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Retrieval and scientific interpretation of ecotoxicological information

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Project conducted on behalf of Savannah Environmental (Pty) Ltd

Human Health Risk Assessment for the Phakwe Richards Bay Gas Power 3 Combined Cycle Power Plant in Richards Bay

Report No 035-2022 Rev 1.0

Compiled by

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6 May 2022

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6 May 2022

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 (Pr Sci Nat, Environmental Science, No 400284/04). Dr Van Niekerk has specialised in chemical toxicology and human health risk assessment, but he has experience in many other areas in the disciplines of analytical and environmental sciences.

Dr Marlene Fourie has BSc and Hons BSc degrees from the University of Stellenbosch and MSc and PhD degrees from the University of Pretoria. Her field of specialisation is reproductive biology/toxicology. Dr Fourie also has an MSc-degree in epidemiology from the University of Pretoria. Following positions as Medical Natural Scientist at the Andrology Unit, Department of Urology, University of Pretoria and the Pretoria Academic Hospital from 1987 to 2001, she joined INFOTOX as a Medical Biological Scientist. Dr Fourie has conducted many health risk assessments and projects relating to the health status of communities. She is registered as a Professional Natural Scientist (Pr Sci Nat, Toxicological Science, No 400190/14).

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

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

Willem Christiaan Abraham van Niekerk

6 May 2022

Executive Summary

Savannah Environmental (Pty) Ltd appointed INFOTOX (Pty) Ltd ("INFOTOX") to conduct a rapid appraisal health impact assessment (RAHIA) for the development of the Phakwe Richards Bay Gas Power 3 combined cycle power plant and related infrastructure located in Alton North, Richards Bay, within the uMhlathuze Local Municipality (LM) in the uThungulu District Municipality (DM), KwaZulu-Natal. This document presents the human health risk assessment (HHRA) for the RAHIA. The results of the HHRA feed into the RAHIA, presented in a separate report, 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 main focus of the HHRA is the health risks in surrounding receptor communities due to the dispersion of substances emitted by the proposed power plant operations (the source of exposure) into air (the pathway of exposure).

The assessment of exposure in the residential areas to the likely airborne emissions from the project site is vital for the purposes of the HHRA. The impact of such emissions on air quality has been determined by an air dispersion modelling specialist of Airshed Planning Professionals (Pty) Ltd ("Airshed"). The ambient air contaminants of concern in the operational phase of the plant are four criteria pollutants, namely, the PM2.5 fraction of airborne inorganic particulate matter, sulfur dioxide (SO_2), nitrogen oxides as NO_2, and carbon monoxide (CO). VOCs (as an unspecified group of substances) associated with the gas turbine operations are not regulated and health risk values for assessment of the unspecified group are not available. Thus, the generally accepted approach is to model and assess benzene as a surrogate chemical substance representing the group of VOCs.

Relative risk (RR) ratios are the health risk ratios most often used in large epidemiological studies of the effects of criteria air pollutants on the health of receptor populations. The criteria pollutant HHRA is approached through the calculation of attributable fractions of disease (AFs) based on the incremental change in the air concentration of the pollutant of interest, using the RRs associated with exposure to a specific pollutant over the short- or long term. Only those health outcomes that are shown to be causally related to exposure to the criteria pollutants are included in the assessments. INFOTOX follows the methodology adopted by the World Health Organization (WHO), the US Environmental Protection Agency (USEPA) and international HHRA experts.

The AFs are interpreted as the fraction of the risk of a specific health effect, e.g., asthma exacerbation, experienced by the receptor population, that can reasonably be attributed to exposure to the assessed criteria pollutant originating from the investigated source, at the ambient air concentration modelled by the air dispersion specialist. Thus, an AF of 1 per cent is interpreted as an indication that 1 per cent of the total risk of a specific health effect in an exposed person can be reasonably attributed to exposure to the pollutant of interest, at the modelled concentration. The balance of the risk, e.g., 99 per cent, is attributable to other known or unknown factors, and/or to background levels of exposure to the criteria pollutant of interest, and/or to factors specific to the exposed person, that are not related to the investigated source of the criteria pollutant of interest. In this report, the investigated source is the Phakwe Richards Bay Gas Power 3 Combined Cycle Gas to Power Plant. The assessed exposure concentrations are those modelled by the air dispersion specialist for a scenario of normal operations at the plant.

All of the AFs calculated for the criteria pollutants are in the range less than 10 per cent. The lowest AFs, all lower than 0.1 per cent, are calculated for exposure to PM2.5, whether over the short- or long-term periods of exposure to modelled concentrations. Such AFs are for all practical purposes not significant and in the negligible range. The reported AFs cannot be interpreted as

indicating any reason for concern with regard to human health risks associated with the slightly increased ambient air PM2.5 concentrations as a consequence of the proposed operations of the Phakwe power plant.

The single highest AF calculated for asthma exacerbation associated with short-term exposure to SO_2 is 8.4 per cent. The calculated AFs are centred around a middle value (the median) of approximately 1 per cent. Considering the entire set of SO_2 risk results, there is insufficient cause to conclude that the risk to health due to short-term exposure to SO_2 associated with the proposed power plant is significantly higher than the background risk to health.

The highest AFs calculated for asthma exacerbation leading to an asthma-related emergency care visit or hospitalisation associated with short-term exposure to NO_2 are approximately 9 per cent, the reported upper range limit. Most calculated AFs are centred around a middle value (the median) of approximately 3 per cent. The calculated AFs are in the range viewed as very low to negligible and cannot be interpreted as indicating a significant or serious risk to health.

Regarding CO exposure associated with operations at the proposed power station, there are no residences within the impact area delineated by the results of air dispersion modelling. The impacted area is mostly within Zone 1F of the Richards Bay Industrial Development Zone (RBIDZ), within agricultural fields and covering only a small are within the light industrial area just to the north of the RBIDZ boundary. It is INFOTOX's considered opinion that, although daily concentrations were not calculated, the 99th percentile of the daily concentrations at even the closest receptor or residential area is likely to not be higher than background concentrations, or that the difference from background concentrations would be so slight as to be of no practical significance as far as risks to health are concerned. Therefore, it is concluded that exposure to CO associated with the proposed power plant is highly unlikely to result in health risks perceptibly higher than the background risk to health and cannot be viewed as a reason for concern in the exposed receptor population.

The assessment of benzene as a surrogate chemical substance representing the group of VOCs begins with Tier 1 risk-based screening levels, derived by international regulatory agencies using exposure parameters incorporating large safety factors to enable decisions on the side of caution. The Tier-1 assessment is generic in nature, designed to be on the conservative side, thus overestimating rather than underestimating health risks. Should the Tier-1 assessment indicate potential health risks, the HHRA assessment proceeds to the Tier-2 level. This approach and the methods followed therein have been developed and are supported by international regulatory agencies and the International Programme on Chemical Safety, which is a collaboration between three United Nations bodies, namely the WHO, the International Labour Organization and the United Nations Environment Programme.

The maximum total VOC concentration within the modelling domain is 6 x 10^{-6} µg/m^3, but there are no residences within the relevant impact area, which is mostly within Zone 1F of the RBIDZ and slightly into the light industrial area just to the north of the RBIDZ boundary. However, even if there had been residential exposure in the impact area, the maximum concentration, assessed as benzene, is orders of magnitude lower than the Tier-1 screening values protective of cancer by inhalation (0.36 µg/m^3) or non-cancer haematopoietic effects (3.1 µg/m^3, incorporating an additional safety factor of 0.1). Thus, there is not any substantive reason to view the modelled ambient total VOC concentrations resulting from operations at the proposed Phakwe Richards Bay Gas Power 3 Combined Cycle Gas to Power Plant as a risk to health.

The HHRA results conclusively exclude a risk to health significantly higher than the current background risk experienced by sensitive receptors within the modelled impact area. In summary,

the impact of emissions from the proposed power plant on health risks associated with exposure to PM2.5, SO_2, NO_2, CO and VOCs in air, in communities surrounding the smelter, is not of concern. Implementation of the proposed power plant is not associated with a risk to health that would be significantly higher than existing background risks at any of the sensitive receptors included in the modelled impact area., whether cancer or non-cancer effects are considered.

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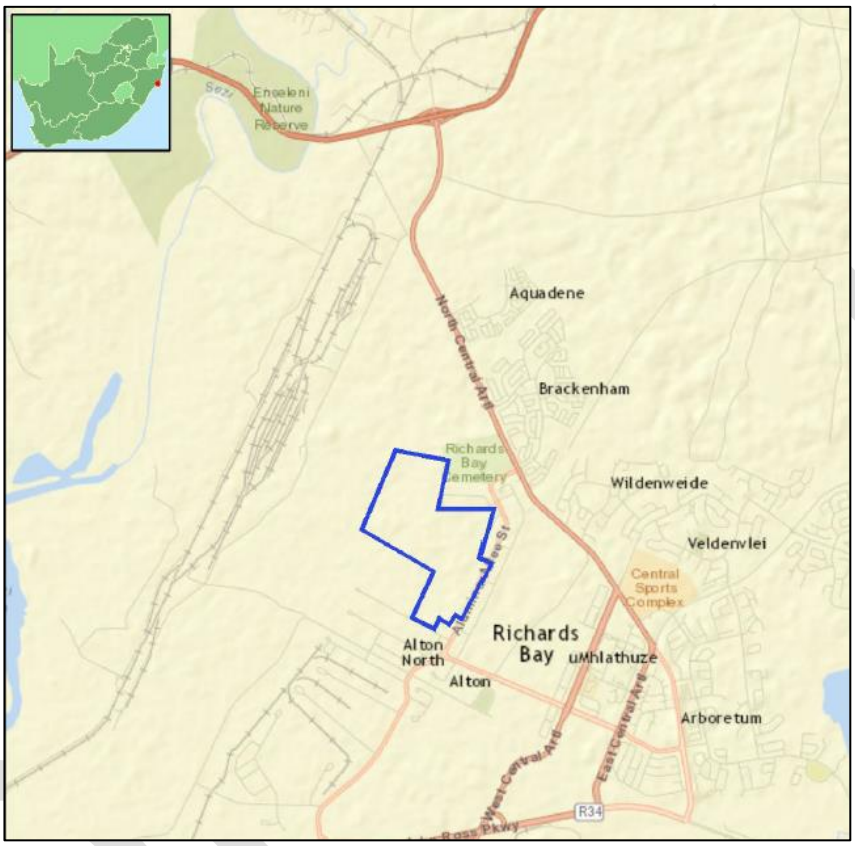
1 Introduction

Savannah Environmental (Pty) Ltd appointed INFOTOX (Pty) Ltd ("INFOTOX") to conduct a rapid appraisal health impact assessment (RAHIA) for the development of the Phakwe Richards Bay Gas Power 3 combined cycle power plant and related infrastructure located in Alton North, Richards Bay, within the uMhlathuze Local Municipality (LM) in the uThungulu District Municipality (DM), KwaZulu-Natal. The current General Orientation Map, compiled by Maroga (2020) is presented in Figure 1.1.



