Appendix G.11

HEALTH SCOPING REPORT

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Project conducted on behalf of
WSP Group Africa (Pty) Ltd

Scoping and Baseline Health Assessment for the Rapid Appraisal Health Impact Assessment of the Sasol Secunda Gas to Power Facility

Report No 016-2026 Rev 2.0

Compiled by

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24 Aug 2026

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Managing Director

24 August 2026

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Expertise and Declaration of Independence

This report was prepared by INFOTOX (Pty) Ltd ("INFOTOX"). Established in 1991, INFOTOX is a professional scientific company, highly focused in the discipline of Health Sciences. Both occupational and environmental human health risks, as well as risks to ecological receptors, are addressed.

Dr Willie van Niekerk, Managing Director of INFOTOX, has BSc, Hons BSc and MSc degrees from the University of Potchefstroom and a PhD from the University of South Africa. He is a Qualified Environmental Professional (Environmental Toxicologist QEP), certified by the Institute of Professional Environmental Practice (IPEP) in the USA (No 07960160), and a registered Professional Natural Scientist registered in South Africa (Pr Sci Nat, Environmental Science, No 400284/04). Dr Van Niekerk has specialised in chemical toxicology and human health risk assessments, but he has experience in many other areas in the disciplines of analytical and environmental sciences.

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This specialist report was compiled for WSP Group Africa (Pty) Ltd. We do hereby declare that we are financially and otherwise independent of WSP Group Africa (Pty) Ltd.

Signed on behalf of INFOTOX (Pty) Ltd, duly authorised in the capacity of Managing Director:

Willem Christiaan Abraham van Niekerk

24 August 2026

Executive Summary

Sasol South Africa Limited Ltd ("Sasol", the Applicant) proposes to develop a gas turbine power plant and associated infrastructure at the Sasol Secunda Factory in the Govan Mbeki Local Municipality ("LM"), within the Gert Sibande District Municipality ("DM") in the Mpumalanga Province of South Africa. The proposed project is to be known as the "Sasol Secunda Gas to Power ("GtP") Project".

The Environmental Impact Assessment ("EIA") process for the proposed project is undertaken by WSP Group Africa (Pty) Ltd ("WSP"), for which purpose INFOTOX (Pty) Ltd ("INFOTOX") was appointed to undertake a Rapid Appraisal Health Impact Assessment ("RAHIA") to assess the impact of the proposed project on human health in receptor communities. The main focus of the RAHIA is the impact of substances released or dispersed into air on the health of surrounding communities.

This document presents the scoping report and baseline health assessment for the RAHIA, according to the Good Practice guidance of the International Finance Corporation ("IFC"), a member of the World Bank Group. INFOTOX is guided, amongst other IFC guidelines, by the *Introduction to Health Impact Assessment*.

The most likely hazardous gaseous and particulate pollutants emitted during the combustion of natural gas are nitrogen oxides (NOx, modelled as NO_2 by air dispersion modellers), carbon monoxide (CO) and volatile organic carbons ("VOCs"), with trace amounts of sulfur dioxide (SO_2) and particulate matter ("PM"). PM2.5[1], rather than PM10[2], is the health risk indicator of choice for the assessment of the burden of disease or health risks associated with PM exposure.

The baseline health status of the exposed communities is an important determining factor considered in the assessment of health impacts. The baseline health status is examined by means of a desktop study of available health data for the receptor area, to provide data on the underlying burden of disease for the RAHIA and to identify specific health vulnerabilities that might influence health impacts associated with the proposed operations.

The cardiovascular and respiratory systems are the main targets of exposure to the identified air contaminants of interest. Vulnerability of the receptor population is assessed from available health statistics, such as causes of mortality and hospital admission numbers. Hospital admission numbers are not readily available in the public domain, but cause-of-death statistics are reported annually by Statistics South Africa ("Stats SA") and used to assess the health vulnerabilities of the receptor communities. The receptor communities are tentatively identified as those within a 25-km radius of the proposed Sasol GtP site, but this may be updated at a later stage when die air dispersion modelling domain has been finalised by the air dispersion modellers. The receptor communities currently included are located in the Govan Mbeki LM (Gert Sibande DM) and a small area of the Emalahleni LM within the Nkangala DM of the Mpumalanga Province.

The main health vulnerability findings are:

- In the general all-ages population, for which distinctions are not made between different age groups, a uniformly increased or decreased health vulnerability is not observed in the receptor communities, as compared to the Mpumalanga province at large.

[1] Particulate matter with a nominal mean aerodynamic diameter ≤ 2.5 µm.

[2] Particulate matter with a nominal mean aerodynamic diameter ≤ 10 µm.

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- The comparison of Gert Sibande and Nkangala communities show a mixed pattern in mortality causes related to the cardiovascular and respiratory systems, so that it cannot be concluded that one community is consistently more vulnerable than the other.

- Increased vulnerability to the health effects of air pollution in the aged (65+) in the Sasol GtP receptor communities is indicated, based on provincial mortality numbers. A similar pattern but involving fewer causes is observed for children younger than 15 years of age.

The health hazards of the pollutants of interest are described in this report, with internationally accepted methods that will be used to quantify the risk of health effects associated with exposure.

- The preliminary health impact assessment statement is:
  - During normal operational phases, the releases of particulate matter and gaseous pollutants are expected to be negligible to minimal, because of the properties of the feeding gas and the advanced emissions abatement technology to be used in the GtP facility.
  - Therefore, associated health impacts should be minimal, even when considering that children and the aged community are vulnerable to the health effects of these pollutants.

- The plan of study for the impact assessment for the next phase of the project entails:
  - Use of the established internationally accepted methods to calculate health risks based on modelled ambient air concentrations of the pollutants of interest, which will be available from air dispersion modelling specialists in later stages of the Environmental Impact Assessment ("EIA") for the proposed Sasol Secunda GtP facility.
  - Calculated health risks will be combined with the health vulnerability assessment of the receptor communities, in order to conduct the community health impact assessment for the GtP facility, to be reported in the future RAHIA report.

Based on the preliminary assessment, there is no health-based reason not to proceed to the RAHIA stage.

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List of Symbols and Abbreviations

Δp	Modelled change in air pollutant concentration
β	Coefficient of the pollutant concentration in the function describing the exposure-response curve
Ln	The natural logarithm
AF	Attributable fraction of cases
ALRI	Acute lower respiratory infections
AQG	Air quality guideline
AQIA	Air Quality Impact Assessment
ATSDR	US Agency for Toxic Substances and Disease Registry
B	The population incidence of mortality or morbidity (e.g., deaths or hospitalisation rates per 1 000 people)
BenMAP	Benefits Mapping and Analysis Program
CCGT	Combined Cycle Gas Turbines
CHF	Congestive heart failure
CHP	Combined Heat and Power
CNS	Central nervous system
CO	Carbon monoxide
COHb	Carboxyhaemoglobin
COPD	Chronic obstructive pulmonary disease
CRF	Concentration-response function
CVD	Cardiovascular diseases
DM	District Municipality
E	Potential mortalities or morbidities per day or per year due to exposure to the pollutant
ECTF	Expert Consensus Task Force
ED, ED visits	Emergency Department of a hospital, visits to the Emergency Deparment
EIA	Environmental Impact Assessment
Exp	The constant (e), the base of the natural logarithm
GRADE	Grading of Recommendations Assessment, Development and Evaluation
GtP	Gas to Power
HA(s)	Hospital Admission(s)
HHRA	Human health risk assessment
HIV	Human immunodeficiency virus
HRAPIE	Health Risks of Air Pollution in Europe
IARC	International Agency for Research on Cancer
IBL	Inside Battery Limit
ICD	International Statistical Classification of Diseases and Related Health Problems of the WHO
IFC	International Finance Corporation
IHD	Ischemic heart disease
HIV	Human immunodeficiency virus
LM	Local Municipality
LNG	Liquefied Natural Gas
MM	Metropolitan Municipality

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MI	Myocardial infarction
MRG	Methane rich gas
NEM:AQA	National Environment Management: Air Quality Act
NO_2	Nitrogen dioxide
OBL	Outside Battery Limit
OCGT	Open cycle gas turbines
P	Size of the exposed population (number)
PM	Particulate matter (airborne)
PM2.5	Particulate matter with a nominal mean aerodynamic diameter equal to or less than 2.5 µm
PM10	Particulate matter with nominal mean aerodynamic diameter equal to and less than 10 µm
RAHIA	Rapid Appraisal Health Impact Assessment
REVIHAAP	Review of Evidence on Health Aspects of Air Pollution
RR, RR ratio	Relative Risk, Relative Risk Ratio
Stats SA	Statistics South Africa
SO_2	Sulfur dioxide
TB	Tuberculosis
USEPA	United States Environmental Protection Agency
VOCs	Volatile organic carbons
WHO	World Health Organization
$X - X_0$	The modelled incremental change (Δp) in the pollutant concentration ($\mu g/m^3$)

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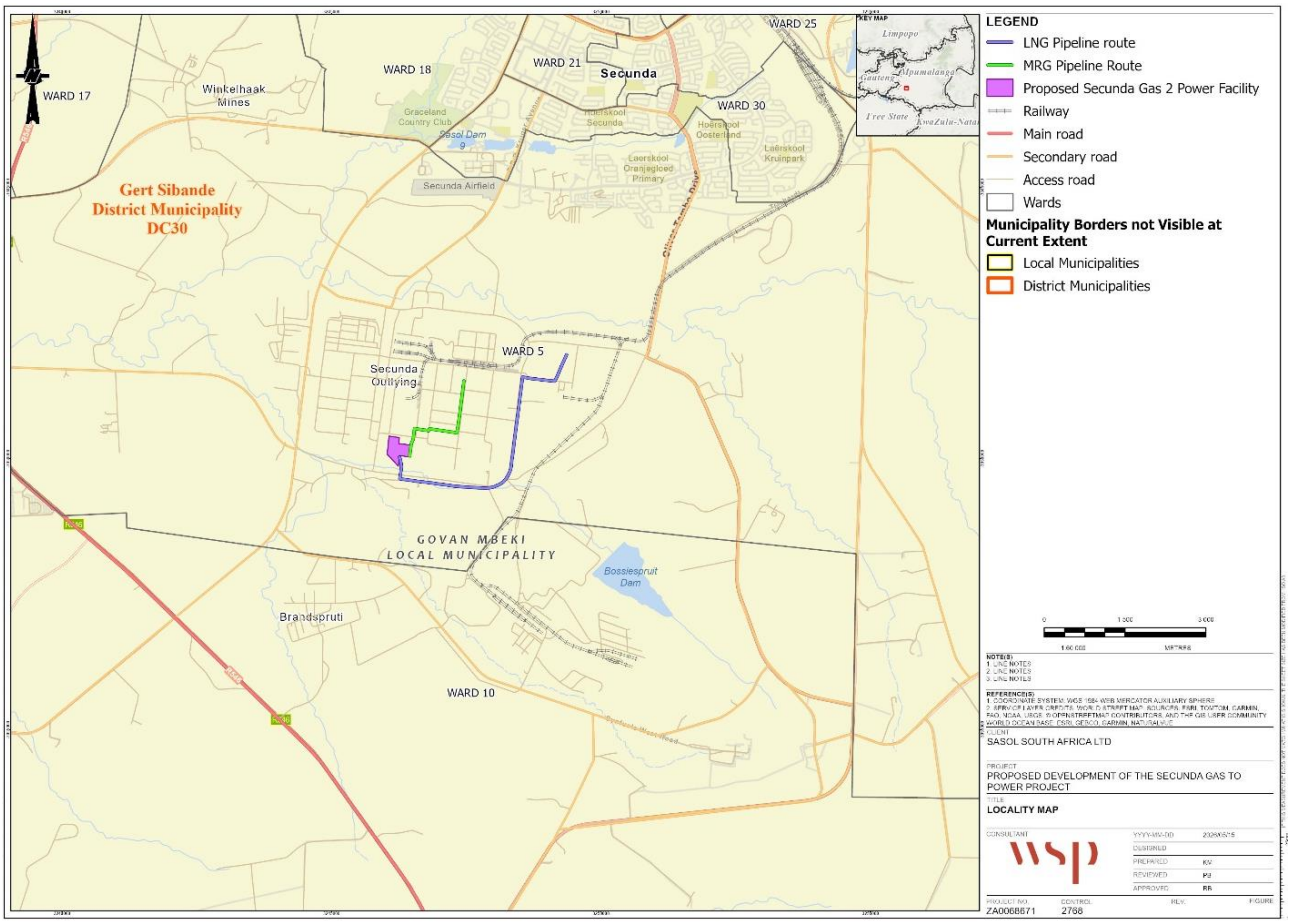
1 Introduction and project description

WSP Group Africa (Pty) Ltd (“WSP”) appointed INFOTOX (Pty) Ltd (“INFOTOX”) to conduct a rapid appraisal health impact assessment (“RAHIA”) for the development of the Sasol Secunda Gas to Power Facility (“Sasol GtP Facility”) and associated infrastructure to be located at the Sasol Secunda Factory in the Govan Mbeki Local Municipality (“LM”), within the Gert Sibande District Municipality (“DM”) in the Mpumalanga Province of South Africa. The locality in Secunda is indicated in the regional locality map for the project (Figure 1.1).



