more toxic than before as a result of different technologies and products being used and produced.

It is therefore important to determine the impact air pollution may have on the population of a country, also in economic terms, as this will inform policies and the setting of air quality standards. To determine these impacts, concentration-response functions, determined mostly through epidemiological studies, are being used. It is important for these concentration-response functions to be valid and they can only be valid if the exposure in the epidemiology studies used to determine them, was accurately measured. Not all areas in all countries where the epidemiology studies were conducted have monitoring networks at a high resolution, and it is therefore necessary to use additional tools to measure exposure, such as Land-Use Regression models, satellite remote sensing, and chemical transport models. These methods

allow for air pollution concentrations to be determined at the level of the residential address of participants.

Since ambient air pollution measurements alone may underestimate personal exposure, integrated exposure was determined in some studies, where exposure to smoking (active and passive) and household fuel use were also taken into account. Despite these measurements to accurately determine concentration-response functions, there are still uncertainties, for example, whether the shape of the concentration-response graphs can be considered linear for all pollutants or whether concentration-response functions developed from studies conducted in developed countries, where air pollution is relatively low (and where most studies have been done), can be used in developing countries where air pollution is relatively high and where there is a lack of information on concentration-response functions? Another uncertainty is the effects that pollution mixtures may have on human health, since epidemiology studies are focused on individual pollutants. However, air pollution mixtures are complex, and probably the reason why indicator pollutants, such as particulate matter are studied instead. In addition, in the case of particulates, concentration-response functions assume all particulates of the same size are equally toxic, which may not be the case, as they may be from different sources, thereby having different characteristics and chemical components. Knowing the characteristics and toxicity of particulates from different sources, will be of use in determining the attribution of specific sources to specific health impacts, which in turn will assist in formulating air pollution control policies.

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APPENDIX A

Some recommended E-R functions per pollutant and health outcome

Pollutant	C-R function per 10 µg/m³ increase (exposure concentrations)*	Health outcome	Reference
$PM_{2.5}$ Long-term	1.06	All-cause mortality	WHO 2013
	1.066	All natural-cause mortality	WHO 2014
	1.12	CV mortality	Pope et al., 2015
	1.10	Respiratory mortality	WHO, 2014
	1.09	Lung cancer mortality	Burnett et al., 2014
	1.05	Ischaemic heart disease mortality	Burnett et al., 2014
$PM_{2.5}$ Short-term	1.012 (daily mean 14 µg/m³)	All-cause mortality	WHO, 2013
	0.40% increase (39 to 177 µg/m³)	Non-accidental mortality	Lu et al., 2015
	1.0091	Hospital admissions for CV disease	WHO, 2013
	0.75% increase	Respiratory mortality	Lu et al., 2015
	1.18	Ischaemic heart disease mortality	Pope et al., 2011
PM_{10} Long-term	1.08 (median 29 µg/m³)	Total mortality	Fischer et al., 2015
	1.0044 (150 to 210 µg/m³)	Total mortality	Maji et al., 2017
	1.06	Cardiovascular mortality	Fischer et al., 2015
	1.05	Cardiovascular mortality	Kim et al., 2017
	1.006	Cardiovascular mortality	Maji et al., 2017
	1.14	Cerebrovascular mortality	Kim et al., 2017
	1.13	Respiratory mortality	Fischer et al., 2015
	1.19	Respiratory mortality	Kim et al., 2017

Pollutant	C-R function per 10 µg/m^3 increase (exposure concentrations)*	Health outcome	Reference
PM_{10} Short-term	0.36 % increase (52 to 174 µg/m^3)	Total mortality	Lu et al. 2015
	0.32% excess risk (44 and 172 µg/m^3)	All-cause mortality	Shang et al. 2013
	1.7% increase	Cardiovascular mortality	Wichmann & Voyi 2012
	0.36% increase	Cardiovascular mortality	Lu et al. 2015
	Excess risk 0.43%	Cardiovascular mortality	Shang et al., 2013
	3.2% increase	Cerebrovascular mortality	Wichmann & Voyi 2012
	1.1% increase	Respiratory mortality	Wichmann & Voyi 2012
	0.42% increase	Respiratory mortality	Lu et al. 2015
SO_2 Long-term	1.0068 (ann ave 16 to 21 µg/m^3)	Total mortality	Maji et al., 2017
	1.0106	Respiratory mortality	Maji et al., 2017
	1.0103	Cardiovascular mortality	Maji et al., 2017
	1.007	Hospital admissions COPD	Maji et al., 2017
	1.0014	Hospital admissions respiratory conditions	Maji et al., 2017
SO_2 Short-term	Excess risk 0.81	All-cause mortality	Shang et al. 2013
	No increase	Respiratory mortality	Wichmann & Voyi 2012
	Excess risk 1.18	Respiratory mortality	Shang et al. 2013
	5.3% increase	Cerebrovascular mortality	Wichmann & Voyi 2012
	3.3% increase	Cardiovascular mortality	Wichmann & Voyi 2012
	Excess risk 0.85	Cardiovascular mortality	Shang et al. 2013
NO_2 Long-term	1.03	All-cause mortality	Jerret et al. 2013; Fischer et al., 2015;

Pollutant	C-R function per 10 µg/m^3 increase (exposure concentrations)*	Health outcome	Reference
			Maji et al., 2017
	1.0206	Cardiovascular mortality	Maji et al., 2017
	No risk	Cardiovascular mortality	Fischer et al., 2015;
	1.048	Cardiovascular mortality	Jerret et al. 2013
	1.02	Respiratory mortality	Fischer et al., 2015
NO_2 Short-term	1.0027	All-cause mortality	WHO, 2013
	Excess risk 1.3	All-cause mortality	Shang et al., 2013
	1.7% increase	Respiratory mortality	Wichmann & Voyi 2012
	Excess risk 1.62	Respiratory mortality	Shang et al., 2013
	2.6% increase	Cardiovascular mortality	Wichmann & Voyi 2012
	Excess risk 1.42	Cardiovascular mortality	Shang et al., 2013

***It is important to read this table together with the spreadsheet containing all concentration-response functions.**