Note to Figure: The blue outline represents the proposed project site location.

Figure 1.1: General Orientation Map of the Richards Bay Gas-to-Power project (Maroga 2020).

This document presents the human health risk assessment (HHRA) for the RAHIA. The results of the HHRA feed into the RAHIA, presented in a separate report, 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 Study approach

According to the Good Practice guidance of the IFC, a RAHIA is suitable for the Phakwe project, because an influx of people settling in the area, due to the construction and operation of the facility, is not foreseen, as explained in the accompanying INFOTOX report (Fourie and Van Niekerk 2022). According to the IFC, the RAHIA does not require new health data collection within

the communities of concern. Baseline health data on the underlying burden of disease, used to identify specific vulnerabilities that might influence health impacts associated with the proposed power plant operations, are extracted from existing health data sources in a desktop review, presented in the accompanying INFOTOX report (Fourie and Van Niekerk 2022). The main focus of the HHRA is the health risks in surrounding receptor communities due to the dispersion of substances emitted by the proposed power plant operations (the source of exposure) into air (the pathway of exposure).

In terms of the RAHIA, the geographical study area considered as impacted includes those areas and communities where the proposed developments may have an impact on the environmental quality. The assessment of exposure in the residential areas to the likely airborne emissions from the project site is vital for the purposes of the HHRA. The impact of such emissions on air quality has been determined by an air dispersion modelling specialist of Airshed Planning Professionals (Pty) Ltd ("Airshed"). Amongst other factors, the specialist takes into account the local topographical and meteorological conditions in the modelling domain (Figure 2.1, provided by Airshed).

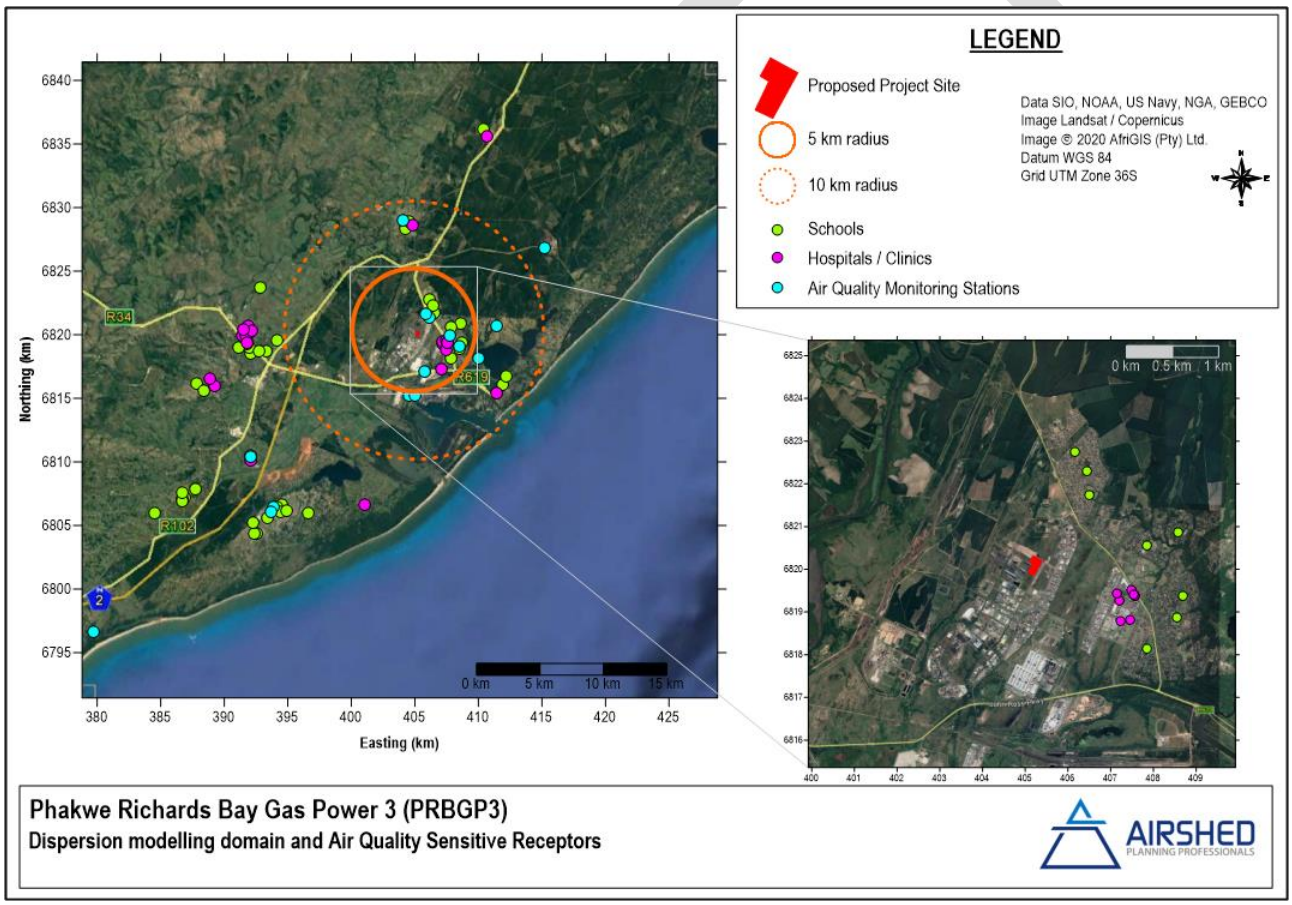


Figure 2.1: Air quality modelling domain of the Phakwe Richards Bay Gas Power 3 combined cycle power plant.

Air dispersion modelling and the results thereof, used by INFOTOX as input into the HHRA, are described in the Airshed Air Quality Impact (AQI) report (Bird and Von Gruenewaldt 2022). The ambient air contaminants of concern in the operational phase of the plant are four criteria pollutants, namely, the PM2.5 fraction of airborne inorganic particulate matter, carbon monoxide (CO), sulfur dioxide (SO_2) and nitrogen oxides as NO_2. VOCs (as an unspecified group of

substances) associated with the gas turbine operations are not regulated and health risk values for assessment of the unspecified group are not available. Thus, the generally accepted approach is to model and assess benzene as a surrogate chemical substance representing the group of VOCs.

3 Criteria pollutant health effects and relative risks for risk assessment

Relative risk (RR) ratios are the health risk ratios most often used in large epidemiological studies of the effects of criteria air pollutants on the health of receptor populations. The criteria pollutant HHRA is approached through the calculation of attributable fractions of disease (AFs) based on the incremental change in the air concentration of the pollutant of interest, using the RRs associated with exposure to a specific pollutant over the short- or long term.

The RRs and identified health outcomes are summarised in Table 3.1. The primary scientific references that INFOTOX consulted are also presented. A complete description of the calculation of criteria pollutant health risks and the review of health effects and RRs described in the epidemiological literature are available in Annexure 1.

Table 3.1: Summary of RRs for the criteria pollutants of interest.

Identified outcome	*RR	Exposure averaging time	Reference
PM2.5			
All-cause (natural) mortality all ages	1.0123	24-hour	WHO 2013
Cardiovascular admissions	1.0091		
Respiratory admissions	1.0190		
All-cause (natural) mortality age 30+	1.0676	Annual	USEPA 2019 Pope et al. 2015
Asthma incidence, ages 4 to 17	1.0167		USEPA 2019 McConnell et al. 2010
SO_2			
Asthma exacerbation	1.011	24-hour	Zheng et al. 2015 USEPA 2017
NO_2			
Asthma exacerbation	1.014	24-hour	Zheng et al. 2015 USEPA 2016
CO			
Myocardial infarction HAs: adults (18 years and older)	1.052**	8-hour	USEPA 2010 Lee et al. 2020

* RR per 10 µg/m^3 incremental pollutant concentration change
** RR per 1 000 µg/m^3 incremental pollutant concentration change

4 Benzene health effects and HHRA methods

4.1 HHRA methodology

The original paradigm for regulatory HHRA in the USA was developed by the US National Research Council (NRC 1983). This model has been adopted and refined by the US Environmental Protection Agency (USEPA) and other international agencies as published under the International Programme on Chemical Safety (IPCS 1999; IPCS 2010) and is widely used for

quantitative human health risk assessments. The elements of the HHRA approach are described below.

Hazard assessment is the identification of chemical contaminants suspected to pose hazards and a description of the types of toxicity that they evoke. The contaminant of interest is benzene and health effects are described in Section 4.2 of this report.

Dose-response assessment (toxicological assessment) addresses the relationship between levels of biological exposure to benzene and the manifestation of adverse health effects in humans, and/or how humans can be expected to respond to different doses or concentrations. The toxicological assessment follows a quantitative procedure that distinguishes between carcinogenic and non-carcinogenic effects (Section 4.2).

Exposure assessment includes a description of the environmental pathways and distribution of hazardous substances, identification of exposed individuals or communities, the routes of direct and indirect exposure, and an estimate of concentrations and duration of the exposure. The exposure assessment of the Phakwe Richards Bay project is described in Section 5.

Risk characterisation involves the integration of each component described above, with the purpose of determining whether specific exposures to an individual or a community would lead to adverse health effects (Section 6).

The assessment begins with Tier 1 risk-based screening levels, derived by international regulatory agencies using exposure parameters incorporating large safety factors to enable decisions on the side of caution. The Tier-1 assessment is generic in nature, designed to be on the conservative side, thus overestimating rather than underestimating health risks. Should the Tier-1 assessment indicate potential health risks, the HHRA assessment proceeds to the Tier-2 level.

4.2 Benzene inhalation toxicity values

4.2.1 Cancer

The International Agency for Research in Cancer (IARC) has classified benzene as "(*C)arcinogenic to humans"* (Group 1) based on sufficient evidence that benzene is carcinogenic to man (IARC 1982). The USEPA has classified benzene as a "*known"* human carcinogen (Group A), based upon convincing human evidence as well as supporting evidence from animal studies (IRIS 2003). Chronic (long-term) exposure to benzene may cause cancer of the blood-forming organs, resulting in the development of a particular type of leukaemia called acute myeloid leukaemia (AML) (ATSDR 2007).

Carcinogenic risk is assessed in terms of the IUR determined by the USEPA (IRIS 2003), which is 2.2×10^{-6} per $\mu g/m^3$ to 7.8×10^{-6} per $\mu g/m^3$ $(\mu g/m^3)^{-1}$. This unit risk is based on the development of leukaemia.

4.2.2 Chronic non-cancer effects

The haematopoietic system is a critical target for non-carcinogenic benzene toxicity. This system is the organ and cellular system responsible for the production of the cellular and non-cellular components of blood. Benzene is known to affect the capacity of bone marrow to produce the different types of blood cells. This may result in diminished numbers of red blood

cells, presenting as anaemia in affected persons, and diminished numbers of cells related to the immune system, such as leukocytes and lymphocytes (ATSDR 2007).