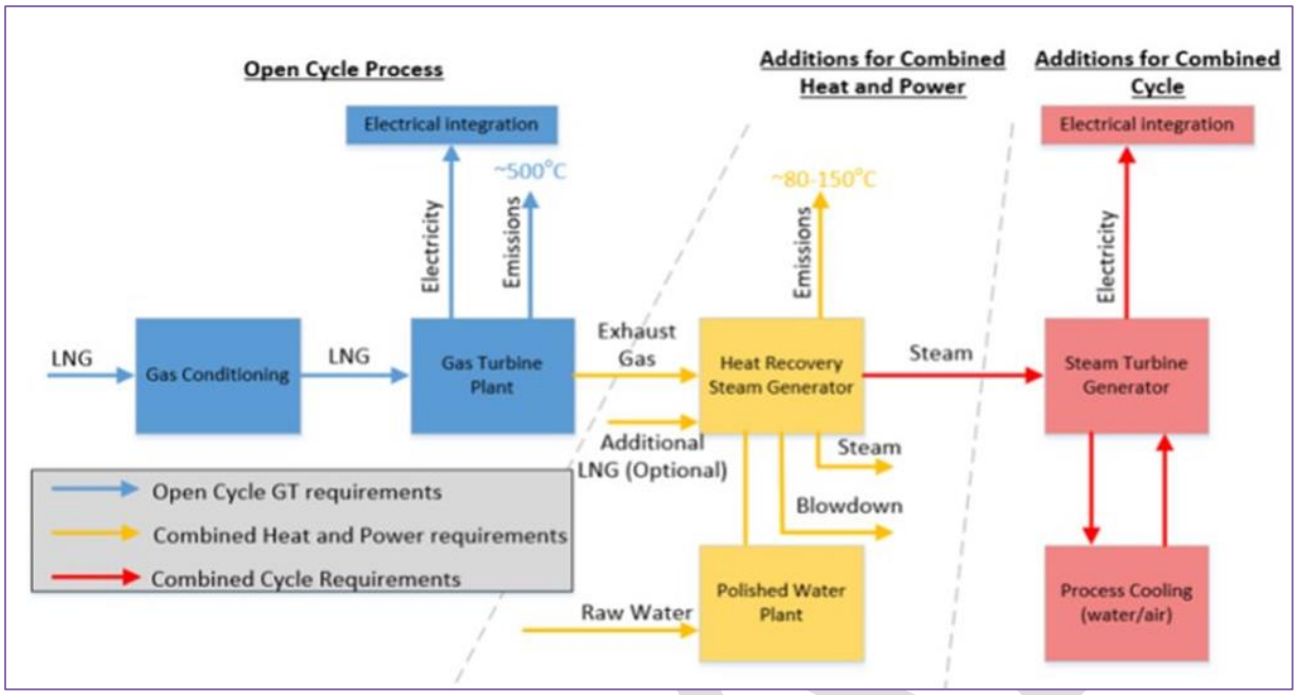
Source: WSP Group Africa (Pty) Ltd. (2026a).

Figure 1.1: Regional locality map of the Sasol Secunda Gas to Power Facility.

The plant will generate electricity through the combustion of Liquefied Natural Gas (“LNG”), with methane rich gas (“MRG”) being considered as back-up feed. The objective is to provide power to the national grid, supporting South Africa’s growing energy needs.

The relevant listed activity in terms of the National Environment Management: Air Quality Act 39 of 2004 (NEM:AQA) is the combustion of natural gas.

Two configurations were evaluated for the Gas to Power (“GtP”) facility, presented in Figure 1.2. These were Open Cycle Gas Turbines (“OCGT”) indicated in blue in Figure 1.2, Combined Heat and Power (“CHP”) in blue and yellow, and combined cycle gas turbines (“CCGT”) indicated in blue, yellow and red. Alternatives were considered, but it has been established that CCGT is the option to be taken forward (WSP Group Africa (Pty) Ltd. 2026a).



Note to figure: LNG = Liquefied Natural Gas. GT = Gas Turbine. Source: Sasol South Africa Ltd (2024).

Figure 1.2: Overview of configurations evaluated for the GtP facility.

The plant infrastructure will include the following main equipment:

- The Inside Battery Limit ("IBL") scope includes the main equipment for generating electricity.
- The Outside Battery Limit ("OBL") scope will focus on the external infrastructure and utilities required to support the GtP facility (integration into the Secunda facility):
  - The electrical scope will cover the reconfiguration of the electrical network.
  - The installation and routing of various pipelines, including for LNG, MRG and any required utilities such as cooling water, raw water, firewater, instrument air, and any effluent, depending on the IBL requirements.
  - Raw water treatment will also be included to achieve the necessary water quality specifications.

The current land use for the immediate project area is industrial, but the relative agriculture theme sensitivity in the surrounding area is indicated as high to medium (WSP Group Africa (Pty) Ltd. 2026b). More details are not currently available.

This document presents the scoping report and baseline health assessment for the RAHIA, according to the Good Practice guidance of the International Finance Corporation ("IFC"), a member of the World Bank Group (IFC 2009). INFOTOX is guided, amongst other IFC guidelines, by the *Introduction to Health Impact Assessment*.

2 Terms of reference for the RAHIA

2.1 Health baseline

According to the IFC, the RAHIA format does not require new health data collection within the communities of concern, because data necessary to assess potential health impacts of the proposed development are extracted from existing health data sources in a desktop review. Concerning the Sasol GtP facility, the main focus of the RAHIA is the impact of substances released or dispersed

into air on the health of surrounding communities. A significant influx of people settling in the area, due to the construction and operation of the facility, is not foreseen.

The baseline health status of the exposed communities (this report) is an important determining factor considered in the assessment of health impacts. It is assessed based on a desktop study of available health data for the receptor area. The intention is to provide baseline health data on the underlying burden of disease for the RAHIA and to identify specific vulnerabilities that might influence health impacts associated with the proposed operations.

In terms of the RAHIA, the geographical study area considered as impacted include those areas and communities where the proposed developments might have an impact on the environmental quality. Assessment of exposure in the residential areas to the likely airborne emissions from the project site is vital. The impact of such emissions on air quality is determined by an air dispersion modelling specialist. Amongst other factors, the specialist takes into account the local topographical and meteorological conditions in the modelling domain.

2.2 Ambient air contaminants of interest

It is expected that the air quality impact of the proposed facility could result in elevated ambient air pollutant concentrations. The most likely hazardous gaseous and particulate pollutants emitted during the combustion of natural gas are nitrogen oxides (NOx, modelled as NO_2 by the air dispersion modellers), carbon monoxide (CO) and volatile organic carbons ("VOCs"), with trace amounts of sulfur dioxide (SO_2) and particulate matter ("PM") (USEPA online). PM2.5[1], rather than PM10[2], is the health risk indicator of choice for the assessment of the burden of disease or health risks associated with PM exposure, as discussed in Section 7.1.

2.3 Plan of study

The plan of study for the RAHIA proceeds via two main items, namely, the scoping report and the RAHIA report.

For the purposes of the scoping report:

- Locate the geographical areas most likely to be impacted by airborne GtP facility emissions. Identify the general urban or rural communities in the area of impact (see Section 3.1).
- Obtain estimates of population numbers and the age group structure of the identified receptor communities (provided in Section 3.2).
- Obtain statistics to assess and summarise the baseline health of the identified receptors. The baseline health assessment is used to understand the likely health vulnerabilities of receptor communities to the health effects of the air pollutants of interest. Relevant data are presented in Section 3.3.
- Review the health hazards of pertinent substances expected in project emissions (presented in Annexures 1 to 4). The identification of pertinent substances is dependent on the project parameters, that is, the proposed operations and operating conditions of the GtP facility. Identification of emissions are mostly done by specialist engineers, or the air dispersion modelers may consult regulatory emission factor documents (e.g., USEPA online).
- Establish and describe methods that will be used to quantify the health risks of identified hazardous airborne substances (Section 4).

[1] Particulate matter with a nominal mean aerodynamic diameter ≤ 2.5 µm.
[2] Particulate matter with a nominal mean aerodynamic diameter ≤ 10 µm.

For the purposes of the RAHIA:

- Obtain modelled air concentrations of the pollutants of interest, expected in the identified receptor communities, from the final Air Quality Impact Assessment ("AQIA").
- Conduct the human health risk assessment ("HHRA") for the health effects associated with the pollutants of interest, based on the methods presented in Sections 5 and 7 of this report.
- Conduct the health impact assessment by integrating the health baseline vulnerability assessment (see Section 3.3) and the health risk results to be obtained from the planned HHRA.

3 Study area

3.1 Receptor area

The proposed project area is located in Ward 5, Govan Mbeki LM. For the purposes of the scoping report, it is assumed that the area of impacted air quality would involve the surrounding 25 km range in Figure 3.1.1. This will be adjusted in the RAHIA, to the modelling domain established by the air dispersion specialists. A ward collage is presented in Figure 3.1.2.

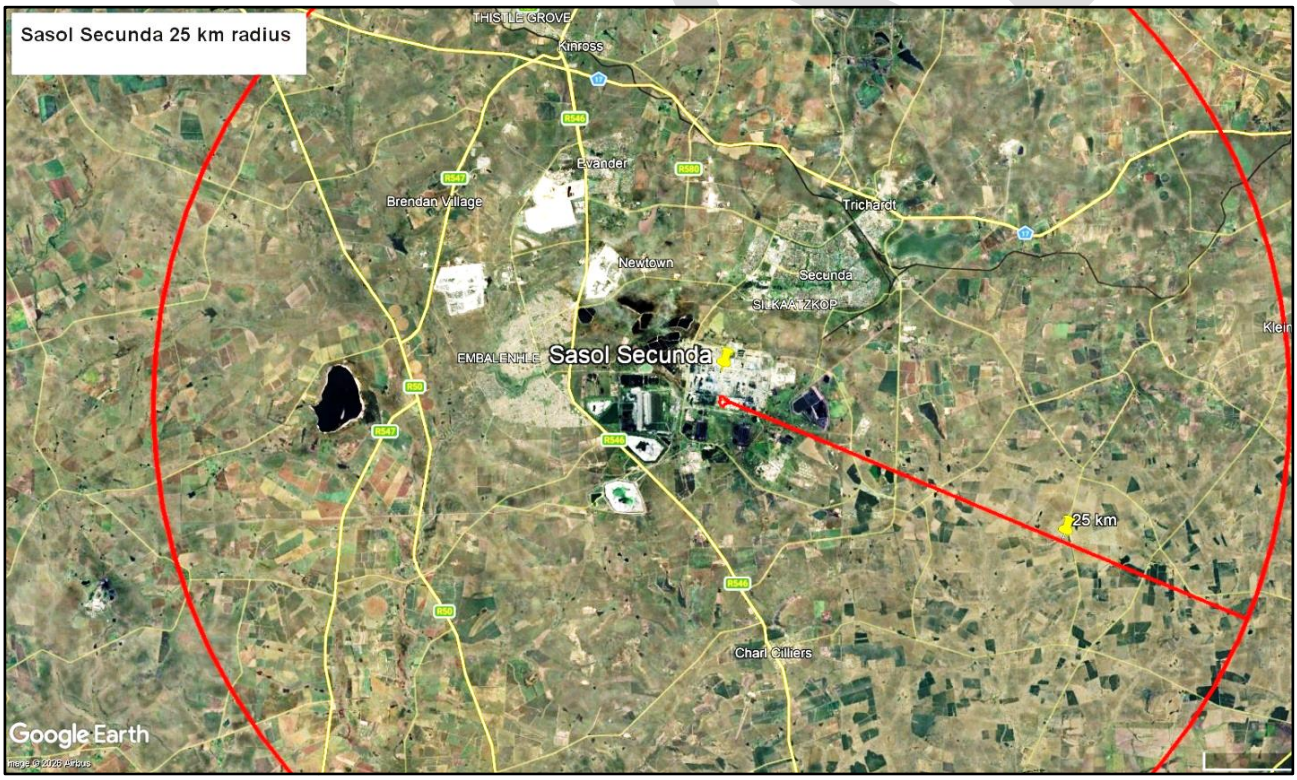
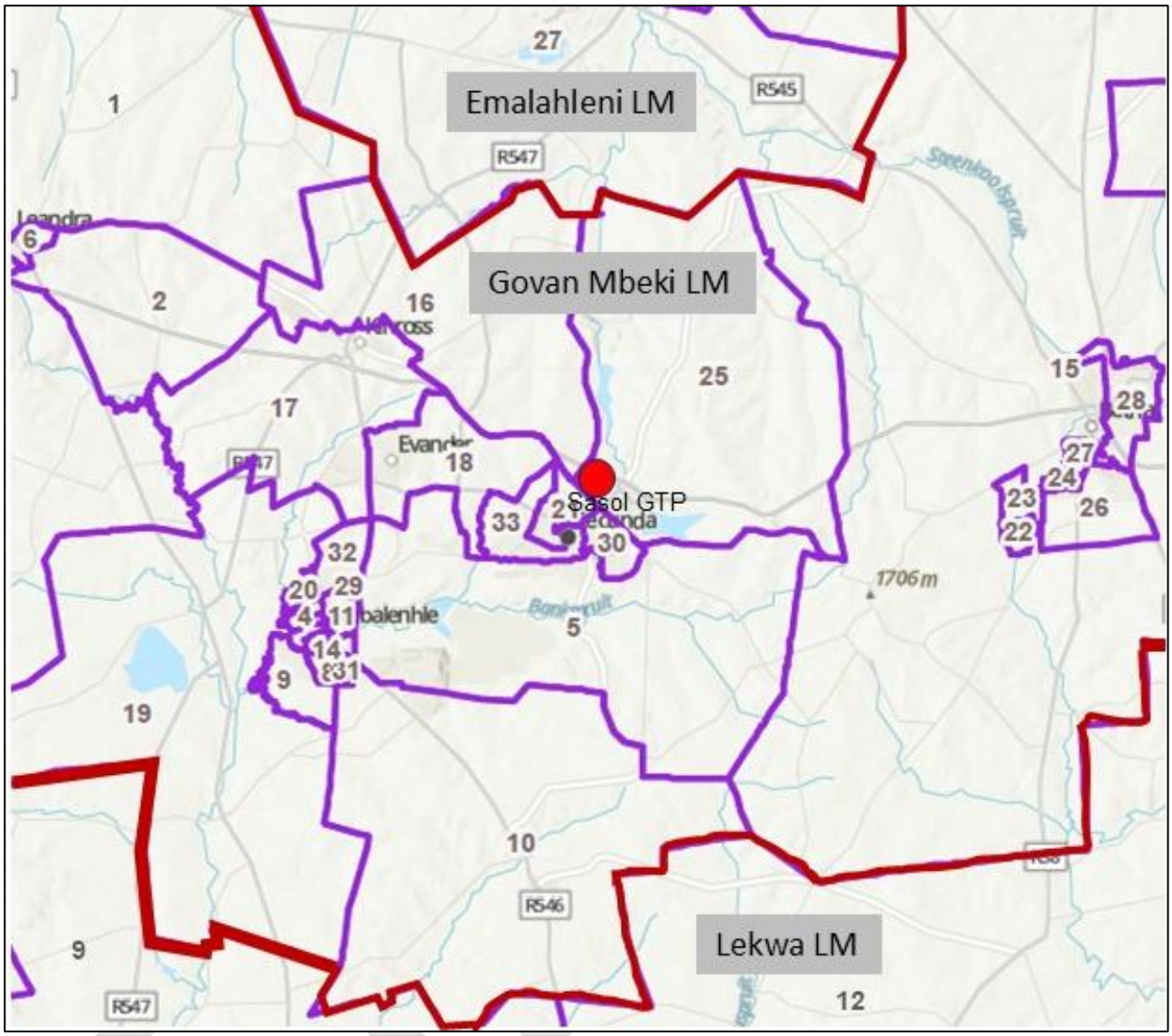


Figure 3.1.1: 25-Km range surrounding the Sasol Secunda Gas to Power Facility.



Note to figure: The Sasol GtP location is approximate.
Source: Municipal Demarcation Board (2025).

Figure 3.1.2: Municipal wards surrounding the Sasol Secunda Gas to Power Facility.

3.2 Demography and age-related vulnerability to air pollutant health effects

Younger children and older adults are more vulnerable to the effects of air pollution in general. The Expert Consensus Task Force of the China CDC (ECTF 2022), for example, identifies children, the elderly, and patients with cardiovascular or respiratory diseases as subpopulations with increased vulnerability to the health risks of PM2.5 pollution. This agrees with other and earlier opinions and findings. Millar et al. (2022), citing Lake et al. (2019) and Chen et al. (2015), emphasised the particular vulnerability of children and adolescents, even at low levels of exposure to air pollution.

Children and adolescents are particularly vulnerable to the effects of air pollution in general, for several reasons (Millar et al. 2022, WHO 2018 and American Lung Association 2022):

- Children are more active and breathe more rapidly than adults, absorbing more pollutants.

- Children inhale more polluted outdoor air than adults typically do, because they are outside for longer periods and are usually more active when outdoors, e.g., walking to school, participating in sport, or playing outdoors.
- The lungs and brain, along with other organs, and their metabolic functions, are still in the process of development and maturation.

Children are more vulnerable to specifically the respiratory effects of air pollution, due to the following reasons (WHO 2018 and American Lung Association 2022):

- Their lungs and airways are smaller, which increases vulnerability to respiratory effects.
- Their immune systems are still developing, and with this their ability to fight off infections, also of the respiratory tract.
- Children have more respiratory infections than adults, which also seems to increase their susceptibility to air pollution.

Since younger children and older adults are more vulnerable to the effects of air pollution by default, it is important to assess the proportion of younger (<15 years) and older (≥65 years) age groups in the receptor population. The receptor population is constituted of the communities in municipal wards within the air quality impact area (Figure 3.1.2). The impacted are is assumed, for the time being, to fall within the 25-km radius from the project site (Figure 3.1.1), but this might change when the final air dispersion modelling domain is decided by the air dispersion modelling specialists. Statistics South Africa ("Stats SA") has made available the ward-level population- and age-group numbers from the 2022 Population Census. National (SA), provincial, DM, LM and ward-level age group percentages are summarised in Table 3.2.1.

The comparison of age group percentages in Table 3.2.1 shows that the proportions of vulnerable age groups in the receptor communities within the modelling domain are fairly comparable to the respective larger community, on the provincial, DM and LM level. Although there are small differences, there is not a concentration of younger or older vulnerable people in the receptor wards, as would be seen, in the example of older people, in peri-urban or rural residential areas that are popular retirement destinations.

It is concluded that there is not an indication of unusual age-related vulnerability in the receptor communities.

Table 3.2.1: National, provincial, DM and LM age-group percentages from the 2022 Census.

Province, DM and LM	<5 years (%)	<15 years (%)	65+ years (%)
South Africa	9.4	26.3	6.5
Mpumalanga Province	10.6	28.4	5.2
Gert Sibande DM	10.0	27.3	5.1
Govan Mbeki LM	9.9	25.4	4.7
Govan Mbeki LM wards within the modelling domain	9.8	25.2	4.5
Nkangala DM	9.8	26.7	6.0
Emalahleni LM	9.8	26.7	6.0
Emalahleni LM (only one ward within the modelling domain)	6.7	18.3	10.4
Reference: Stats SA (online).			

3.3 Baseline health status and receptor community vulnerability

3.3.1 Health conditions indicating vulnerability to air pollutant health effects

The cardiovascular and respiratory systems are the main targets of exposure to the air contaminants of interest identified in Section 2.2. Sensitivity of the receptor population is assessed from available health statistics, such as causes of mortality and hospital admission numbers. Hospital admission numbers are not available in the public domain, unless admissions numbers are the subject of health survey research, which is usually focussed on specific hospitals or causes for admission. Cause-of-death statistics reported annually by Stats SA are readily available for assessment and serve as the proxy for assessment of the health vulnerabilities of the receptors.

Causes of death are broadly categorised as either natural or non-natural causes. Examples of non-natural causes of death are transport accidents, intentional self-harm and assault. Natural causes are non-violent, non-intentional, non-accidental causes, such as heart attacks and pneumonia. These are not the result of accidental or intentional harm.

The baseline health status of the receptor population, and their vulnerability to the health effects of air pollution, are assessed from the statistics on natural causes, with a focus on mortality caused by poor health of the respiratory and cardiovascular systems, and other health indicators of vulnerability of these systems to the impacts of air pollution. Health effects and causality determinations for PM2.5, SO_2, NO_2 and CO are summarised in Annexures 1 to 4.

People living with asthma, chronic obstructive pulmonary disease ([3]"COPD") and other chronic diseases, including cardiovascular disease and diabetes, are vulnerable to the effects of air pollution. The cellular injury and inflammation triggered by breathing air pollution puts additional stress on the lungs, heart and other organs that are already compromised by disease. This can result in a worsening of symptoms, increased medication use and an overall reduced quality of life (American Lung Association 2023). Regarding diabetes as a factor indicating vulnerability to air pollutant effects in general, it is important to note that air pollution does not *cause* increased rates of diabetes in exposed populations (WHO 2021 and USEPA 2019).

Diseases indicating vulnerability of the respiratory tract include viral infections, tuberculosis ("TB"), and a compromised immune system, such as in persons infected with the human immunodeficiency virus ("HIV"), increasing vulnerability to respiratory diseases.

Regarding the cardiovascular system, the causes of death from Stats SA (2026) included by INFOTOX in the cardiovascular (with stroke) mortality group are:

- Ischaemic heart diseases (codes I20 to I25)
- Cerebrovascular diseases (codes I60 to I69)
- Other forms of heart disease (codes I30 to I52)

For the purpose of this report, cerebrovascular diseases are equated to stroke. The codes in brackets are the Stats SA (2026) grouping according to the 10th Revision of the ICD-10 codes, the

[3] Chronic inflammatory lung disease that causes obstructed airflow from the lungs, causing difficulty in breathing.

disease classification codes of the International Statistical Classification of Diseases and Related Health Problems, maintained by the World Health Organization (WHO online).

3.3.2 Available age-related Stats SA mortality data

Given the known vulnerability of children and the aged, the baseline health assessment should focus on the young, the elderly and those at risk of cardiovascular and respiratory diseases. The relevant age groups for which mortality data are readily available from Stats SA are the group of children younger than 15 years of age and adults aged 65 years and older. Specific attention is given to the health status of these groups.
Stats SA's most recent cause-of-death data are for 2023, released in 2026 (Stats SA 2026). Cause-of-death data specific to LMs are not available, and mortality data for DMs are only available for the all-ages population, not differentiated according to age groups. Stats SA provides age-group differentiated mortality data only to the provincial level; therefore, provincial mortality data for children (<15 years) and the aged (≥65 years) are used as a proxy for the DMs and LMs within the receptor area.

Cause-of-death data reported in Stats SA (2026) for the under 15 population are divided into the infant (younger than 1 year of age) and the 1-to-14 years age group. By far the majority of causes of infant deaths are specific to the perinatal[4] period and do not play a significant role, or no role at all, in deaths after age 1. Causes of infant deaths are mostly affected by maternal factors and complications of pregnancy, labour and delivery, which are not related to environmental factors, but to factors specific to the foetus, infant, mother or events at the time of delivery. Therefore, causes of death indicating vulnerability to the health effects of air pollutant exposure in the 1-to-14 age group are viewed as representative of the vulnerability of infants (<1 year of age), and only mortality data for the 1-to-14 age group is assessed to understand the vulnerability of the under-15 (child) population.

To illustrate this point, the first three most important causes of death in infants in South Africa (Stats SA 2026) were respiratory and cardiovascular disorders specific to the perinatal period (15.9% of all mortalities in this age group), followed by other disorders originating in the perinatal period (9%) and infections specific to the perinatal period (5.5%). The most highly ranked cause of mortality indicating vulnerability to the health effects of air pollution exposure in infants was influenza and pneumonia, the 5th most important (5%). Compared to the infant group, influenza and pneumonia was the most important cause of mortality in the 1-to-14 age group (6% of all mortalities in this age group). Thus, for vulnerability to air pollutants, mortality causes in the 1-to-14 age group can be viewed as sufficiently representative of vulnerability in the under-15 age group (including infants).

3.3.3 Mortality data in the receptor communities

Mortality causes amongst people of all ages, which indicate potential vulnerability to the health effects of air pollutants in the Mpumalanga province DM receptor areas are summarised in Table 3.3.3.1. DM percentages of deaths notably higher than the provincial percentages, indicating increased vulnerability in the DM communities, are shaded in red and those notably lower are shaded in green.

Mortality causes indicating potential vulnerability to air pollution health impacts in children and the aged are presented in Tables 3.3.3.2 and 3.3.3.3, respectively. Average provincial percentages of

[4] A number of definitions are used, including from gestation week 22 up to 7 days after birth. It encompasses the critical phase just before and just after birth, relevant for studying maternal and newborn health outcomes.

deaths notably higher than the national percentages, indicating increased vulnerability, are shaded in red and those notably lower are shaded in green. Malnutrition mortality is additionally assessed in the 1-to-14 age group, because malnutrition affects the immune system detrimentally, increasing vulnerability to the respiratory health effects of air pollution (Morales et al. 2023).