Noncarcinogenic risks are determined in terms of the RfC. The RfC for benzene is 0.03 mg/m^3, based on animal toxicology studies indicating decreased lymphocyte counts (IRIS 2003).

4.2.3 Tier-1 screening

The Tier-1 HHRA approach compares modelled air concentrations with health risk-based screening levels (Table 4.2.3.1). The USEPA (2021) Regional Screening Levels (RSLs) are risk-based concentration (RBC) limits available for a number of generic exposure scenarios. For the purposes of the assessment of possible inhalation of benzene in air by exposed receptor communities, INFOTOX used the RSLs for residential air.

Table 4.2.3.1: Tier-1 RSL values for benzene in air.

Target effect	Toxicity value
Cancer by inhalation	0.36 µg/m^3
Non-cancer haematopoietic effects	31.0 µg/m^3
Non-cancer haematopoietic effects with additional safety factor of 0.1	3.1 µg/m^3

5 Exposure assessment

Modelled air concentration isopleths of the criteria pollutants PM2.5, SO_2, NO_2 and CO, and of total VOCs, assessed as benzene, are presented in Annexure 2. Modelled criteria pollutant delta (Δ) concentrations at the 20 receptor locations identified as closest to the proposed Phakwe power plant are presented Table 5.1. These are the concentration differences between the background (the current baseline without emissions from the proposed plant) and the simulated prevalent ambient air concentrations modelled with the inclusion of normal operations at the plant (Bird and Von Gruenewaldt 2022).

Modelled air concentrations of CO and of total VOCs are not presented in tabulated format. In the case of CO, the maximum hourly concentration within the area plotted (Figure 12.5 in Annexure 2) is 14.94 µg/m³ and, although daily concentrations were not calculated, it is unlikely that the 99th percentile of the daily concentrations would be higher than 14.94 µg/m³. Furthermore, according to the air dispersion modelling specialist (Bird 2022), there are no houses within the impact area delineated by the isopleth, which is mostly within Zone 1F of the Richards Bay Industrial Development Zone (RBIDZ), within agricultural fields and slightly into the light industrial area just to the north of RBIDZ boundary.

The total VOC isopleth map (Figure 12.6 in Annexure 2) indicated prevalent annual air concentrations lower than 5 x 10^{-6} µg/m^3 over the residential areas. According to the air dispersion modelling specialist (Bird 2022) the maximum concentration in the isopleth as plotted in Figure 12.6 is 6 x 10^{-6} µg/m^3. There are no houses within the impact area delineated by the isopleth, which is mostly within Zone 1F of the Richards Bay Industrial Development Zone (RBIDZ) and slightly into the light industrial area just to the north of RBIDZ boundary.

Table 5.1: Operational phase criteria pollutant concentrations at receptors in the air dispersion modelling domain for the proposed Phakwe plant.

Receptor name	Daily PM2.5 (µg/m³)	Annual average PM 2.5 (µg/m³)	Daily SO_2 (µg/m³)	Daily NO_2 (µg/m³)
Richards Bay Christian School	0.708	0.041	13.280	1.466
Richards Bay Secondary School	0.516	0.043	7.365	0.854
Richards Bay Primary School	0.551	0.055	6.601	0.717
Richardsbaai Hoërskool	0.588	0.097	17.815	2.240
Veldenvlei Primary School	0.890	0.045	16.167	1.930
Arboretum Primary School	0.462	0.048	8.313	0.930
Bay Primary School	0.740	0.037	13.539	1.501
Brackenham Primary School	0.479	0.046	7.824	0.899
John Ross College	0.502	0.055	6.480	0.718
St Francis Pre-Primary School	0.708	0.041	13.280	1.466
Empangeni High School	0.708	0.041	13.280	1.466
Phesheya Primary School	0.412	0.038	5.578	0.590
Old Mill High School	0.530	0.052	9.764	1.133
Pinocchio Pre-Primary School	0.477	0.046	7.675	0.881
Empangeni Christian School	0.520	0.048	7.701	0.915
St Catherine's High School	0.502	0.047	7.366	0.890
Empangeni Preparatory School	0.472	0.046	7.130	0.833
Heuwelland Primary School	0.464	0.044	6.848	0.781
Thuthukani Special School	0.516	0.043	7.365	0.854
Felixton College	0.498	0.043	6.805	0.787
Tisand Technical High School	0.504	0.056	6.545	0.725
Hlamvana Secondary School	0.512	0.059	6.802	0.792
Thanduyise High School	0.435	0.040	6.079	0.612
Ilembe Primary School	0.509	0.057	6.693	0.737
Imizikayifani Primary School	0.513	0.053	6.354	0.686
Khula High School	0.548	0.060	6.838	0.810
Umdlamfe Secondary School	0.508	0.052	6.242	0.681
Bajabulile Primary School	0.566	0.063	7.077	0.826
Thambolini High School	0.551	0.055	6.601	0.717
Dlamvuzo Secondary School	0.536	0.050	6.303	0.671
University of Zululand	0.423	0.035	5.022	0.574
Khandisa Primary School	0.400	0.034	4.574	0.521
Dlangezwa High School	0.404	0.034	4.652	0.530
Ongoye Secondary School	0.398	0.034	4.565	0.520
Matshangule Primary School	0.401	0.034	4.566	0.520
Kwavulindlela Primary School	0.394	0.033	4.621	0.527

Receptor name	Daily PM2.5 (µg/m³)	Annual average PM 2.5 (µg/m³)	Daily SO_2 (µg/m³)	Daily NO_2 (µg/m³)
Qambokwethu Primary School	0.340	0.030	4.022	0.436
Mkhobosa Primary School	0.623	0.073	8.940	1.068
Tholokuhle Secondary School	0.588	0.097	17.815	2.240
Vondlo Primary School	0.578	0.097	16.797	2.112
Sinaye Primary School	0.587	0.100	19.354	2.437
Kwambonambi Primary School	0.484	0.070	10.910	1.286
Nseleni - Community Health Care	0.619	0.106	18.614	2.339
Mens Clinic International - Richards Bay	0.850	0.045	19.514	2.331
Richards Bay Municipal Clinic	0.917	0.049	17.909	2.139
The Bay Hospital	0.761	0.044	18.890	1.997
Better2Know Private STD Health Centre Richards Bay	0.852	0.046	19.870	2.379
Headache Clinic | Bay Chiropractic | Smile Dent	0.485	0.027	7.322	0.863
Umhlathuze Dental	0.890	0.045	16.167	1.930
Mandlazini Clinic	0.749	0.044	16.845	1.824
Mondi Felixton - Clinic	0.489	0.043	6.561	0.757
Pietermaritzburg Medi Clinic	0.453	0.046	6.938	0.824
Hope Clinic	0.457	0.047	7.229	0.888
Isiboniso Clinic	0.570	0.071	9.673	1.202
Better2Know Private STD Health Centre Empangeni	0.450	0.046	7.893	0.863
Blue Ladies Clinic	0.462	0.047	7.125	0.848
Life Empangeni Garden Clinic	0.447	0.047	7.709	0.947
Ngwelezana Hospital	0.452	0.041	6.589	0.649
Lower Umfolozi District War Memorial Hospital - Paediatric Ward	0.462	0.048	8.313	0.930
Ngwelezana clinic	0.437	0.040	5.861	0.635
Richardsbay Medical Institute	0.760	0.044	18.962	1.941
Bethlehem recovery centre Empangeni.	0.445	0.046	7.726	0.841
Sinalo Cerebral Palsy Centre	0.469	0.046	7.016	0.832
Ethembeni Care Centre	0.512	0.071	10.583	1.313
Esikhawini	0.538	0.059	6.823	0.810
Aquadene	1.324	0.258	49.139	6.135
Wild En Weide	0.760	0.061	19.727	2.419
Richards Bay Central	0.898	0.049	18.088	2.119
Arboretum	0.740	0.037	13.539	1.501
Birdswood	0.619	0.034	10.931	1.220
Richards bay - New	0.517	0.027	6.862	0.690
Meer en See	0.450	0.023	5.993	0.696
Ntshingimipisi	0.393	0.022	5.345	0.547

Receptor name	Daily PM2.5 (µg/m³)	Annual average PM 2.5 (µg/m³)	Daily SO_2 (µg/m³)	Daily NO_2 (µg/m³)
Nzalabantu	0.328	0.019	5.619	0.670
Vulindela A	0.397	0.034	4.556	0.519
Felixton	0.492	0.043	6.635	0.767
Eniwe	0.441	0.039	5.617	0.648
Hillview	0.432	0.043	6.955	0.841
Empangeni	0.479	0.046	7.824	0.899
Dondolo	0.427	0.039	5.973	0.678
Ngwelezana B	0.427	0.039	5.774	0.600
Nseleni A	0.576	0.099	19.217	2.416
Matshana	0.388	0.035	4.901	0.512
Airport (RBCAA)	0.597	0.031	9.188	0.968
Arboretum (RBCAA)	0.710	0.170	13.450	1.414
Brackenham (RBCAA)	1.459	0.180	46.214	5.775
CBD (RBCAA)	0.640	0.045	14.285	1.781
Esikhawini (RBCAA)	0.502	0.055	6.480	0.718
Felixton (RBCAA)	0.496	0.043	6.766	0.783
Harbour West (RBCAA)	0.976	0.028	27.400	3.413
Scorpio (RBCAA)	1.294	0.095	34.225	4.232
eNseleni (RBCAA)	0.541	0.090	15.970	1.989
Brackenham (uMhlathuze)	2.023	0.328	78.813	9.852
Arboretum (uMhlathuze)	0.512	0.029	10.320	1.200
eSikhaleni (uMhlathuze)	0.525	0.055	6.558	0.725

Note to Table: the daily concentrations are the simulated 99th percentile of each pollutant.

6 HRA results and interpretation

6.1 Results of the criteria pollutant risk assessment

AFs calculated for the various pollutants at the various receptors are presented in detail in Annexure 3. The added risk to health, above the background risk, due to resultant ambient air concentrations of criteria pollutants originating from the proposed power plant, is expressed as the AFs of all-cause natural mortality and hospitalisation for cardiovascular and respiratory causes related to short-term exposure to PM2.5, summarised in Table 6.1.1. The added AF of all-cause natural mortality in those older than 30 years of age and the avoided fraction of asthma incidence in those aged 4 to 17 years of age, related to long-term exposure to PM2.5, are summarised in Table 6.1.2.

Table 6.1.3 presents a summary of the added fraction of asthma exacerbation in exposed asthmatics of all ages, associated with the modelled changes in 24-hour SO_2 concentrations. Avoided risks of asthma-related emergency visits and hospitalisation associated with short-term exposure to NO_2 are summarised in Table 6.1.4.