Table 3.3.3.1: Leading underlying natural causes of death in 2023, all ages, indicating vulnerability to air pollutants in Mpumalanga province and receptor DMs.

Cause of death with ICD10 codes	Mpumalanga Province		Gert Sibande DM		Nkangala DM		Receptor DMs average %
	Rank	%	Rank	%	Rank	%	
Diabetes mellitus (E10-E14)	1	6.3	2	6.7	2	6.2	6.5
Cerebrovascular diseases (I60-I69)	4	4.9	4	4.9	4	4.2	4.6
Hypertensive diseases (I10-I15)	2	6.2	1	7.0	1	7.4	7.2
Influenza and pneumonia (J09-J18)	7	3.8	7	3.7	3	4.4	4.1
Other forms of heart disease (I30-I52)	9	3.0	8	3.4	8	3.2	3.3
Ischaemic heart diseases (I20-I25)	3	4.9	Not ranked	<2.6	6	3.7	3.2
Tuberculosis (A15-A19)	6	3.9	6	4.0	7	3.2	3.6
Other viral diseases (B25-B34)	8	3.7	5	4.2	Not ranked	<2.2	3.2
Human immunodeficiency virus [HIV] disease (B20-B24)	5	4.2	3	5.1	5	3.8	4.5
Chronic lower respiratory diseases (J40-J47)	Not ranked	<2.1	Not ranked	<2.6	10	2.2	2.4

%: Percentage of deaths due to cause.
Rank: Rank of cause of death, from 1 to 10 based on the number of deaths.
Not ranked: Not among the top 10 causes of death. The percentages of unranked causes were assumed to be lower than the cause ranked 10th.
Source: Stats SA (2026).

The burden of death summary (Table 3.3.3.1) informs the following conclusions on the all-ages baseline health of receptor communities and their potential vulnerability to the effects of air pollutants:

- The vulnerability of the Sasol GPT receptor area, approximated from the mortality rates in the Gert Sibande and Nkangala DMs, is not uniformly increased or decreased in comparison to the Mpumalanga province.

- The cardiovascular and respiratory systems are not conclusively more or less vulnerable than in the province as a whole, with mixed lower and higher mortality rates.

- Gert Sibande communities are more vulnerable than Nkangala communities in some aspects related to the respiratory system, as indicated by notably higher mortality numbers due to HIV and “other” viral diseases. On the other hand, moderately increased influenza and pneumonia mortality in the Nkangala communities suggest increased vulnerability. This mixed pattern is also observed in causes related to the cardiovascular system; therefore, it cannot be concluded that one community is consistently more vulnerable than the other.

Regarding the populations of children (1-to-14-years) and older people (65+), the mortality percentages due to specific causes in the receptor provinces are compared with percentages in the general South African population, to derive an indication of the potential health vulnerabilities of the

Sasol GtP facility receptor communities. Percentages of deaths notably higher than the national percentages, potentially indicating increased vulnerability in the receptor population, are shaded in red and those notably lower are shaded in green (Tables 3.3.3.2 and 3.3.3.2).

There is a pattern of increased vulnerability in the aged in Mpumalanga in comparison to South Africa in general (Table 3.3.3.2), with a similar but less accentuated pattern observed in the mortality numbers for children (Table 3.3.3.3).

Table 3.3.3.2: Leading underlying causes of death in 2023 in the population older than 65 years of age.

Cause of death with ICD10 codes	South Africa		Free State	
	Rank	%	Rank	%
Hypertensive diseases (I10-I15)	1	9.2	1	11.9
Diabetes mellitus (E10-E14)	2	8.8	2	10.2
Cerebrovascular diseases (I60-I69)	3	8.4	3	8.3
Ischaemic heart disease (I20-I25)	4	4.2	4	7.0
Other forms of heart disease (I30-I52)	5	4.2	5	4.4
Influenza and pneumonia (L09-J18)	7	3.1	6	3.8
Chronic lower respiratory diseases (J40-J47)	6	3.7	7	2.9

Table 3.3.3.3: Leading underlying causes of death in 2023 for the 1-to-14-years population.

Cause of death with ICD10 codes	South Africa		Mpumalanga	
	Rank	%	Rank	%
Influenza and pneumonia (L09-J18)	1	6.0	2	7.0
Tuberculosis (A15-A19)	4	2.2	7	1.9
Malnutrition (E40-E46)	6	2.1	9	1.6
Other forms of heart disease (I30-I52)	7	1.7	Not Ranked	<1.6
HIV (B20-B24)	9	1.5	Not Ranked	<1.6
Diabetes mellitus (E10-E14)	Not Ranked	<1.5	Not Ranked	<1.6
Other diseases of the respiratory system (J95-J99)	Not Ranked	<1.5	Not Ranked	<1.6
Other viral diseases (B25-B34)	10	1.5	Not Ranked	<1.6
Other acute lower respiratory infections (J20-J22)	Not Ranked	<1.5	6	2.1

3.3.4 Baseline health status summary

It must be stated at the outset that mortality due to diabetes does not indicate a detrimental effect of current air pollution on health of communities, because diabetes is caused primarily by dietary and lifestyle factors, and not by air pollutants. Furthermore, factors such as service delivery problems in the provincial public health sectors can contribute to mortality due to diabetes. The presented

diabetes mortality numbers must be interpreted with this in mind. However, persons with diabetes can be viewed as potentially vulnerable to the health effects of air pollution, because persistent high blood sugar directly damages blood vessels and nerves, leading to vascular disease and organ failure (Banday et al. 2020).

It is concluded that the comparison of relevant causes of death indicate a likely increased vulnerability to the health effects of air pollution in the aged (65+) in the Sasol GtP receptor communities.

There is a degree of uncertainty related to this comparative assessment, since mortality data specific to the receptor municipal wards are not available, but it is unlikely that an unobserved significantly increased vulnerability above that indicated by available data should exist.

4 Risk calculations for criteria pollutants

In general, predicted (modelled) or measured (monitored) impacts of industrial emissions on air concentrations are used as a basis to quantify impacts on health. This is achieved by calculating the potential increase in or contribution to hospital admissions or mortality due to specific causes, associated with air concentrations of specific pollutants. These calculations are based on results of epidemiological studies reported in the international scientific literature in which statistical methods were used to compare changes in hospitalisation or mortality rates with changes in air quality. Current statistical methods use the concept of relative risk ("RR") to derive the potential percentage increase in or contribution to effects.

The potential number of deaths or hospital admissions associated with the concentration of a pollutant contributed by a specific source is calculated using the following equations and methods of the World Health Organization (WHO 2005 and Ostro 2004).

$$E = AF \times P \times B / 1000 \quad (4.1)$$

Where:

E	Potential mortalities or morbidities per day or per year due to exposure to the pollutant
AF	The attributable fraction of mortalities or morbidities due to exposure to the pollutant
B	The population incidence of mortality or morbidity (e.g., deaths or hospitalisation rates per 1 000 people)
P	Size of the exposed population (number)

The *AF* may be considered as the fraction of the health effect incidence in the exposed population that could be prevented if exposure to the pollutant was eliminated (Last et al. 2000) and is calculated as follows:

$$AF = \frac{(RR - 1)}{RR} \quad (4.2)$$

Where:

RR	The relative risk of mortality or morbidity due to exposure to the pollutant

In cases where incremental changes in pollutant concentrations (Δp) are estimated, the relative risk of mortality or morbidity in the exposed population may be calculated.

The equation for the calculation of the RR is as follows (Ostro 2004 and USEPA 2022):

$$RR = exp[\beta (X - X_0)] \quad (4.3)$$

Where:

Exp	Is the constant (*e*), the base of the natural logarithm
β	Is the coefficient of the pollutant concentration in the function describing the exposure-response curve
$X - X_0$	The modelled incremental change (Δp) in the pollutant concentration (µg/m^3)

Epidemiological studies often report a RR for a given Δp, rather than the coefficient, *β*.

β can be derived from Equation 4.4 as:

$$\beta = [Ln(RR)] / \Delta p \quad (4.4)$$

Where:

β	Is the coefficient of the pollutant concentration in the function describing the exposure-response curve
Ln	Is the natural logarithm
RR	The RR reported in the epidemiological study
Δp	The modelled incremental change reported in the epidemiological study

The Δp used in the above equations can be calculated per day or per year. If the averaging time of the pollutant was reported as daily (the daily mean concentration) in the literature, the daily population incidence of the health effect must be estimated (per 1 000 people) and the modelled daily mean concentration is used to calculate the potential daily increase in the particular health effect incidence. These health risks may also be expressed as a personal risk of experiencing key health effects associated with exposure to a criteria air pollutant.

Health effects and causality determinations for PM2.5, SO_2, NO_2 and CO are summarised in Annexures 1 to 4.

5 Conclusion

- The purpose of the scoping report was to assess the potential ambient air emissions of the Sasol GtP facility with the aim of identifying potential impacts on health, for which risks need to be assessed in the RAHIA report.

- The identified ambient air contaminants of interest are PM2.5, SO_2, NO_2 and CO. The associated potential health effects are mortality due to natural causes, cardiovascular and respiratory causes of death.

- Internationally accepted methods of calculating community risks of health effects due to an air quality impact of emissions from the proposed GtP facility were described.

- The vulnerability of the receptor communities to the health impacts of the ambient air contaminants of interest was evaluated based on the assessment of provincial and DM mortality statistics for the identified health effects.

- The conclusion regarding vulnerability is that the aged community (65+ years) and children younger than 15 years of age in the project area are vulnerable to the health effects of gaseous pollutants (primarily SO_2, NO_2 and CO) and particulate matter potentially emitted from the proposed GtP facility.

- The preliminary health impact assessment statement is:
  - During normal operational phases, the releases of particulate matter and gaseous pollutants are expected to be negligible to minimal, because of the properties of the feeding gas and the advanced emissions abatement technology to be used in the GtP facility.
  - Therefore, associated health impacts should be minimal, even when considering that children and the aged community are vulnerable to the health effects of these pollutants.

- The plan of study for the impact assessment for the next phase of the project entails:
  - Use of the established internationally accepted methods to calculate health risks based on modelled ambient air concentrations of the pollutants of interest, which will be available from air dispersion modelling specialists in later stages of the Environmental Impact Assessment ("EIA") for the proposed Sasol Secunda GtP facility.
  - Calculated health risks will be combined with the health vulnerability assessment of the receptor communities, in order to conduct the community health impact assessment for the GtP facility, to be reported in the future RAHIA report.

- Based on the preliminary assessment, there is no health-based reason not to proceed to the RAHIA stage.

6 References

American Lung Association. 2023. Who is Most Affected by Outdoor Air Pollution? American Lung Association. https://www.lung.org/clean-air/outdoors/who-is-at-risk.

Banday MZ, Sameer AS, Nissar S. 2020. Pathophysiology of diabetes: An overview. Avicenna J Med, 10(4):174-188. doi: 10.4103/ajm.ajm_53_20. PMID: 33437689; PMCID: PMC7791288.

Chen Z, Salam MT, Eckel SP, Breton CV, Gilliland FD. 2015. Chronic effects of air pollution on respiratory health in Southern California children: findings from the Southern California Children's health study. J Thorac Dis, 7(1):46. Cited in Millar et al. (2022).

ECTF. 2022. Expert Consensus Task Force, Shi X, Duan G. Recommendations of Controlling and Preventing Acute Health Risks of Fine Particulate Matter Pollution - China, 2021. China CDC Wkly, Apr 22;4(16):329-341. doi: 10.46234/ccdcw2022.078. PMID: 35548322; PMCID: PMC9081889.

IFC. 2009. Introduction to Health Impact Assessment. Washington DC, USA: International Finance Corporation.
http://www.ifc.org/wps/wcm/connect/corp_ext_content/ifc_external_corporate_site/home.

Lake L, Shung-King M, Hendricks M, et al. 2019. Prioritising child and adolescent health: a human rights imperative. Child and adolescent health. In: Shung-King M, Lake L, Sanders D, Hendricks M, editors. South African child gauge 2019 chapter: part 2. Cape Town: Children's Institute, University of Cape Town. Cited in Millar et al. (2022).

FINAL

Last JM (Ed), Spasoff RA, Harris SS and Thuriaux MC (Ass Eds). 2000. A Dictionary of Epidemiology. 4th Edition. New York: Oxford University Press.

Millar DA, Kapwata T, Kunene Z, et al. 2022. Respiratory health among adolescents living in the Highveld Air Pollution Priority Area in South Africa. BMC Public Health, 21;22(1):2136. doi: 10.1186/s12889-022-14497-8. PMID: 36411414; PMCID: PMC9677637.

Morales F, Montserrat-de la Paz S, Leon MJ, Rivero-Pino F. 2023. Effects of malnutrition on the immune system and infection and the role of nutritional strategies regarding improvements in children's health status: a literature review. Nutrients, 16:1. https://doi.org/10.3390/nu16010001.

Ostro B. 2004. Assessing the environmental burden of disease at national and local levels. Geneva: World Health Organization.

Sasol South Africa Ltd. 2024. Professional Services Contract: Scope Document for the Water Use License (WUL), Air Emissions License (AEL) Amendment & Full EIA Applications for the Secunda Gas to Power Facility, Secunda Operations, Sasol, Secunda. Revision 1. Secunda: Sasol South Africa Ltd; 2024 Dec 20.

Stats SA. Online. Statistics South Africa, Census 2022, Ward Estimates 2022. Ward by Age Group Estimates. https://superweb.statssa.gov.za/webapi/jsf/dataCatalogueExplorer.xhtml.

Stats SA. 2026. Mortality and causes of death in South Africa: Findings from death notification 2023. Statistical Release P0309.3. Pretoria: Statistics South Africa. https://www.statssa.gov.za/publications/P03093/P030932023.pdf.

USEPA. Online. Air Emissions Factors and Quantification. AP 42, Fifth Edition, Volume I, Chapter 1: External Combustion Sources. https://www.epa.gov/air-emissions-factors-and-quantification/ap-42-fifth-edition-volume-i-chapter-1-external-0.

USEPA. 2022. Environmental Benefits Mapping and Analysis Program – Community Edition User's Manual. January 2022. Updated for BenMAP-CE Version 1.5.8. https://www.epa.gov/benmap/benmap-community-edition.

USEPA. 2019. Integrated Science Assessment for Particulate Matter. Report number EPA/600/R-19/188. December 2019, Contains Errata Sheet created September 2021. Research Triangle Park, NC: US Environmental Protection Agency. www.epa.gov/isa.

WHO. Online. https://www.who.int/classifications/icd/icdonlineversions/en/.

WHO. 2021. WHO global air quality guidelines. Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. Geneva: World Health Organization. Licence: CCBY-NC-SA 3.1IGO.

WHO. 2018. Air pollution and child health: prescribing clean air. Summary. Geneva: World Health Organization (WHO/CED/PHE/18.01). Licence: CC BY-NC-SA 3.0 IGO. https://www.who.int/publications/i/item/air-pollution-and-child-health.

FINAL

WHO. 2016. International Statistical Classification of Diseases and Related Health Problems, 10th edition. Geneva: World Health Organization.

WHO. 2005. WHO Air Quality Guidelines Global Update. Meeting Report. Geneva: World Health Organization.

WSP Group Africa (Pty) Ltd. 2026(a). The Proposed Secunda Gas to Power Facility. Draft Project Description. Prepared for Sasol South Africa Limited. PROJECT NO. ZA0068671.2768. July 2026.

WSP Group Africa (Pty) Ltd. 2026(b). Screening Report for an Environmental Authorization as Required by the 2014 EIA Regulations – Proposed Site Environmental Sensitivity: The Proposed Secunda Gas to Power Facility. Prepared for Sasol South Africa Limited. 2026 Mar 31.

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7 Annexure 1: Assessment of PM2.5 health effects

7.1 Background

Particulate matter ("PM") consists of a mixture of solids and liquid droplets. PM can either be emitted directly into the atmosphere, referred to as primary PM, or pollutants from various sources can react in the atmosphere to form secondary PM.

Inhalable dust consists of airborne particles with an aerodynamic diameter of up to 100 µm, which can enter the nose and upper respiratory tract when breathed in. Unlike respirable dust (which can reach deep into the lungs), inhalable dust tends to accumulate in the nose, throat, and upper airways. PM10 refers to particulate matter, with aerodynamic diameter ≤10 µm. This size range of airborne particulates represents the respirable fraction, which can penetrate deep into the lungs. PM2.5 is the fraction of fine particulate matter, which has an aerodynamic diameter of ≤2.5 µm. This size range is small enough to reach the gas-exchange region (alveoli) of the lungs.

7.2 Causality assessments

The International Agency for Research on Cancer ("IARC") has classified outdoor air pollution, including PM, as a Group 1 carcinogen - carcinogenic to humans (IARC 2016). Studies evaluated in the IARC assessment examined individual PM components and specific PM size fractions. The IARC conclusion was based primarily on epidemiology studies of ambient PM2.5 exposures and lung cancer incidence and mortality, on inhalation studies in mice exposed to ambient air PM10, and on evidence from mechanistic studies using PM of various size fractions.

The USEPA's conclusions regarding short- and long-term exposure to PM2.5 and the key health effects for which causality has been accepted (USEPA 2019) are summarised in Tables 7.2.1 and 7.2.2.

Table 7.2.1: USEPA (2019) causality determination for short-term PM2.5 exposure.

Health effects	Causality determination	Associated health endpoints
Respiratory effects	Likely to be causal	Emergency Department (ED) visits and hospital admissions (HAs) for: • Asthma exacerbation in asthmatic adults and children • Chronic obstructive pulmonary disease (COPD)* • Respiratory infections
Cardiovascular effects	Causal	ED visits and hospital admissions for: • Cardiac conditions • Stroke or thrombo-embolic disease
Mortality	Causal	All-cause: total from all natural causes (excludes accidents, self-harm or homicide)
		Mortality due to the above cardiovascular effects (strong evidence)
		Mortality due to the above respiratory effects (limited evidence)

* Chronic inflammatory lung disease that causes obstructed airflow from the lungs, causing difficulty in breathing. The most common COPDs are chronic bronchitis and emphysema.

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Table 7.2.2: USEPA (2019) causality determination for long-term PM2.5 exposure.

Health effects	Causality determination	Associated health endpoints
Respiratory effects	Likely to be causal	Decrements in lung function
		Asthma incidence and prevalence increase in children
Cardiovascular effects	Causal	Cardiac heart disease or stroke in those with pre-existing disease
Nervous system effects	Likely to be causal	Reduced cognitive function and neurodegeneration in adults (consistent evidence)
		Neurodevelopmental effects in children (limited and inconsistent)
Reproductive effects	Suggestive of, but not sufficient to infer	Male and female reproduction and fertility Pregnancy and birth outcomes
Cancer	Likely to be causal	Lung cancer
Mortality	Causal	Mortality, natural all-cause, all ages
		Cardiovascular disease (consistent)
		Respiratory disease (generally consistent)
		COPD (modest and generally less precise)

With reference to Table 7.2.1, lung cancer (likely to be causal) is included in the health assessment, but not nervous system effects, also likely to be causal. The reason is that concentration-response functions ("CRFs") are available for lung cancer mortality, but not for any particular or collective neurological health endpoints.

The WHO (2021) systematic reviews include GRADE[5] assessments to evaluate the certainty of the overall body of evidence, with a focus on the validity and precision of effect estimates. A summary of the assessments involving PM2.5 is given in Table 7.2.3.

The Review of Evidence on Health Aspects of Air Pollution – REVIHAAP report compiled by the WHO (2013a) also provided evidence for the causality of health effects of exposure to PM2.5. Pairs of health outcomes and causally related air pollutants were identified in the report Health Risks of Air Pollution in Europe – HRAPIE (WHO 2013b) and recommended for inclusion in analyses of air pollution abatement and related prevention of health effects. These are summarised in Table 7.2.4. Meta-analyses with GRADE assessments were not performed in the 2013 assessments; therefore, WHO (2021) assessments are given precedence if effect estimates for pollutant-outcome pairs are provided in both the 2021 and the 2013 assessment reports. However, if effect estimates are not available from the 2021 assessments, estimates from the 2013 assessments are used.

Table 7.2.3: WHO (2021) outcomes involving PM2.5.

Health outcome	GRADE assessment of certainty of evidence	Meta-analysis authors
Short-term exposure – PM2.5, daily mean		
Mortality, natural all-cause	High	Orellano et al. (2020)
Mortality due to: • Cardiovascular causes • Respiratory causes • Cerebrovascular causes (including stroke)	High	Orellano et al. (2020)

[5] Grading of Recommendations Assessment, Development and Evaluation.

Health outcome	GRADE assessment of certainty of evidence	Meta-analysis authors
Long-term exposure – PM2.5, annual mean		
Mortality: all-cause (natural)	High	Chen and Hoek (2020)
Mortality due to: • Cardiovascular (circulatory system) diseases • Ischaemic heart disease • Stroke • Chronic obstructive pulmonary disease (COPD) • Acute lower respiratory infections (ALRI) • Lung cancer	High	Chen and Hoek (2020)
Mortality due to respiratory diseases	Moderate	Chen and Hoek (2020)

The HRAPIE report (WHO 2013b) classified recommended pollutant-outcome pairs, for which causality is accepted, into two categories, groups A and B:

- Group A: pollutant-outcome pairs for which sufficient data are available to enable reliable quantification of effects. In the case of daily mean and long-term exposure, the PM2.5 pairs were all assigned to group A.

- Group B: pollutant-outcome pairs for which there is less certainty about the precision of the data used for quantification of effects. Daily mean and long-term PM2.5 pairs were not assigned to this group.

The PM2.5 group A pair not covered in the WHO 2021 assessment, but which is required for the health risk assessment, is given in Table 12.1.4.

Table 7.2.4: WHO (2013(b)) outcomes involving PM2.5.

Health outcome
Short-term exposure – PM2.5, daily mean
Hospital admissions, cardiovascular diseases (CVDs) (includes stroke), all ages

The purpose of considering both the USEPA and the WHO causality assessments is to operate within the full spectrum of international authoritative and regulatory assessments, which make complementary contributions to the field of calculation of the burden of disease associated with community exposures to ambient are pollution. The assessment terms and conditions of the two authorities were somewhat different because the goal of the USEPA was to support USA-specific burden of disease assessments contributing to cost-benefit calculations. The outcomes of the WHO assessments are also supportive of ambient air quality regulation processes, and to support burden of disease assessments contributing to cost-benefit calculations with application on a wider international geographical scale. Thus, the WHO excluded health outcomes that were considered difficult to monetise, such as low birth weight and lung function (WHO 2013b).

CRFs proposed for short- and long term PM2.5 exposure by the USEPA and WHO are discussed in Section 21.

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7.3 References to Annexure 1

Chen J and Hoek G. 2020. Long-term exposure to PM and all-cause and cause-specific mortality: a systematic review and meta-analysis. Environ Int. 143:105974.
doi: 10.1016/j.envint.2020.105974. License: CC BY-NC-ND. Cited in WHO (2021).

IARC. 2016. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Outdoor air pollution. IARC monographs on the evaluation of carcinogenic risks to humans, volume 109. International Agency for Research on Cancer, Lyon, France: World Health Organization.

Orellano P, Reynoso J, Quaranta N, Bardach A, Ciapponi A. 2020. Short-term exposure to particulate matter (PM10 and PM2.5), nitrogen dioxide (NO_2), and ozone (O3) and all-cause and cause-specific mortality: Systematic review and meta-analysis. Environment International, 142:105876. ISSN 0160-4120.

USEPA. 2019. Integrated Science Assessment for Particulate Matter. Report number EPA/600/R-19/188. December 2019, Contains Errata Sheet created September 2021. Research Triangle Park, NC: US Environmental Protection Agency. www.epa.gov/isa.

WHO. 2021. WHO global air quality guidelines. Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. Geneva: World Health Organization. Licence: CCBY-NC-SA 3.1IGO

WHO. 2013(a). Review of evidence on health aspects of air pollution – REVIHAAP project. Copenhagen, Denmark: World Health Organisation Regional Office for Europe.

WHO. 2013(b). Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide. Copenhagen, Denmark: World Health Organisation Regional Office for Europe.

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8 Annexure 2: Assessments of SO_2 health effects

8.1 Background

Conversion of SO_2 to other sulfur compounds in the atmosphere has been disregarded in the exposure and risk calculations in this document. Any conversions and associated health effects are implicitly included in the epidemiological studies in which health effects of SO_2 were quantified.

8.2 Causality assessments

There is no definitive evidence for an increased cancer potential from SO_2 in humans. According to ATSDR (1998), there are no studies that clearly show carcinogenic effects of sulfur dioxide in humans or animals. The results of studies that investigated workers in the copper smelting and pulp and paper industries were determined to be inconclusive, because the workers were also exposed to arsenic and other chemicals. The USEPA (2017) concluded that the overall evidence for long-term SO_2 exposure and cancer is inadequate to infer a causal relationship. The International Agency for Research on Cancer (IARC) has classified sulfur dioxide as Group 3, not classifiable as to human carcinogenicity (IARC 1992).

The USEPA's conclusions regarding exposure to SO_2 and the key health effects for which causality has been accepted (USEPA 2017) are summarised for short- and long-term exposure, in Tables 8.2.1 and 8.2.2, respectively. Only respiratory effects associated with short-term exposure are causally related to SO_2 exposure. Thus, only short-term SO_2 exposure is included in the HHRA and the only effects of interest are respiratory effects such as asthma exacerbation and respiratory mortality.