Short-term exposure to CO is assessed as the added fraction of hospitalisation for myocardial infarction, attributable to increased ambient air concentrations of CO due to operations at the proposed power plant. However, as indicated by Bird (2022) and as discussed in Section 5, the maximum hourly concentration of 14.94 µg/m³ is modelled in an area where there are no houses (Figure 12.5 in Annexure 2). According to Figure 12.5, the hourly CO concentrations outside of the modelled isopleth are all lower than 5 µg/m³. It is INFOTOX's considered opinion that, although daily concentrations were not calculated, the 99th percentile of the daily concentrations at the closest receptor or residential area is likely to not be higher than background concentrations, or that the difference from background concentrations would be so slight as to be of no practical significance as far as risks to health are concerned. Therefore, CO risk calculations would not provide any substantial additional information and are not performed.

AFs are presented in scientific notation. Therefore, 7.2E-06 is equal to 7.2 x 10^{-6} or 0.000007, etc.

Table 6.1.1: AFs of mortality and hospital admissions associated with short-term exposure to PM2.5.

Statistics parameter	All-cause (natural) mortality	Cardiovascular hospitalisation	Respiratory hospitalisation
Mean	5.9E-04	5.9E-04	5.9E-04
Median	5.2E-04	5.2E-04	5.2E-04
Range: lowest – highest values	3.3E-04 – 2.0E-03	3.3E-04 - 2.0E-03	3.3E-04 - 2.0E-03
95% Confidence interval (CI) of the mean	5.9E-04 – 6.0E-04	5.9E-04 – 6.0E-04	5.9E-04 – 6.0E-04

Table 6.1.2: AFs of mortality and hospital admissions associated with long-term exposure to PM2.5.

Statistics parameter	All-cause (natural) mortality age 30+	Asthma incidence, ages 4 to 17
Mean	9.5E-05	9.5E-05
Median	7.6E-05	7.6E-05
Range: lowest – highest values	3.2E-05 - 5.5E-04	3.2E-05 - 5.5E-04
95% Confidence interval (CI) of the mean	7.2E-05 - 1.2E-04	8.9E-05 - 1.0E-04

Table 6.1.3: AFs of asthma exacerbation associated with short-term exposure to SO_2.

Statistics parameter	AF (unitless)
Mean	1.3E-02
Median	8.1E-03
Range: lowest – highest values	4.5E-03 – 8.4E-02
95% Confidence interval (CI) of the mean	1.0E-02 - 1.5E-02

Table 6.1.4: AFs of asthma-related emergency visits and hospitalisation associated with short-term exposure to NO_2.

Statistics parameter	AF (unitless)
Mean	3.4E-02
Median	2.9E-02
Range: lowest – highest values	8.3E-04 - 9.1E-02
95% Confidence interval (CI) of the mean	2.8E-02 – 4.0E-02

6.2 Interpretation of the criteria pollutants risk results

The AFs are interpreted as the fraction of the risk of a specific health effect, e.g., asthma exacerbation, experienced by the receptor population, that can reasonably be attributed to exposure to the assessed criteria pollutant originating from the investigated source, at the ambient air concentration modelled by the air dispersion specialist. Thus, an AF of 1 per cent is interpreted as an indication that 1 per cent of the total risk of a specific health effect in an exposed person can be reasonably attributed to exposure to the pollutant of interest, at the modelled concentration. The balance of the risk, e.g., 99 per cent, is attributable to other known or unknown factors, and/or to background levels of exposure to the criteria pollutant of interest, and/or to factors specific to the exposed person, that are not related to the investigated source of the criteria pollutant of interest. In this report, the investigated source is, of course, the Phakwe Richards Bay Gas Power 3 Combined Cycle Gas to Power Plant. The assessed exposure concentrations are those modelled by the air dispersion specialist for a scenario of normal operations at the plant.

All of the AFs are in the range less than 10 per cent. The lowest AFs are calculated for exposure to PM2.5, whether over the short- or long-term periods of exposure to modelled concentrations. The AFs are all lower than 0.1 per cent in the case of exposure to PM2.5. Such AFs are for all practical purposes not significant and in the negligible range. The reported AFs cannot be interpreted as indicating any reason for concern with regard to human health risks associated with the slightly increased ambient air PM2.5 concentrations as a consequence of the proposed operations of the Phakwe power plant.

The single highest AF calculated for asthma exacerbation associated with short-term exposure to SO_2 is 8.4 per cent (AF of 8.4E-02, the upper range limit reported in Table 6.1.3). Aside from this single highest AF, AFs of approximately 1 per cent were calculated in 36 per cent of the receptors and less than 1 per cent in all remaining (almost 64 per cent) receptors. The calculated AFs are centred around a middle value (the median in Table 6.1.3) of approximately 1 per cent. In conclusion, the AFs calculated for the receptors are in the range viewed as very low to negligible. Even the highest calculated AF cannot be viewed as a significant or serious risk to health. It should be noted that the calculated risks are only applicable to persons already diagnosed as asthmatic and must not be interpreted as an indication that additional asthma cases will be diagnosed after the start of operations at the proposed Phakwe power plant. The risk of asthma exacerbation in those persons after operations have started is only slightly higher than the existing background risks in such persons. Furthermore, the calculated risks at each receptor (Table 12.3) are applicable only on the days (approximately 1 per cent, or 4 days in a year) when the modelled highest daily SO_2 concentrations (Table 5.1) are actually reached. On all other days the risk in the impacted area will be even lower. Considering the entire set of SO_2 risk results, there is

insufficient cause to conclude that the risk to health due to short-term exposure to SO_2 associated with the proposed power plant is significantly higher than the background risk to health.

The highest AFs calculated for asthma exacerbation leading to an asthma-related emergency care visit or hospitalisation associated with short-term exposure to NO_2 are approximately 9 per cent, the upper range limit reported in Table 6.1.4. Most calculated AFs are centred around a middle value (the median in Table 6.1.4) of approximately 3 per cent. The calculated AFs are in the range viewed as very low to negligible and cannot be interpreted as indicating a significant or serious risk to health. Thus, there is a slight, but likely insignificant risk of the infrequent exacerbation of asthma in those persons known to be diagnosed as asthmatic and residing in the receptor area. As explained for exposure to SO_2, the calculated risks are applicable only to persons already diagnosed as asthmatic and are not to be interpreted as an indication that additional asthma cases will develop after the start of operations at the proposed power plant. The calculated slight risks are applicable only on those days when the modelled highest daily NO_2 concentrations are actually reached. On all other days the risk will be lower. Considering all NO_2 risk results, it is concluded that the risk to health due to short-term exposure to NO_2 associated with the proposed power plant is only slightly to insignificantly higher than the background risk to health. Such health risks cannot be viewed as serious or as a reason for concern in the exposed receptor population.

As already explained in Section 5 for CO exposure associated with operations at the proposed power station, there are no residences within the impact area delineated by the results of air dispersion modelling. The impacted area is mostly within Zone 1F of the RBIDZ, within agricultural fields and covering only a small area within the light industrial zone just to the north of the RBIDZ boundary. It is INFOTOX's considered opinion (Section 6.1) that, although daily concentrations were not calculated, the 99th percentile of the daily concentrations at even the closest receptor or residential area is likely to not be higher than background concentrations, or that the difference from background concentrations would be so slight as to be of no practical significance as far as risks to health are concerned. Therefore, it is concluded that exposure to CO associated with the proposed power plant is highly unlikely to result in health risks perceptibly higher than the background risk to health and cannot be viewed as a reason for concern in the exposed receptor population.

6.3 Total VOC assessed as benzene

The maximum total VOC concentration within the modelling domain is 6 x 10^{-6} µg/m^3 (Bird 2022, compare Figure 12.6). There are no residences within the relevant impact area, which is mostly within Zone 1F of the RBIDZ and slightly into the light industrial area just to the north of the RBIDZ boundary. However, even if there had been residential exposure in the impact area, the maximum concentration, assessed as benzene, is orders of magnitude lower than the Tier-1 screening values protective of cancer by inhalation (0.36 µg/m^3) or non-cancer haematopoietic effects (3.1 µg/m^3, incorporating an additional safety factor of 0.1). Thus, there is not any substantive reason to view the modelled ambient total VOC concentrations resulting from operations at the proposed Phakwe Richards Bay Gas Power 3 Combined Cycle Gas to Power Plant as a risk to health.

7 Uncertainties

The HHRA in this report is based on modelled ambient air concentrations of PM2.5, SO_2, NO_2, CO and total VOCs provided by Airshed (Bird and Von Gruenewaldt 2022). If is expected that uncertainties associated with the modelled concentrations are discussed in the Air Quality Impact Assessment report submitted by Airshed and are not elaborated here.

The HHRA for the criteria pollutants PM2.5, SO_2, NO_2 and CO followed standard international practice, based on methodologies applied in epidemiological studies. The centrepin of the quantification of the health risk assessment is the relative risk (RR) ratio, used to calculate the likely health effect response following on a modelled exposure to the pollutant of interest. The results of the HHRA are presented with a high degree of confidence in the RRs used to quantify health risks assessed in this report. The RRs were derived from large international epidemiological studies reviewed by international regulatory and scientific agencies, namely the USEPA and the WHO, and from strong epidemiological studies using the systematic review and meta-analysis methodology.

Uncertainty in the results of the study is vested in the use of RRs mostly based on studies in developed countries, since RRs applicable to a developing country such as South Africa are not available. However, the estimates presented in this report are the most accurate that are currently achievable. The ideal source of RRs for risk quantification would be South African epidemiological studies, since socio-economic factors unique to South Africa might influence the estimated outcomes. However, a sufficient database of such epidemiological studies is not currently available in South Africa. Nonetheless, the use of RRs determined in systematic review and meta-analysis studies mitigates this limitation, as the systematic reviews are not limited to westernised or developed countries only. It is not expected that the potential influence of these factors would significantly affect the outcome of the assessments, and the interpretations presented in this report are the most valid that can be achieved in view of the acknowledged limitations.

Short-term exposure is assessed by the calculation of AFs on the basis of the 99th percentile of daily concentrations, which is considered a highly conservative upper estimate of the daily exposure concentrations for HHRA purposes. The 99th percentile represents the concentration exceeded by only 1 per cent of the modelled days, which would be at most 3 to 4 days in a 365-day period.

The risks of exposure to total VOC originating from the proposed power plant are assessed based on the conservative assumption that the total VOCs are represented by benzene. This is a significantly conservative assumption, because not all VOCs are carcinogenic (as is benzene) and not all VOCs are considered as hazardous to health. Thus, the assumptions underlying the assessment of total VOC exposure ensure a significant margin of safety; overestimating rather than underestimating the risks to health posed by exposure to VOCs. The assessment is presented with confidence, as it is based on the cancer and non-cancer toxicity values for benzene, applied and supported by major international health risk assessment and regulatory agencies. The tiered risk assessment approach used for the assessment is internationally accepted and the HHRA paradigm applied by INFOTOX is considered best practice for community HHRAs in the international scientific risk assessment community.