Table 8.2.1: USEPA (2017) causality determination for short-term SO_2 exposure.

Health effects	Causality determination	Associated health endpoint
Respiratory effects	Causal	Asthma exacerbation
Cardiovascular effects	Inadequate to infer a causal relationship	Not assessed
Total mortality	Suggestive of, but not sufficient to infer a causal relationship	Not assessed

Table 8.2.2: USEPA (2017) causality determination for long-term SO_2 exposure.

Health effects	Causality determination	Associated health endpoint
Respiratory effects	Suggestive of, but not sufficient to infer a causal relationship	Not assessed
Cardiovascular effects and diabetes	Inadequate to infer a causal relationship	Not assessed
Reproductive and developmental effects:		
Fertility, reproduction, and pregnancy	Inadequate to infer a causal relationship	Not assessed
Birth outcomes	Suggestive of, but not sufficient to infer a causal relationship	Not assessed
Postnatal development	Inadequate to infer a causal relationship	Not assessed
Total mortality	Inadequate to infer a causal relationship	Not assessed
Cancer	Inadequate to infer a causal relationship	Not assessed

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The WHO (2021) systematic reviews include GRADE assessments to evaluate the certainty of the overall body of evidence, with a focus on the validity and precision of effect estimates. A summary of the assessments involving SO_2 is given in Table 8.2.3.

The certainty of the overall body of evidence for asthma exacerbation (ER visits and HAs for asthma) and 24-hour mean and 1-hour maximum SO_2 concentrations, respectively, were graded as moderate. However, only the outcome associated with the 24-hour mean concentration is included in Table 8.2.3, because the 95 per cent confidence interval ("CI") for the 1-hour maximum SO_2 concentration included the null-effect (95 per cent CI: 0.992, 1.014) (WHO 2021).

Table 8.2.3: WHO (2021) outcomes involving SO_2.

Health outcome	GRADE assessment of certainty of evidence	Meta-analysis authors
Short-term exposure – SO_2, daily (24-hours) mean		
Mortality, natural all-cause	High	Orellano et al. (2021)
Respiratory mortality	Moderate	Orellano et al. (2021)
Asthma exacerbation (ER visits and HAs for asthma)	Moderate	Zheng et al. (2021)
Short-term exposure – SO_2, 1-hour max		
Mortality, natural all-cause	Low	Orellano et al. (2021)
Respiratory mortality	High	Orellano et al. (2021)

Concentration-response functions proposed for short term SO_2 exposure by the USEPA and WHO are discussed in Annexure 6.

8.3 References to Annexure 2

ATSDR. 1998. Toxicological Profile for Sulfur Dioxide. Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services.
http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=46.

IARC. 1992. Occupational Exposures to Mists and Vapours from Strong Inorganic Acids; and Other Industrial Chemicals. IARC monographs on the evaluation of carcinogenic risks to humans, volume 54. International Agency for Research on Cancer, Lyon, France: World Health Organization.

Orellano P, Reynoso J, Quaranta N. 2021. Short-term exposure to sulphur dioxide (SO_2) and all-cause and respiratory mortality: A systematic review and meta-analysis. Environment International, 150:106434.

USEPA. 2017. Integrated Science Assessment for Sulfur Oxides – Health Criteria. Report number EPA/600/R-17/451. December 2017. Research Triangle Park, NC: US Environmental Protection Agency. www.epa.gov/isa.

WHO. 2021. WHO global air quality guidelines. Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. Geneva: World Health Organization. Licence: CCBY-NC-SA 3.1IGO.

Zheng X-Y, Orellano P, Lin H-I, Jiang M, Guan W-J. 2021. Short-term exposure to ozone, nitrogen dioxide, and sulphur dioxide and emergency room visits and hospital admissions due to asthma: a systematic review and meta-analysis. Environ Int, 150:106435.
Doi:10.1015.j.envint.2021.106435.

9 Annexure 3: Assessments of NO_2 health effects

9.1 Background

Nitrogen oxides exist as many chemical species in the atmosphere. From the point of view of human health, the air pollutant species of most interest is nitrogen dioxide (WHO 2021). The USEPA reviewed a significant body of epidemiological, human clinical and animal toxicological studies and concluded that there is no definitive evidence for an increased cancer potential from NO_2 in humans. The epidemiological and experimental evidence was inadequate to infer the presence or absence of a causal relationship with cancer risks (USEPA 2016).

The WHO (2021) recommended an AQG level for long-term NO_2 based on all non-accidental mortality and cause-specific, respiratory mortality. The respective relevant RRs are 1.02 and 1.03 per 10 µg/m³ increase in NO_2, as reported in the systematic review of Huangfu and Atkinson (2020). The concentration-response functions ("CRFs") for both health effects are linear at concentrations in the range of 10 to 40 µg/m³. At higher concentrations, the CRF may no longer be linear (WHO 2021).

The WHO also noted that one review (Mills et al. 2016) specifically investigated the sensitivity to adjustment for different PM metrics of the association between short-term NO_2 and mortality. Associations with NO_2 were found to be generally robust. Mills et al. (2016) concluded that evidence was found for associations between NO_2 and adverse health outcomes that are independent of PM mass. However, the authors cautioned that there was limited evidence on adjustment of the NO_2 associations for "*primary combustion particles that are thought to be responsible for the NO_2 associations*". Therefore, Mills et al. (2016) were of the opinion that "*some uncertainty remains regarding possible confounding and health impact assessments should reflect this*".

The WHO (2021) developed an AQG level for short-term (24-hour) NO_2 using the CRFs derived by Orellano et al. (2020) and by Zheng et al. (2021). The concentration-response functions (CRFs) for health effects are linear at concentrations in the range of 25 to 100 µg/m³. At higher concentrations, the CRF may no longer be linear (WHO 2021).

9.2 Causality assessments

The USEPA's conclusions regarding short- and long-term exposure to NO_2 and the key health effects for which causality has been accepted (USEPA 2016) are summarised in Tables 9.2.1 and 9.2.2.

Table 9.2.1: USEPA causality determination for short-term NO_2 exposure.

Health effects	Causality determination	Associated health endpoint
Respiratory effects	Causal	Asthma exacerbation
Cardiovascular effects	Suggestive of, but not sufficient to infer a causal relationship	Not assessed
Total mortality	Suggestive of, but not sufficient to infer a causal relationship	Not assessed

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Table 9.2.2: USEPA causality determination for long-term NO_2 exposure.

Health effects	Causality determination	Associated health endpoint
Respiratory effects	Suggestive of, but not sufficient to infer a causal relationship	Asthma development
Cardiovascular effects and diabetes	Inadequate to infer a causal relationship	Not assessed
Reproductive and developmental effects	Inadequate to infer a causal relationship	Not assessed
Total mortality	Inadequate to infer a causal relationship	Not assessed
Cancer	Inadequate to infer a causal relationship	Not assessed

The WHO (2021) systematic reviews include GRADE assessments to evaluate the certainty of the overall body of evidence, with a focus on the validity and precision of effect estimates. A summary of the assessments involving NO_2 is given in Table 9.2.3.

Table 9.2.3: WHO (2021) outcomes involving NO_2.

Health outcome	GRADE assessment of certainty of evidence	Meta-analysis authors
Short-term exposure – NO_2, daily (24-hours) mean		
Mortality, natural all-cause	High	Orellano et al. (2020)
Asthma exacerbation (ER visits and HAs for asthma)	Moderate	Zheng et al. (2021)
Long-term exposure – NO_2, annual mean		
Mortality, natural all-cause	Moderate	Huangfu and Atkinson (2020)
Respiratory mortality, not differentiated according to respiratory cause	Moderate	Huangfu and Atkinson (2020)
Respiratory mortality as COPD mortality, including for asthma	High	Huangfu and Atkinson (2020)

Summary of short-term exposure to NO_2

In summary, the USEPA (2016) and the WHO (2021) are not in agreement that conclusive evidence exists of a causal relationship between short-term exposure to NO_2 and all-cause mortality. The USEPA concluded that evidence from the literature was suggestive of, but not sufficient to infer a causal relationship, while the meta-analysis by Orellano et al. (2020), commissioned by the WHO, concluded that the certainty of evidence for an effect justified a grading of “high”.

The USEPA (2016) and the WHO (2021) are in fair agreement regarding short-term exposure to NO_2 and an effect on asthma exacerbation, which is viewed as causal by the USEPA and as associated with a moderate degree of certainty of evidence by the WHO, based on the meta-analysis by Zheng et al. (2021). The WHO subsequently used the CRFs derived by Orellano et al. (2020) and by Zheng et al. (2021) in the development of the short-term (24-hour) AQG level.

Summary of long-term exposure to NO_2

The USEPA (2016) and the WHO (2021) are not in agreement regarding causality/certainty of evidence of long-term exposure to NO_2 and all-cause (or respiratory) mortality. The USEPA concluded that evidence from the literature was inadequate to infer a causal relationship, while the meta-analysis commissioned by the WHO (Orellano et al. 2020) graded the certainty of evidence for an effect as moderate to high. However, the two agencies are in some agreement that asthma appears to be an important effect associated with long-term NO_2 exposure. The USEPA assigned a rating of higher rank (although still only “suggestive, but not sufficient”) to causality of an asthmatic

diagnosis and the WHO the highest degree of certainty of association to respiratory mortality as COPD, including asthma.

The WHO subsequently based the long-term (annual average) AQC level on the systematic review of Huangfu and Atkinson (2020).

CRFs for health effects of NO_2

Concentration-response functions proposed for short- and long term NO_2 exposure by the USEPA and WHO are discussed in Annexure 5.

9.3 References to Annexure 3

Huangfu P, Atkinson R. 2020. Long-term exposure to NO_2 and O_3 and all-cause and respiratory mortality: A systematic review and meta-analysis. Environ Int, 144:105998.

Mills IC, Atkinson RW, Anderson HR, Maynard RL, Strachan DP. Distinguishing the associations between daily mortality and hospital admissions and nitrogen dioxide from those of particulate matter: a systematic review and meta-analysis. BMJ Open. 2016 Jul 21;6(7):e010751. doi: 10.1136/bmjopen-2015-010751. PMID: 27443553; PMCID: PMC4964176.

Orellano P, Reynoso J, Quaranta N, Bardach A, Ciapponi A. 2020. Short-term exposure to particulate matter (PM10 and PM2.5), nitrogen dioxide (NO_2), and ozone (O_3) and all-cause and cause-specific mortality: Systematic review and meta-analysis. Environment International, 142:105876. ISSN 0160-4120.

USEPA. 2016. Integrated Science Assessment for Oxides of Nitrogen – Health Criteria. Report number EPA/600/R-15/068. January 2016. Research Triangle Park, NC: US Environmental Protection Agency. w9ww.epa.gov/isa.

WHO. 2021. WHO global air quality guidelines. Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. Geneva: World Health Organization. Licence: CCBY-NC-SA 3.1IGO.

Zheng X-Y, Orellano P, Lin H-I, Jiang M, Guan W-J. 2021. Short-term exposure to ozone, nitrogen dioxide, and sulphur dioxide and emergency room visits and hospital admissions due to asthma: a systematic review and meta-analysis. Environ Int, 150:106435. Doi:10.1015.j.envint.2021.106435.

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10 Annexure 4: Assessments of CO health effects

10.1 Background

Carbon monoxide (CO) is a colourless, non-irritant, odourless and tasteless toxic gas. It is produced by the incomplete combustion of carbonaceous fuels. Acute toxicity by exposure to high concentrations of exogenous CO is due to a reaction with haemoglobin to form carboxyhaemoglobin (COHb) (WHO 2021). Low-level exposures to CO, relevant to ambient exposure, target the heart and cardiovascular system, the respiratory system, the central nervous system, and the foetus and neonate (ATSDR 2012).

The US Agency for Toxic Substances and Disease Registry ("ATSDR") compiled a toxicological profile for CO (ATSDR 2012) and concluded that growing evidence had revealed that endogenously produced CO is a cell signalling agent. As a signalling agent it contributes to the regulation of numerous physiological systems, including brain and muscle oxygen storage and utilisation, relaxation of vascular and extra-vascular smooth muscle, modulation of synaptic neurotransmission, anti-inflammatory and anti-thrombosis mechanisms. Endogenous CO is produced from the catabolism of haem and other endogenous precursors and is not associated with toxicity (ATSDR 2012).

There is not any indication of a potential association between CO exposure and cancer in humans. The USEPA reviewed the epidemiological and experimental literature regarding CO exposure and found that a potential positive association was not indicated in any of the reviewed documents (USEPA 2010).

The WHO (2021) developed the AQG level for short-term, 24-hour mean carbon monoxide concentrations based on the association of CO with myocardial infarction hospital admissions and mortality. Supporting epidemiological evidence was obtained from the meta-analysis by Lee et al. (2020).