8 Conclusions

- Modelled changes in daily and annual PM2.5 ambient air concentrations and in daily SO_2, NO_2 and CO concentrations, due to operations at the proposed Phakwe power plant, were used to assess changes in health risks in communities surrounding the project area. Very slight to negligible changes in health risks associated with inhalation of these criteria pollutants originating from the proposed power plant are indicated. Therefore, there is not any reason for concern with regard to human health risks associated with the air quality consequences of the emission of PM2.5, SO_2, NO_2 and CO by the proposed power plant.

- The impact of the proposed Phakwe power plant total VOC emissions on health risks in communities surrounding the project area is not of concern. Normal power plant operations were modelled and assessed, and are not associated with a risk to health, whether cancer or non-cancer effects are considered, at any of the sensitive receptors included in the modelling domain.

- The HHRA results conclusively exclude a risk to health significantly higher than the current background risk experienced by sensitive receptors within the modelled impact area. In summary, the impact of emissions from the proposed power plant on health risks associated with exposure to PM2.5, SO_2, NO_2, CO and VOCs in air, in communities surrounding the smelter, is not of concern. Implementation of the proposed power plant is not associated with a risk to health that would be significantly higher than existing background risks at any of the sensitive receptors included in the modelled impact area, whether cancer or non-cancer effects are considered.

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10 Annexure 1: PM2.5, SO_2, NO_2 and CO health risk calculations: methods, literature review and update of risk ratios (RRs)

10.1 Background

The air quality report presented the modelled existing ambient air concentrations of the criteria pollutants particulate matter (PM2.5[1]), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and carbon monoxide (CO). It is important to note that it is common to observe increases in mortality or hospitalisation rates even when the prevalent air concentrations do not exceed the environmental air quality guidelines or standards. For example, health effects from exposure to PM2.5 concentrations below particulate matter air quality guidelines are well documented (WHO 2000 and 2005). Simplistic comparisons between exposure concentrations and ambient air quality guidelines are inadequate to quantify health outcomes, mainly because ambient air quality guidelines are used for management of air quality and are not intended for risk quantification. Furthermore, researchers have not been able to establish a safe threshold below which there are no health risks (WHO 2000 and 2005). Assessment of impacts of air pollutants on health may not be restricted to areas in which the guideline concentrations are exceeded, but should also include areas in which air concentrations are within the limits.

Mortality or hospitalisation rates for respiratory or cardiovascular causes are the measures of associated illness that are mostly applied in epidemiological studies of community health risks associated with exposure to criteria pollutants. The international scientific literature is not static and major regulatory agencies such as the US Environmental Protection Agency (USEPA) and the UK Committee on the Medical Effects of Air Pollutants (COMEAP) regularly review their risk models.

10.2 Causality

Epidemiological and experimental human exposure studies are used to investigate the relationship between health effects and exposure to particulate matter (PM), SO_2, NO_2 and CO, respectively. Epidemiological studies typically focus on incidence rates for various health endpoints such as cases of respiratory and cardiovascular disease, hospital admission and premature mortality. The purpose is to show a cause - effect relationship where cause relates to exposure and effect is the disease or death as a result of the cause (the exposure). Causation is an essential concept in epidemiology; yet there is no single, clearly expressed definition for causation. A statistically significant association between cause and an effect does not infer a causal relationship, although a strong association is often an indication of causality. Adequate evidence is necessary to establish a causal relationship between exposure and a consequence. Criteria used to determine causality include the strength of association, temporality, consistency, theoretical plausibility, coherence, specificity in the causes, dose response relationships, experimental evidence and analogy. Causality determinations are therefore based on the evaluation and synthesis of evidence from across scientific disciplines. The USEPA assessment system used a five-level hierarchy:

1. Causal relationship

2. Likely to be causal relationship

[1] Particulate matter with aerodynamic diameter equal to and smaller than 2.5 microns.

3. Suggestive of, but not sufficient to infer, a causal relationship

4. Inadequate to infer the presence or absence of a causal relationship

5. Not likely to be a causal relationship

A causal relationship is assigned if the consistency and coherence of evidence integrated across scientific disciplines and related health outcomes are sufficient to rule out chance, confounding, and other biases with reasonable confidence. If evidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain, the relationship is referred to as "*likely to be causal*". This means that chance and bias can be ruled out with reasonable confidence, but potential issues may remain (USEPA 2016, 2017 and 2019). The "*causal"* and "*likely to be causal"* relationships are associated with most certainty and are the focus of this report.

The USEPA's conclusions regarding short and long-term exposure to PM2.5 (USEPA 2019), SO_2 (USEPA 2017), NO_2 (USEPA 2016) and CO (USEPA 2010) and key health effects for which causality is accepted are summarised in Sections 10.5, 10.6, 10.7 and 10.8. respectively.

The Review of Evidence on Health Aspects of Air Pollution – REVIHAAP report compiled by the WHO (2013a) provided evidence for the causality of health effects of exposure to PM and NO_2. 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 cost-benefit analyses of air pollution abatement and related prevention of health effects. The recommended pollutant-outcome pairs, for which causality are accepted, were classified 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. Only two-week PM2.5 averages were assigned to this group.

For the purposes of this study, only those pairs for which reliable concentration-response functions were recommended (Group A) by the WHO (2013b) are included in the burden of disease assessment for the HHRA, although causality is accepted for both Group A and Group B pairs. The WHO's conclusions regarding short and long-term exposure to PM2.5 and NO_2 and key health effects for which reliable quantification of effects is possible are summarised in Sections 10.5 and 10.7, respectively.

10.3 Airborne particulate matter (PM) size fractions

According to the USEPA (2019) *Integrated Science Assessment (ISE) for Particulate Matter,* PM in ambient air is a complex mixture of solid particles and liquid droplets comprised of various components (e.g., metals or black carbon) with multiple size fractions, varying in mass and composition, depending on the locality and source of the PM (USEPA 2019). The total suspended particulates (TSP) in air may be divided into the following main fractions of significance to health:

- Fine PM (PM2.5), particulate matter with a nominal mean aerodynamic diameter less than or equal to 2.5 µm

- Thoracic coarse or coarse PM (PM10 to 2.5), particulate matter with a nominal mean aerodynamic diameter greater than 2.5 µm and less than or equal to 10 µm

- Ultrafine particles (UFPs), generally considered as particulates with a diameter less than or equal to 0.1 µm (100 nm), based on physical size, thermal diffusivity or electrical mobility

Combustion particles, organic compounds and fine metal particles are to be found in the PM2.5 fraction, but pollen and mould spores are mostly found in the coarse PM fraction (USEPA 2019).

PM2.5, rather than PM10 is the health risk indicator of choice with regard to assessment of the burden of disease or health risks associated with PM exposure (USEPA 2009). COMEAP (2007) had concluded that PM2.5 was the most satisfactory index of particulate air pollution for quantitative assessments. The World Health Organization (WHO 2005) based the particulate matter guidelines on epidemiological studies of PM2.5, and then extrapolated the resulting PM2.5 guideline to a PM10 guideline, because the majority of PM monitoring data published at that time were for PM10. The most recent WHO reports (2013a and b) provided evidence for the causality of health effects associated with exposure to PM and also concluded that quantification of risks based on PM2.5 was more reliable than quantification based on PM10. Therefore, PM risk analysis in this study uses concentration-response functions for PM2.5.

10.4 Risk quantification

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).

$$E = AF \times P \times B / 1000 \qquad (10.4.1)$$

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:

$$AF = \frac{(RR-1)}{RR} \quad (10.4.2)$$

Where:

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

In cases where incremental contributions in pollutant concentrations are estimated, the relative risk of mortality or morbidity (*RR*) in the exposed population may be calculated as follows:

$$RR = e^{(\Delta_{deaths} \times \Delta p)} \quad (10.4.3)$$

Where:

Δ_{deaths}	Potential proportion change in mortality associated with a change in the pollutant concentration of 1 µg/m^3
Δ_p	The modelled change in the pollutant concentration in µg/m^3

Regarding Equation 10.4.3, it should be noted that RRs are often derived in terms of incremental increases of 10 µg/m^3 in the pollutant concentration, in which case the potential proportion change would be associated with the incremental increase in terms of units of 10 µg/m^3. In this case, the practical implication is that the modelled change in the pollutant concentration must be divided by 10 for input into Equation 10.4.3.

The above changes 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.

10.5 Assessments of PM2.5 health effects

10.5.1 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 $PM_{2.5}$ exposures and lung cancer incidence and mortality, on inhalation studies in mice exposed to ambient air PM_{10}, 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 10.5.1.1 and 10.5.1.2.

Table 10.5.1.1: USEPA causality determination for short-term PM2.5 exposure.

Health effects	Causality determination	Associated health endpoint
Respiratory effects	Likely to be causal	ED visits and hospital admissions for asthma exacerbation in asthmatic adults and children
		ED visits and hospital admissions for chronic obstructive pulmonary disease (COPD)* (mostly adults)
		ED visits and hospital admissions for respiratory infections (adults and children)
Cardiovascular effects	Causal	ED visits and hospital admissions for exacerbation of existing heart conditions, including arrhythmia, heart failure ischaemic heart disease and myocardial infarction
		ED visits and hospital admissions for 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.

Table 10.5.1.2: USEPA causality determination for long-term PM2.5 exposure.

Health effects	Causality determination	Associated health endpoint
Respiratory effects	Likely to be causal	Decrements in lung function growth and attainment in children and lung functions in adults
		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)

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

The Review of Evidence on Health Aspects of Air Pollution – REVIHAAP report compiled by the WHO (2013a) provided evidence for the causality of health effects of exposure to PM. 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 cost-benefit analyses of air pollution abatement and related prevention of health effects. The recommended pollutant-outcome pairs, for which causality are accepted, were classified 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. Only two-week PM2.5 averages were assigned to this group.

For the purposes of this study, only those pairs for which reliable concentration-response functions were recommended by the WHO (2013b) for PM2.5 are included in the burden of disease assessment for the HHRA. A summary of these pairs involving PM2.5 is given in Table 10.5.1.3.

Table 10.5.1.3: WHO outcomes involving PM2.5.

Health outcome
Short-term exposure – PM2.5, daily mean
Hospital admissions, respiratory diseases, all ages
Hospital admissions, cardiovascular diseases (CVDs) (includes stroke), all ages
Mortality, natural all-cause, all ages
Long-term exposure – PM2.5, annual mean
Mortality: all-cause (natural), age 30+ years
Mortality due to cerebrovascular disease (includes stroke), ischaemic heart disease, chronic obstructive pulmonary disease (COPD) and trachea, bronchus and lung cancer, age 30+ years

Table 10.5.1.4 presents a comparison of the USEPA (2019) and WHO (2013b) assessments. Only health endpoints indicated as causal or likely to be causal by at least one agency are included. The causality assessments are in general agreement.