10.2 Causality assessment

10.2.1 USEPA

Short-term exposure

The USEPA (2010) concluded that epidemiologic studies of short-term exposure to CO and mortality provide suggestive evidence of a causal relationship.

Associations with respiratory morbidity, observed in epidemiologic studies, are supported by animal toxicology studies indicating potential underlying biological mechanisms. The combined evidence suggests a causal relationship (USEPA 2010).

Controlled short-term human exposure studies on neural and behavioural effects showed inconsistent results, but toxicological studies in rodents indicated that perinatal exposure to CO can have a range of effects on the adult nervous system. The combined evidence was viewed as suggestive of a causal relationship between both short- and long-term CO exposure and CNS effects (USEPA 2010).

Compared to CNS effects, more convincing evidence is available of an impact on the cardiovascular system. The USEPA reviewed a significant body of epidemiological, human clinical and animal

toxicology studies and concluded that sufficient evidence was provided to infer a likely causal relationship for cardiovascular effects with short-term exposure to CO. A series of controlled human exposure studies among individuals with coronary artery disease was emphasised as the most compelling evidence of a CO-induced effect on the cardiovascular system at levels relevant to the current US National Ambient Air Quality Standards (USEPA 1991 and USEPA 2000). The studies showed that the time needed to elicit a negative cardiovascular response to exercise while exposed to CO was consistently shortened when increasing concentrations of CO were inhaled (USEPA 2010).

The USEPA (2010) reviewed epidemiologic studies reporting associations with ED visits and HAs for ischemic heart disease (IHD)[6], for congestive heart failure (CHF)[7] and for cardiovascular diseases (CVD)[8] as a group. Consistent associations with HAs and ED visits for myocardial infarction (MI)[9] and angina[10] were found, but not with stroke. It was concluded that consistent and coherent evidence from epidemiologic and human clinical studies, along with biological plausibility provided by the role of CO in limiting O_2 availability, is sufficient to conclude that a causal relationship is likely to exist between short-term CO exposures and cardiovascular morbidity.

Long-term exposure

The USEPA (2010) identified only two epidemiologic studies that investigated the relationship between long-term exposure to CO and cardiovascular effects, which provided very limited evidence of an association. Considering the lack of evidence from controlled human exposure studies and the very limited evidence from toxicological studies, the available evidence was inadequate to infer a causal relationship. The evidence on respiratory morbidity was also inadequate.

Epidemiologic studies of CO exposure during pregnancy (long-term exposure) provided some evidence of an association with detrimental birth outcomes including increased risk of preterm birth, cardiac birth defects, small reductions in birth weight, and infant mortality in the post-neonatal period. Toxicological studies in rodents also indicated that perinatal exposure to CO can have a range of effects on the adult nervous system. The epidemiological and toxicological studies provide evidence suggestive of a causal relationship between long-term exposure to CO and birth and developmental effects (USEPA 2010).

Epidemiologic studies of long-term exposure to CO and mortality reported consistent null associations. The lack of respiratory and cardiovascular morbidity and the absence of a proposed biological mechanism for mortality further indicate that a causal relationship with long-term exposure is unlikely (USEPA 2010).

The USEPA's conclusions regarding short- and long-term exposure to CO and the key health effects for which causality has been accepted (USEPA 2010) are summarised in Tables 10.2.1.1 and 10.2.1.2.

[6] Heart problems caused by narrowed heart arteries, which result in decreased blood flow and oxygen to the heart muscle.
[7] Failure of the heart to pump blood with sufficient force from the heart to the body. As blood flow out of the heart slows, blood returning to the heart through the veins backs up, causing congestion in the body's tissues. Often swelling (oedema) results. Most often there's swelling in the legs and ankles, but it can happen in other parts of the body.
[8] Disease of the heart and/or blood vessels, causing decreased blood flow to the heart, brain or body.
[9] Commonly known as a heart attack, an MI occurs when blood flow stops to a part of the heart, causing damage to the heart muscle.
[10] Chest pain or discomfort caused by an inadequate supply of oxygen-rich blood to the heart muscle.

Table 10.2.1.1: USEPA causality determination for short-term CO exposure.

Health effects	Causality determination	Associated health endpoint
Total mortality	Suggestive of a causal relationship	Total non-accidental mortality
Respiratory effects	Suggestive of a causal relationship	Pulmonary function, medication use, respiratory symptoms, HAs and ED visits
Central nervous system (CNS)	Suggestive of a causal relationship	Behavioural and other effects
Cardiovascular	Likely to be causal	HAs and ED visits for myocardial infarction and angina, but not with stroke.

Table 10.2.1.2: USEPA causality determination for long-term CO exposure.

Health effects	Causality determination	Associated health endpoint
Cardiovascular effects	Inadequate to infer a causal relationship	Not assessed in detail
Respiratory effects	Inadequate to infer a causal relationship	Not assessed in detail
Reproductive and developmental effects	Suggestive of a causal relationship	Preterm birth, cardiac birth defects, small reductions in birth weight, infant mortality
Total mortality	Unlikely	Consistent null effect
Central nervous system (CNS)	Suggestive of a causal relationship	Behavioural and other effects

10.2.2 WHO

The meta-analysis by Lee et al. (2020) included an assessment of the certainty of the evidence of an association between short-term CO exposure and emergency department visits or hospital admissions and mortality due to myocardial infarction. The certainty of evidence, based on the GRADE system, was considered moderate (Table 10.2.2.1).

The systematic review by Lee et al. (2020) focused on only myocardial infarction (MI, ICD-10 codes I21– I22) as an outcome of IHD, because it is not possible to establish the precise time of onset of other IHD outcomes. Furthermore, the WHO concluded that other conditions within the spectrum of IHD are routinely managed in outpatient settings rather than in the emergency hospital setting, or in hospital wards (WHO 2021).

The WHO did not commission a meta-analysis study of long-term CO exposure, likely motivated by several factors, including the negative outcomes of the US EPA causality assessments and a general acceptance that only short-term CO exposure is important to the establishment of ambient air quality guidelines.

Table 10.2.2.1: WHO (2021) outcomes involving CO.

Health outcome	GRADE assessment of certainty of evidence	Meta-analysis authors
Short-term exposure – CO, daily (24-hours) mean		
EM visits or HAs and mortality due to myocardial infarction (MI)	Moderate	Lee et al. (2020)
Long-term exposure – CO, annual mean		
Not assessed		

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10.2.3 Risk factors for health risk calculations

The ATSDR (2012) has concluded that, although there may be a level of carbon monoxide exposure that can be tolerated with minimal risk of adverse effects, the currently available toxicological and epidemiological data do not identify such minimal risk levels. Lowest-observed-adverse-effect levels (LOAELs) are reported, but the studies in which LOAELS are identified fail to identify no-observed-adverse-effect levels (NOAELs). Therefore, health risk assessment of exposure to ambient CO follows the relative risk approach, and not the reference dose approach.

Concentration-response functions proposed for short- and long-term CO exposure by the USEPA and WHO are discussed in Annexure 6.

10.3 References to Annexure 4

ATSDR. 2012. Toxicological Profile for Carbon Monoxide. Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services. http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=191&tid=34.

Lee KK, Spath N, Miller MR, Mills NL, Shah ASV. 2020. Short-term exposure to carbon monoxide and myocardial infarction: A systematic review and meta-analysis. Environ Int. 2020, 143:105901. doi: 10.1016/j.envint.2020.105901.

USEPA. 2010. Integrated science assessment for carbon monoxide. EPA/600/R-09/019F. Research Triangle Park, NC; US Environmental Protection.

USEPA. 2000. Air quality criteria for carbon monoxide. National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency. Research Triangle Park, NC. EPA 600/P-99/001F. 000907.

USEPA. 1991. Air quality criteria for carbon monoxide. US Environmental Protection Agency. Research Triangle Park, NC. EPA/600/8-90/045F. http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=3000554R.txt.

WHO. 2021. WHO global air quality guidelines. Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. Geneva: World Health Organization. Licence: CCBY-NC-SA 3.1IGO.

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11 Annexure 5: Concentration-response functions and β-values for criteria pollutant health effect quantification

11.1 Background

Concentration-response functions (CRFs) yield numerical effect predictor values, namely, RRs and β-values, used for health effect quantification, discussed in Section 4.

Both the USEPA and the WHO have paid considerable attention to the shape of the CRF curve. The shapes and the β-values used in the USEPA software tool *Benefits Mapping and Analysis Program - Community Edition* (BenMAP-CE) (USEPA 2021), which is freely downloadable from the USEPA website, and available from the BenMAP-CE user's manual (USEPA 2022) are presented in the various tables below.

The WHO/Europe's software tool AirQ+, which enables quantification of the health effects of exposure to air pollution (WHO 2019), the WHO (2021) guidelines report and the WHO (2013) HRAPIE report, provides similar information, recorded in the various tables.

CRFs developed by the USEPA and the WHO are both considered, and the choice of CRF to be used is motivated for each pollutant-outcome pair in the relevant tables.

In order to avoid double-counting of the mortality effect, only all-cause natural mortality (not accidental, not homicidal, not self-harm), will be calculated by INFOTOX for the purposes of the CBA, not the cause-specific mortalities. That is, cardiovascular, respiratory and lung cancer mortality cases are not also quantified, since these are already included in the all-cause mortality cases.

However, cardiovascular HAs, asthma symptoms, acute and chronic bronchitis and lung cancer incidence (number of new cases diagnosed per year) are quantified.

11.2 International CRFs

11.2.1 Background

This section presents CRFs derived by the USEPA and the WHO. Short-term PM2.5, CO, SO_2 and NO_2 β-values are summarised in Table 11.2.4.1 and long-term PM2.5 shapes and values in Table 11.2.4.2. If only RRs are provided in the original source, the β-values are calculated with Equation 4.3. Adjustment of the effect estimates (RRs or β-values) for the effects of co-exposure to other pollutants is also noted.

CRFs for specific pollutant-outcome pairs are often available from both agencies. The most suitable CRF for a specific pair, for the purposes of the risk assessment, is indicated in such cases with a brief motivation for the choice.

11.2.2 Notes on CRFs for short-term exposure to CO

The USEPA did not propose CRFs for CO in the BenMAP user's manual (USEPA 2022) or in the BenMAP software tool (USEPA 2021). However, the USEPA is in agreement with the WHO (2021) that short-term CO is the more important exposure measure, and not long-term exposure. The

USEPA concluded that short-term CO is likely to be causally related to HAs and ED visits for MI and angina, but not for stroke. This is in agreement with the final conclusions of the WHO, focusing on MI HAs and ED visits as the outcome measures on which the AQC for CO are based. The position of the WHO is discussed in detail in the following paragraphs.

The WHO used the risk coefficients from the systematic review by Lee et al. (2020), assuming a linear CRF, to calculate the increase in MI hospital and emergency room admissions and mortality relative to a daily mean carbon monoxide concentration of 0 mg/m^3. The Lee et al. (2020) review showed that the magnitude of the RR estimate was highly dependent on inclusion of three partly overlapping studies from East Asia conducted in low CO, high NO_2 and high PM atmospheres (Hsieh et al. 2010, Cheng et al. 2009 and Tsai et al. 2012). Excluding these studies produced an RR of 1.016 (95% CI: 1.009–1.023) from the remaining studies.

Amongst the remaining studies, there were only three effect estimates for MI mortality, none of which suggested an effect from CO. Therefore, these studies were also excluded. The remaining studies produced an effect estimate (RR) for MI admissions of 1.015 (95% CI: 1.007–1.024).

The effects on MI admissions were mostly seen in studies with higher CO levels, with a median CO level exceeding 1.15 mg/m^3. In this group the RR was 1.019 (95% CI: 1.011−1.027). In the remaining studies with lower CO levels, the RR was 1.00 (95% CI: 0.998−1.003).

For guideline development, the GDG of the WHO considered as more relevant the RR of 1.019, observed in studies with a median CO of more than 1.15 mg/m^3, because it excludes obvious outliers, is focused on one outcome (MI admissions), rather than two (admissions plus mortality) and is restricted to the concentration range (>1.15 mg/m^3) over which effects were actually demonstrated.

The implications for health effect quantification of the cardiovascular effect of short-term exposure to CO is:

- Only MI admissions are quantified
- The relevant RR is 1.019
- The quantification should only be attempted in the ambient concentration range higher than 1.15 mg/m^3, over which effects were actually demonstrated.

11.2.3 Notes on CRFs for exposure to NO_2

The USEPA did not propose CRFs for NO_2 in the BenMAP user's manual (USEPA 2022) or in the BenMAP software tool (USEPA 2021).

In terms of short-term exposure to NO_2, the USEPA (2016) and the WHO (2021) are in fair agreement regarding short-term exposure to NO_2 and an effect on asthma exacerbation, but not regarding an effect on all-cause mortality (Section 9.2). Therefore, only asthma exacerbation is assessed, using the CRF supported by the WHO (Table 11.2.4.1).