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 updated ambient air quality regulations. Although the outcomes of the WHO assessments are also supportive of ambient air quality regulation processed, the main goal of the WHO was to support burden of disease assessments contributing to cost-benefit calculations. Thus, the WHO excluded health outcomes that were considered difficult to monetise, such as low birth weight and lung function (WHO 2013b). It is also noteworthy that the WHO estimates effects in adult populations in the 30+ age group, as most of the evidence providing the concentration-response functions for burden of disease estimation comes from studies that focused on populations around 30 years of age and above (WHO 2013b). Concentration-response functions are not always proposed for short- and long term PM2.5 exposure, but available functions derived from large studies and proposed by the USEPA or WHO are discussed in Section 10.5.2.

Table 10.5.1.4: WHO and USEPA causality and WHO effect quantification.

Health outcome		WHO (2013a and b)	USEPA (2019)
Short-term exposure – PM2.5, daily mean			
Hospital admissions for respiratory diseases, all ages		Causal, reliable quantification of effects	Likely to be causal
Hospital admissions for cardiovascular diseases (includes stroke), all ages		Causal, reliable quantification of effects	Causal
Mortality - natural all-cause, all ages		Causal, reliable quantification of effects	Causal
Mortality: cause-specific	Cardiovascular disease	Causal, quantification not proposed	Causal (strong evidence)
	Respiratory disease	Causal, quantification not proposed	Causal (limited evidence)
Long-term exposure – PM2.5, annual mean			
Respiratory effects – lung function decrement, all ages, asthma incidence and prevalence increase in children		Linked, but causality not specified	Likely to be causal
Cardiovascular: Cardiac heart disease or stroke in those with pre-existing disease		Causal, insufficient evidence to propose quantification	Causal
Nervous system - reduced cognitive function and neurodegeneration in adults		Causality not assessed	Likely to be causal (consistent)
Nervous system effects - neurodevelopmental effects in children		Causality not assessed	Likely to be causal (limited and inconsistent)
Cancer - lung, all ages		Causal, insufficient evidence to propose quantification	Likely to be causal
Mortality - natural all-cause, all ages		Causal, age group 30+ specified	Causal
Mortality: cause-specific	Cardiovascular	Causal, specified as ischaemic heart disease and stroke	Causal
	Respiratory disease	Causal, specified as acute lower respiratory infections in children 0 to 5	Causal
	COPD	Causal, specified in adults age 25+	Causal
	Lung cancer	Causal, specified in adults age 25+	Mortality not specified as causally linked

10.5.2 Concentration-response functions for health risk calculations

The concentration-response functions (CRFs) yield numerical risk predictor values used for risk quantification, discussed in Section 3.4. Without discussing the technical detail, these numerical predictors are relative risk (RR) or odds ratios (OR). RRs were determined by the USEPA (2019) and the WHO (2013b) for short- and long-term exposure to PM2.5. Both authorities have paid considerable attention to the shape and slope of the CRF curve. For long-term (annual 24-hour average) PM2.5 concentrations it was concluded that there is at least initial evidence indicating that a log-linear association persists and that the slope of the function may be steeper, yielding a higher value of the outcome predicter at lower existing air PM2.5 concentrations, generally below 30 µg/m^3 (USEPA 2019 and WHO 2013a). The USEPA (2019) has concluded that initial evidence is also available for the short-term (daily 24-hour concentration) PM2.5 concentrations, but the WHO (2013b) has proposed fixed RR values for assessment of short-term effects, regardless of the existing daily PM2.5 concentration value, whether it be in the lower or higher range.

The WHO (2013b) has recommended in particular for long-term exposure that this problem be handled by implementing integrated exposure–responses functions (IERs) for the differentiated mortality outcomes and for acute lower respiratory infection (ALRI) in children younger than 5 years of age (see Table 4.1.4). This recommendation was realised in the WHO/Europe's software tool AirQ+, which performs calculations that allow quantification of the health effects of exposure to air pollution (WHO 2019). An IER is a function that integrates RR information from studies of ambient air pollution and other PM sources such as second-hand tobacco smoke, household solid cooking fuel, and active smoking, and incorporates the existing annual average PM2.5 concentration as a determinant of the RR to be deployed in the CRF and were originally developed in the seminal publication by Burnett et al. (2014). The AirQ+ software tool is updated at regular intervals and uses the most recent IERs developed for this purpose by various authors. The equivalent USEPA software tool is the open-source environmental Benefits Mapping and Analysis Program - Community Edition (BenMAP-CE) (USEPA 2021), which replaces the proprietary version of the program that the USEPA had first developed in 2003 to analyse national-scale air quality policies.

10.5.3 RRs for health risk calculations

CRFs and RRs are not necessarily available for all health endpoints, e.g., mortality or hospitalisation for specific causes for which health effects causality is accepted. The focus in Tables 10.5.3.1 and 10.5.3.2 is firstly on health effects with RRs recommended by the USEPA and the WHO. Secondly, double counting is explained and avoided.

RRs for short-term exposure developed by the USEPA and the WHO are both considered (Table 10.5.3.1), but risks are calculated with the highest of the USEPA and WHO RRs. This is done in order to assure a conservative assessment, which might overestimate risk slightly to moderately. The USEPA found that cause-specific mortality (cardiovascular and respiratory) was also causally related to short-term PM2.5 concentrations. However, individual causes were also statistically related to all-cause mortality. Thus, in order to avoid double-counting of the mortality effect, only all-cause natural mortality (not-accidental, not-homicidal, not self-harm), not the cause-specific mortalities, will be calculated by INFOTOX.

Table 10.5.3.1: Short-term PM2.5 relative risk ratios used by the USEPA and WHO.

Identified outcome	RR per 10 µg/m^3 PM2.5 increase or contribution	
	USEPA	WHO (2013b) - Europe
All-cause (natural) mortality	1.0019 (US less urban and rural locations in the analysis) to 1.0028 (mostly US urban) (USEPA 2019)	1.0123
Cardiovascular admissions	RRs cited in BenMap CE v1.5 (2021) not used by INFOTOX: limited to age group >65 (Bell et al. 2015, cited in USEPA 2021)	1.0091
Respiratory admissions	RRs cited in BenMap CE v1.5 (USEPA 2021) not used by INFOTOX: either limited to children, or adults >65, or based on only one US state	1.0190

RRs for long-term exposure, calculated by the USEPA and the WHO are considered, as indicated in Table 11.5.3.2. The USEPA ISA (2019) and the WHO (2013b) presents RRs standardised for a 5 or 10 µg increase in PM2.5 and the BenMap CE software indicates RRs associated with a 1 µg/m^3 PM2.5 concentration increase. For comparative purposes, INFOTOX adjusted the RRs linearly to an increased PM2.5 concentration difference of 10 µg, to facilitate comparison with the WHO (2013b) RRs. This adjustment technique is acceptable and is used by authors of meta-analyses, who need to standardise RRs from different studies to a common concentration difference (Huang et al. 2021 and Zheng et al. 2015(b)). If more than one RR is indicated in BenMap CE or in the USEPA ISA, the most conservative (highest) RR value is adjusted. The highest RR value among BenMap CE, the USEPA ISA (2019) and the WHO (2013b) is chosen for the burden of disease assessment and is shaded in Table 11.5.3.2.

The generation of multiple sets of results was prevented by focusing on all-cause natural mortality associated with exposure over the long term, not differentiating between cardiovascular and respiratory mortality causes, and also not lung cancer mortality. Hospital admissions are not considered over the long term, because these are already covered in the short-term exposure assessment. Asthma incidence increases in children were likely to be causally related to PM2.5 exposure over the long term (USEPA 2019) and are assessed in the HHRA.

Table 10.5.3.2: Long-term PM2.5 relative risk ratios used by the USEPA and WHO.

All-cause (natural) mortality age 30+	
USEPA RR	1.00582 (Turner et al. 2016) and 1.00676 (Pope et al. 2015), both cited in BenMap CE v1.5 (USEPA 2021), both per 1 µg/m^3 PM2.5 increase
*Adjusted USEPA RR	1.0676 (Pope et al. 2015), adjusted RR confirmed in authors' publication
WHO (2013b) RR	1.062 per 10 µg/m^3 PM2.5 increase
Asthma incidence, ages 4 to 17	
USEPA RR	1.02913 (McConnell et al. 2010) cited in BenMap CE v1.5 Manual (USEPA 2021), per 17.4 µg/m^3 PM2.5 increase.
*Adjusted USEPA RR	1.01674
WHO (2013b) RR	Asthma incidence not assessed by WHO

* Adjusted for a 10 µg/m^3 PM2.5 increase, assuming a linear increase in risk

10.6 Assessments of SO_2 health effects

10.6.1 Causality assessments

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. 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 10.6.1.1 and 10.6.1.2, 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 10.6.1.1: USEPA 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 10.6.1.2: USEPA 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

10.6.2 RRs for health risk calculations

The numerical predictors of risks associated with exposure to SO_2 are RRs, derived from the CRFs for SO_2 exposure and health endpoint occurrences, e.g., asthma exacerbation, as explained for PM2.5 (Section 10.5.3). RRs from studies reviewed by the USEPA (2017) varied across a wide range, reflecting imprecision in the risks yielded by the existing collection of related publications. Meta-analyses ameliorate this difficulty, by combining results and statistics from a collection of comparable studies. Meta-analyses have superior statistical power in comparison to individual studies, and improve the precision of the effect size estimates of an exposure-outcome association. Therefore, a literature search for meta-analyses of the relationships between short-term SO_2 exposure and respiratory health endpoint occurrences was conducted. Only respiratory health endpoints were of interest, because only respiratory effects were causally related to short-term SO_2 exposure.

Three recent meta-analyses were obtained, by Huang et al. (2021) and Zheng et al. (2015a and 2015b), of which the first publication is a time series meta-analysis (Zheng et al. 2015a) and is included in the USEPA (2017) ISA for SO_2. All three meta-analyses were focused on the outcome of asthma exacerbation, but only Zheng et al. (2015a and b) found statistically significant positive RRs for the effects of SO_2. Asthma exacerbation was measured as asthma-related hospital admissions and emergency room visits and the obtained RRs were in very good agreement. The time series meta-analysis (Zheng et al. 2015a) yielded an RR of 1.011 (95% CI[1] of 1.007, 1.015) and the 2015(b) publication an RR of 1.010 (95% CI of 1.001, 1.020), all RRs for a 10 µg/m^3 change in the SO_2 concentration.

Being the result of a meta-analyses, the confidence in the derived RRs is significant, and INFOTOX thus assesses SO_2 health risks with the more conservative of the two RRs derived by Zheng and collaborators. Thus, the RR of 1.011, is used to calculated the risks of asthma exacerbation associated with short-term exposure to SO_2.

10.7 Assessments of NO_2 health effects

10.7.1 Causality assessments

Oxidised nitrogen-containing compounds transformed from NO and NO_2 are referred to as “oxides of nitrogen” or “nitrogen oxides”, abbreviated as NO_X. Most studies on the health effects of gaseous NO_X focus on NO_2.

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 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 10.7.1.1 and 10.7.1.2.

[1] CI: Confidence interval of the RR central estimate

Table 10.7.1.1: 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

Table 10.7.1.2: 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 REVIHAAP report compiled by the WHO (2013a) (see detail in Section 4.1) provided evidence for the causality of health effects of exposure to NO_2. For the purposes of this study, only those exposure-outcome pairs for which reliable concentration-response functions were recommended by the WHO (2013b) are included in the burden of disease assessment for the HHRA. A summary of these pairs is given in Table 10.7.1.3.

Table 10.7.1.3: WHO outcomes involving NO_2.

Health outcome
Short-term exposure – NO_2, daily maximum 1-hour mean
Mortality, all-cause, all ages
Hospital admissions, respiratory diseases, all ages
Short-term exposure – NO_2, 24-hour mean
Hospital admissions, respiratory diseases, all ages
Long-term exposure – NO_2, annual mean
None for which reliable concentration-response functions were recommended

Table 10.7.1.4 presents a comparison of the USEPA (2016) and the WHO (2013b) causality assessments for specific health endpoints associated with short-term exposure to NO_2. The causality assessments are aligned in general, but there are differences in finer detail. Long-term assessments are not compared, because the USEPA (2016) did not rate any of the effects associated with long-term exposure as causal or likely to be causal. The WHO also concluded that there is less certainty about the precision of the data used for quantification of long-term effects than short-term effects.

Table 10.7.1.4: Comparison of short-term USEPA and WHO causality assessments.

Health outcome	WHO (2013a and b)	USEPA (2016)
Respiratory effects:		
Asthma exacerbation	Not assessed	Causal
*ED visits and hospital admissions for respiratory diseases and infections, all ages	Reliable quantification based on daily maximum 1-hour mean and daily 24-hour mean	Consistent epidemiologic evidence for respiratory diseases, but uncertainty regarding confounding and exposure measurement error. Inconsistent epidemiologic evidence for respiratory infections and uncertainty regarding NO_2 independent effects
Mortality: all natural causes, all ages	Reliable quantification based on daily maximum 1-hour mean	Suggestive of, but not sufficient to infer a causal relationship

*ED: Emergency Department

In summary, the USEPA (2016) and the WHO (2013b) agreed that conclusive evidence exists of a causal relationship between short-term exposure to NO_2 and respiratory effects. The USEPA assessment concluded that consistent evidence from multiple, high-quality controlled human exposure studies indicated a causal relationship with asthma exacerbation (Table 6.2.4), but this health endpoint was not specifically assessed by the WHO. The WHO proposed reliable quantification of emergency department visits and hospital admissions for respiratory diseases and infections, but the USEPA declared uncertainty regarding these endpoints. Thus, only asthma exacerbation is assessed with regard to respiratory effects.

The causality assessments of total all-cause natural mortality associated with short-term exposure was not in agreement (Table 10.7.1.4); therefore, this health endpoint is not included in the HHRA.

The WHO (2013b) did not recommend reliable concentration-response functions for health endpoints associated with long-term exposure, and the USEPA (2016) did not regard endpoints associated with long-term exposure as causally related. Therefore, INFOTOX did not include chronic exposure to NO_2 in the risk assessment.

10.7.2 RRs for health risk calculations

The USEPA (2016) did not propose a summary RR for exacerbation of asthma associated with short-term exposure to NO_2 and the WHO had not proposed an RR for calculating the risk of asthma exacerbation. Therefore, a literature search for meta-analyses of the relationships between NO_2 exposure and asthma exacerbation was conducted. Table 10.7.2.1 lists RRs provided by these meta-analyses. Zheng et al. (2015b) had noted that the certainty of evidence was high for 24-hour-average NO_2 and low for 1-hour average concentrations. Thus, INFOTOX risk calculations use 24-hour average NO_2 concentrations. Only Zheng et al. (2015b) specified the RR as based on 24-hour average NO_2 concentrations, thus, the RR of 1.014 per 10 µg/m^3 increase is used for the HHRA.

Table 10.7.2.1: RR for exacerbation of asthma associated with short-term exposure to NO_2.

Reference	Health endpoints	RR per 10 µg/m^3 increase	Short-term averaging time
Huang et al. (2021)	Asthma exacerbations associated with outpatient visits, *ER visits, hospitalizations, deaths, or other events (symptoms, lung function changes, and medication use)	1.030	Not reported
Zheng et al. (2015a)	Asthma-related ER visits and hospitalizations (time-series studies)	1.018	Varying, up to 7 days
Zheng et al. (2015b)	Asthma-related ER visits and hospitalizations	1.014	24-hours

* ER: Emergency Room

10.8 HHRA for carbon monoxide

10.8.1 Causality assessments

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 US Agency for Toxic Substances and Disease Registry (ATSDR) compiled a toxicological profile for CO (ATSDR 2012) and concluded that growing evidence over the last decade had revealed that endogenously produced CO is a cell signalling agent. As a cell 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. The acute toxicity effects of high concentrations of exogenous CO are well known. 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 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. 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).

More convincing evidence is available of an impact on the cardiovascular system. The USEPA reviewed a significant body of epidemiological, human clinical and animal toxicological 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 2010). 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)[1], for congestive heart failure (CHF)[2] and for cardiovascular diseases (CVD)[3] as a group. Epidemiologic studies consistently show associations with HAs and ED visits for myocardial infarction (MI)[4] and angina[5], but associations with stroke were not consistent. 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.

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 conclude that a causal relationship exists. The evidence on long-term exposure and respiratory morbidity was also inadequate to infer a causal relationship.

Epidemiologic studies of CO exposure during pregnancy (long-term exposure) provide some evidence of an association with detrimental birth outcomes including increased risk of preterm birth (PTB), 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).

Tables 10.8.1.1 and 10.8.1.2 present summaries of the USEPA causality determination of health effects related to CO.

[1] Heart problems caused by narrowed heart arteries, which result in decreased blood flow and oxygen to the heart muscle.

[2] 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.

[3] Disease of the heart and/or blood vessels, causing decreased blood flow to the heart, brain or body.

[4] 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.

[5] Chest pain or discomfort caused by an inadequate supply of oxygen-rich blood to the heart muscle.

Table 10.8.1.1: USEPA causality determination of short-term CO exposure.

Health endpoint	Causality determination	Likely outcome of exposure
Cardiovascular effects	Likely to be causal	Diseases of the heart and/or vascular system.
		Increased ED* visits and hospitalisation for: • cardiovascular diseases as a group • ischemic heart disease, e.g. CHD** • congestive heart failure • myocardial infarction • angina
Central nervous system effects	Suggestive	• Impairment of neural function • Impaired memory and learning
Respiratory effects	Suggestive	• Impaired pulmonary function • Respiratory symptoms • Increased medication use for respiratory symptoms • Increased ED visits and hospitalisation for upper and lower respiratory tract symptoms
Mortality	Suggestive	Total (non-accidental) and cardiovascular mortality

* ED: Emergency department visits

** CHD: Coronary heart disease: plaque builds up inside the coronary arteries that supply oxygen-rich blood to the heart muscle.

Table 10.8.1.2: USEPA causality determination of long-term CO exposure.

Health endpoint	Causality determination	Likely outcome of exposure
Cardiovascular effects	Inadequate	Diseases of the heart and/or vascular system
Central nervous system effects	Suggestive	• Impairment of neural function • Altered behaviour, impaired memory and learning
Developmental effects	Suggestive	• Risk of preterm birth (PTB) • cardiac birth defects • small reductions in birth weight • infant mortality in the post-neonatal period
Respiratory effects	Inadequate	• Pulmonary function • exacerbation of asthma symptoms
Mortality	Unlikely	Total (non-accidental) mortality

10.8.2 RRs for health risk calculations

The ATSDR (2012) has concluded that, although there may be a level of CO exposure that can be tolerated with minimal risk of adverse effects, the currently available toxicological and epidemiological data does 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.

Epidemiological studies of the relationships between short-term exposure to CO and observed cardiovascular health effects provide estimates of the risk associated with particular levels of exposure. Risk factors for health endpoints other than cardiovascular disease and for exposure periods other than the short term are not given, since these were not causally related to CO exposure (Tables 10.8.1.1 and 10.8.1.2). The USEPA standardised the risk factors to incremental increases in the hourly maximum, the 8-hourly maximum and the 24-h average CO concentration.

INFOTOX chose to use the modelled 8-hourly maximum CO concentration, since concentrations modelled over 8-hour periods are likely to vary on a smaller scale than hourly concentrations, resulting in more stable estimates of potential health risks. The advantage over using 24-hour averaged concentrations is that the expected variability in ambient CO concentrations and the associated potential risks are captured more accurately with the 8-hour maximum concentrations.

Lee et al. (2020) conducted a systematic review and meta-analysis of the updated epidemiological literature studying associations between short-term exposure (in the order of hours up to 7 days) to ambient air CO and hospital admissions or mortality due to myocardial infarction. Overall, myocardial infarction was associated with exposure to ambient CO with a risk ratio of 1.052 per 1 mg/m^3 increase in CO concentration (95 % confidence interval of 1.017 to 1.089). Table 11.8.2.1 lists the risk factors for short-term exposures to CO, derived from the USEPA (2010) and Lee et al. (2020).

Table 10.8.2.1: Summary of short-term CO RRs for health risk assessment.