The two agencies are in some agreement that asthma appears to be an important effect associated with long-term NO_2 exposure, but are not in agreement regarding all-cause (or respiratory) mortality. Therefore, only respiratory mortality as COPD, including asthma, is assessed, using the CRF proposed by the WHO (Table 11.2.4.1)

11.2.4 Summary of exposure durations, health effects and RRs

Summaries of the selected CRFs, based on the reviews presented in Annexures 1 to 4, are presented in Tables 11.2.4.1 and 11.2.4.2.

Table 11.2.4.1: Short-term (24-hour mean) PM2.5, SO_2 and NO_2 CRF shapes, RRs and β-values.

Identified outcome	Pollutant	CRF shape	RR	Adjustment for co-pollutants?	β-value	Source
Cardiovascular HAs						
Cardiovascular (including stroke) HAs	PM2.5	Log-linear	1.0091 per 10 µg/m^3	Not stated in source document	0.00091	WHO (2013)
Cardiovascular HAs	PM2.5	A summary measure (pooled RRs or β-values) from a meta-analysis is not available. Multiple β-values are provided for various US cities.				USEPA (2021)
Choice of CRF: Cardiovascular (including stroke) HAs	PM2.5	The WHO (2013) pooled RR is used. This avoids the generation of multiple results, with a wider range of uncertainty, if the multiple β-values provided by the USEPA were to be used.				
Asthma exacerbation						
Asthma exacerbation as asthma HAs and ER visits	SO_2	Assumed linear by the authors of the meta-analysis (Zheng et al. 2021).	1.010 per 10 µg/m^3	Associations observed in single-pollutant models were no longer significant after the inclusion of a second pollutant. The authors of the meta-analysis (Zheng et al. 2021) concluded that the loss of significance warrants further investigation of the causal association with SO_2. However, the association is accepted until further investigations prove otherwise.	0.0010	WHO (2021)
Asthma exacerbation as cough, shortness of breath and wheeze, respectively	PM2.5	A summary measure (pooled RRs or β-values) from a meta-analysis is not available. Separate β-values are provided for three symptoms of asthma exacerbation (cough, shortness of breath and wheeze, respectively) for 2 US cities.				USEPA (2021)
Choice of CRF between PM2.5 and SO_2: Asthma exacerbation as asthma HAs and ER visits	SO_2	The WHO (2021) pooled RR is used: • This provides a single measure of asthma exacerbation, instead of quantification of multiple symptoms, if the multiple β-values provided by the USEPA were to be used. • SO_2 is rated as causal of asthma exacerbation by the USEPA (Table 8.2.1), while PM2.5 has been rated as likely to be causal (Table 7.2.1). The causality of SO_2 on asthma exacerbation is thus rated as more certain than the causality of PM2.5; therefore, SO_2 is the preferred causative exposure to be assessed.				

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Identified outcome	Pollutant	CRF shape	RR	Adjustment for co-pollutants?	β-value	Source
Asthma exacerbation as asthma HAs and ER visits	NO_2	Assumed linear by the authors of the meta-analysis (Zheng et al. 2021).	1.014 per 10 µg/m^3	Associations observed in single-pollutant models were no longer significant after the inclusion of a second pollutant. The authors of the meta-analysis (Zheng et al. 2021) concluded that the loss of significance warrants further investigation of the causal association with NO_2. However, the association is accepted until further investigations prove otherwise.	0.0014	WHO (2021)
All-cause (natural) mortality						
All-cause (natural) mortality, all ages	PM2.5	Linear	1.0065 per 10 µg/m^3	Yes, significance of associations remains	0.00065	WHO (2021)
All-cause (natural) mortality, all ages	PM2.5	Log-linear	β-value is provided	No, single-pollutant model	0.00033	Baxter et al. (2017) cited in USEPA (2021) and (2022)
All-cause (natural) mortality, all ages	SO_2	Linear CRF with no threshold	1.0059 per 10 µg/m^3	Yes, significance of associations remains	0.00059	WHO (2021)
Choice of CRF: All-cause (natural) mortality (all ages)	The validity of including SO_2 in the all-cause mortality quantification is uncertain, because of the divergence in the causality assessments of the USEPA and the WHO. The USEPA has assessed the strength of causality as *suggestive of, but not sufficient to infer a causal relationship* (Table 7.2.1), while the WHO (2021) has assessed the certainty of evidence for an effect as *high* (Table 7.2.3). The issue is solved by assessing total mortality based on long-term PM2.5 concentrations (Table 11.2.4.2). Both the USEPA (Table 7.2.1) and the WHO (Table 7.2.3) are in agreement on the causality and certainty of evidence, respectively. Regarding assessment of long-term vs. short-term PM2.5 to obtain the burden of disease for the purpose of a CBA, the WHO (2013) indicates a preference for the assessment of long-term exposure (Table 1 in the HRAPIE document recommending CRFs for cost–benefit analysis of particulate matter, WHO (2013)). Assessment of total mortality based on long-term PM2.5 only (not short-term PM2.5 as well) also avoids double-counting of cases.					
Choice of CRF: Work Loss Days, age 18-64	PM2.5	The CRFs available from the USEPA and the WHO are practically identical. The USEPA value is derived from Ostro (1987). The source of the AirQ+ software is not specified in the software manual. The slightly more conservative value of 0.0046 (USEPA 2021 and 2022) is used in the risk assessment.				

HA: Hospital admissions
ER: Emergency room

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Table 11.2.4.2: Long-term (annual mean) PM2.5 and NO_2 CRF shapes, RRs and β-values.

Identified outcome(s)	Pollutant	CRF shape	RR	Adjustment for co-pollutants?	β-value	Source
Acute bronchitis						
Acute bronchitis	PM2.5	Not assessed				WHO 2013 and 2021
Acute bronchitis symptoms, ages 8 to 12 (questionnaire-based)	PM2.5	Logistic (S-shaped curve) derived by Dockery et al. (1996). USEPA (2021) provides a β-value derived from Dockery's curve. It is assumed that the β-value is applicable to the linear portion of the S-shaped curve.	β-value derived by USEPA (2021) is used	No, single-pollutant model	0.0272	USEPA (2021 & 2022) from Dockery et al. (1996)
Choice of CRF: Acute bronchitis symptoms, ages 8 to 12	PM2.5	The USEPA (2021 and 2022) β-value derived from Dockery et al (1996) is used.				
Chronic bronchitis incidence						
Chronic bronchitis incidence	PM2.5	Not assessed				WHO 2013 and 2021
Chronic bronchitis incidence in age group 27+	PM2.5	Logistic (S-shaped curve) derived by Abbey et al. (1995). USEPA (2021) provides a β-value derived from Abbey's curve. It is assumed that the β-value is applicable to the linear portion of the S-shaped curve.	β-value derived by USEPA (2021) is used	"Surrogate relationships" with other pollutants were considered, and it was concluded that the relationship between PM2.5 and chronic bronchitis was not due to such a "surrogate relationship".	0.0137	USEPA (2021 & 2022) from Abbey et al. (1995)
Choice of CRF: Chronic bronchitis incidence, ages 27+	PM2.5	The USEPA (2021 and 2022) β-value derived from Abbey et al (1995) is used. It should be noted that the study group was Seventh-Day Adventists (SDAs), non-smokers who had resided since 1966 in the vicinity of nine airports throughout California (n = 1 868). Thus, the potential for accurate extrapolation of this study results to South African populations is limited.				
All-cause (natural) mortality, all ages						
All-cause (natural) mortality, all ages	PM2.5	Linear	1.08 per 10 µg/m^3	Yes, pooled RR after adjustment is presented	0.0077	WHO (2021)
All-cause (natural) mortality, all ages	PM2.5	Not available for all-ages, only for incremental age groups				USEPA (2021 and 2022)

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Identified outcome(s)	Pollutant	CRF shape	RR	Adjustment for co-pollutants?	β-value	Source
Choice of CRF: All-cause (natural) mortality, all ages	PM2.5	The CRF derived by the WHO (2021) is used, since: • it is a pooled value from a meta-analysis • it is not restricted to specific geographic areas • it is adjusted for confounding effects of co-pollutants • it is not limited to a specific age group				
Choice of CRF: Lung cancer: diagnosis as a chronic disease (incidence) (ages 30+)	PM2.5	The study by Gharibvand et al. (2017) is used to estimate the incidence of persons suffering from lung cancer.				
Lung cancer incidence						
Lung cancer as chronic disease sufferers (ages 30+)	PM2.5	Log-linear	β-value is provided	Yes, consistent association with PM2.5	0.0378	Gharibvand et al. (2017) cited in USEPA (2021 and 2022)
COPD mortality, all ages						
Respiratory mortality as COPD mortality, including for asthma	NO_2	Linear (Huangfu and Atkinson 2020)	1.03 per 10 µg/m^3	Associations between NO_2 and mortality were attenuated upon adjustment for co-pollutants in some, but not all studies (Huangfu and Atkinson 2020)	0.0030	WHO (2021)

HA: Hospital admissions.
ER: Emergency room.

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11.3 References to Annexure 5

Abbey DE, Ostro BE, Petersen F and Burchette RJ. 1995. Chronic Respiratory Symptoms Associated with Estimated Long-Term Ambient Concentrations of Fine Particulates Less Than 2.5 Microns in Aerodynamic Diameter (PM2.5) and Other Air Pollutants. J Expo Anal Environ Epidemiol, 5(2):137-159. Cited in USEPA (2021 and 2022).

Baxter LK, Crooks JL and Sacks JD. 2017. Influence of exposure differences on city-to-city heterogeneity in PM2.5-mortality associations in US cities. Environ Health 16:1. Cited in USEPA (2021 and 2022).

Dockery DW, Cunningham J, Damokosh AI, et al. 1996. Health Effects of Acid Aerosols on North American Children - Respiratory Symptoms. Environmental Health Perspectives, 104(5):500-505. Cited in USEPA (2021 and 2022).

Gharibvand L, Shavlik D, Ghamsary M, et al. 2017. The association between ambient fine particulate air pollution and lung cancer incidence: results from the AHSMOG-2 study. Environ Health Perspect 125 (3): 378–384. Cited in USEPA (2021 and 2022).

Huangfu P, Atkinson R (2020). Long-term exposure to NO_2 and O3 and all-cause and respiratory mortality: a systematic review and meta-analysis. Environ Int., 144:105998. doi: 10.1016/j.envint.2020.105998. License: CC BY-NC-ND.

Lee KK, Spath N, Miller MR, Mills NL, Shah ASV. 2020. Short-term exposure to carbon monoxide and myocardial infarction: a systematic review and meta-analysis. Environ Int., 143:105901. doi: 10.1016/j.envint.2020.105901. License: CC BY-NC-ND.

Ostro BD. 1987. Air Pollution and Morbidity Revisited: A Specification Test. Journal of Environmental Economics and Management, 14:87-98. Cited in USEPA (2021 and 2022).

Tsai SS, Chen PS, Yang YH, et al. 2012. Air pollution and hospital admissions for myocardial infarction: are there potentially sensitive groups? J Toxicol Environ Health Part A 75 (4), 242–251. https://doi.org/10.1080/15287394.2012.641202. [published Online First: 2012/02/23].

USEPA. 2022. Environmental Benefits Mapping and Analysis Program – Community Edition User's Manual. January 2022. Updated for BenMAP-CE Version 1.5.8. https://www.epa.gov/benmap/benmap-community-edition.

USEPA. 2021. Benefits Mapping and Analysis Program (BenMAP) Community Edition 1.5. US Environmental Protection Agency. https://www.epa.gov/benmap/benmap-community-edition.

USEPA. 2016. Integrated Science Assessment for Oxides of Nitrogen – Health Criteria. Report number EPA/600/R-15/068. January 2016. Research Triangle Park, NC: US Environmental Protection Agency. w9ww.epa.gov/isa.

WHO. 2021. WHO global air quality guidelines. Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. Geneva: World Health Organization. Licence: CCBY-NC-SA 3.1IGO.

FINAL

WHO. 2019. AirQ+: software tool for health risk assessment of air pollution. Bonn (Germany): World Health Organization Regional Office for Europe, European Centre for Environment and Health. http://www.euro.who.int/en/health-topics/environment-and-health/air-qualityactivities/airq-software-tool-for-health-risk-assessment-of-air-pollution.

WHO. 2013. Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide. Copenhagen, Denmark: World Health Organisation Regional Office for Europe.

Zheng X-Y, Orellano P, Lin H-I, Jiang M, Guan W-J. 2021. Short-term exposure to ozone, nitrogen dioxide, and sulphur dioxide and emergency room visits and hospital admissions due to asthma: a systematic review and meta-analysis. Environ Int, 150:106435. Doi:10.1015.j.envint.2021.106435. Cited in WHO (2021).

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