Standardised RR*	
Health effect	**RR per standardised increase***
Coronary heart disease HAs**: all ages	1.020
Cardiovascular disease (other than stroke, but not specified) HAs** : all ages	1.025
Myocardial infarction HAs**: adults (18 years and older)	1.052

* Standardised to a 0.86 mg/m^3 increase in 8-h maximum.
** HAs: hospital admissions

10.9 References to Annexure 1

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11 Annexure 2: Modelled concentration isopleths

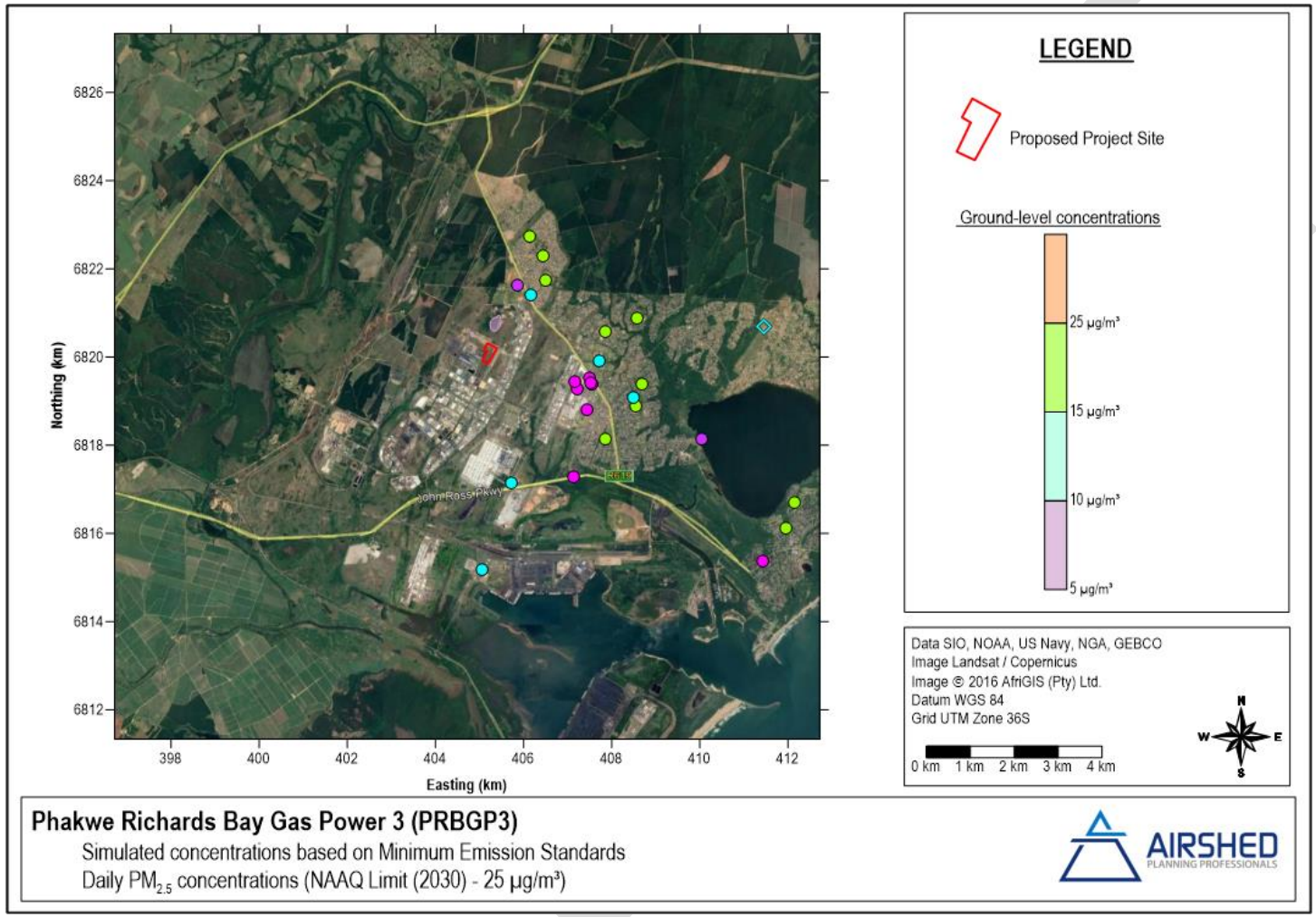

Figure 11.1: Simulated daily PM2.5 concentrations in the operational phase.

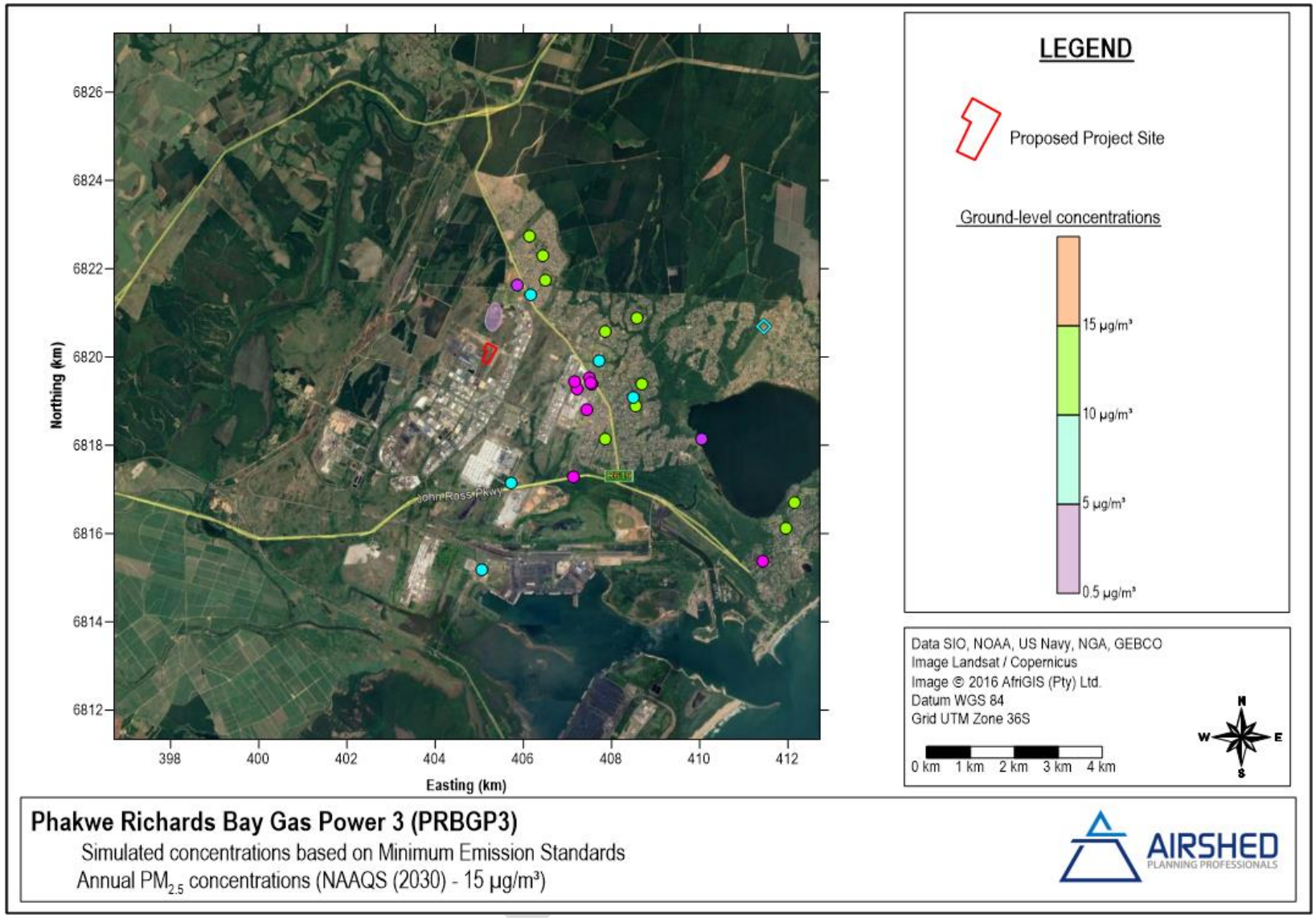

Figure 11.2: Simulated annual average PM2.5 concentrations in the operational phase.

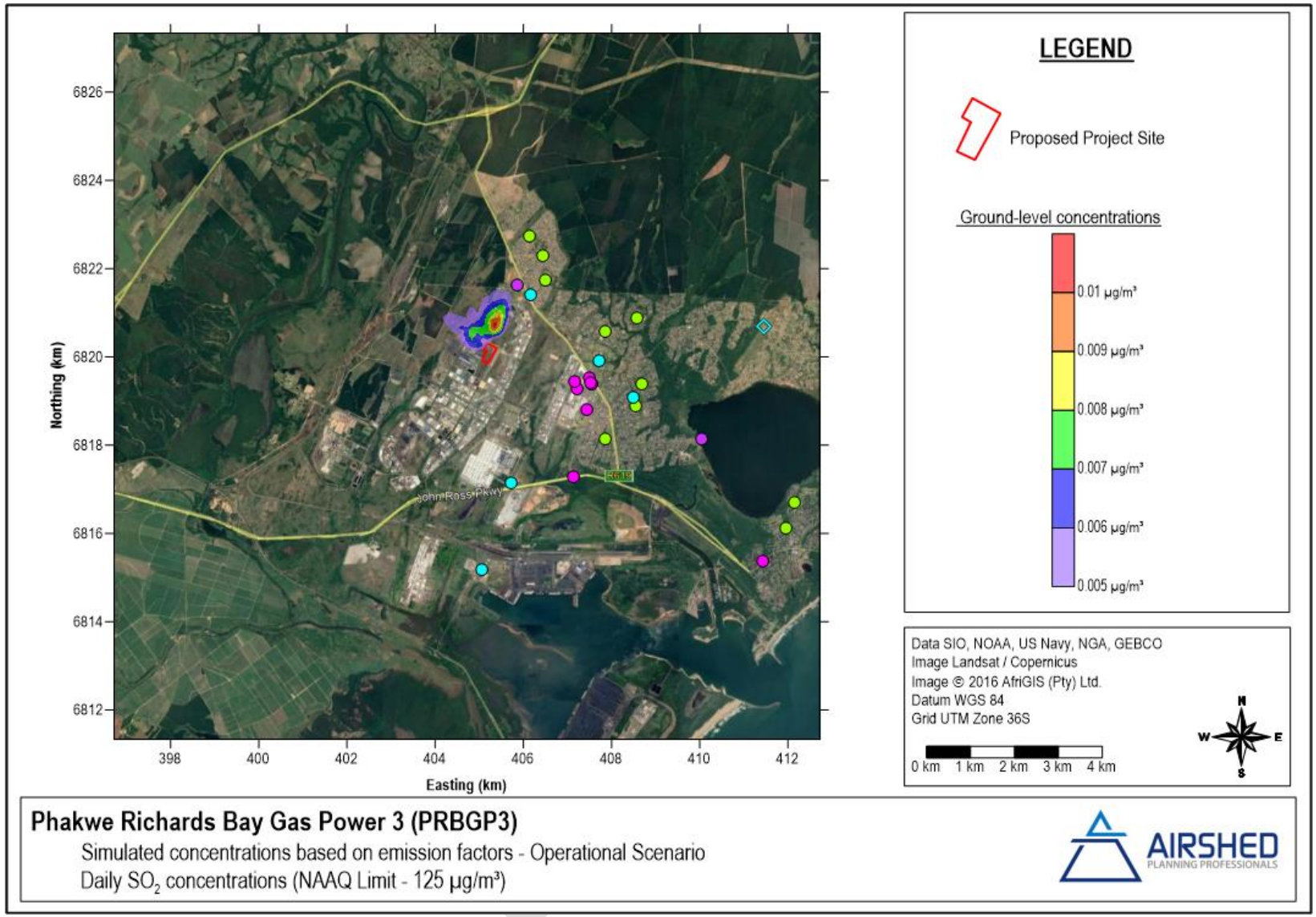

Figure 11.3: Simulated daily SO_2 concentrations in the operational phase.

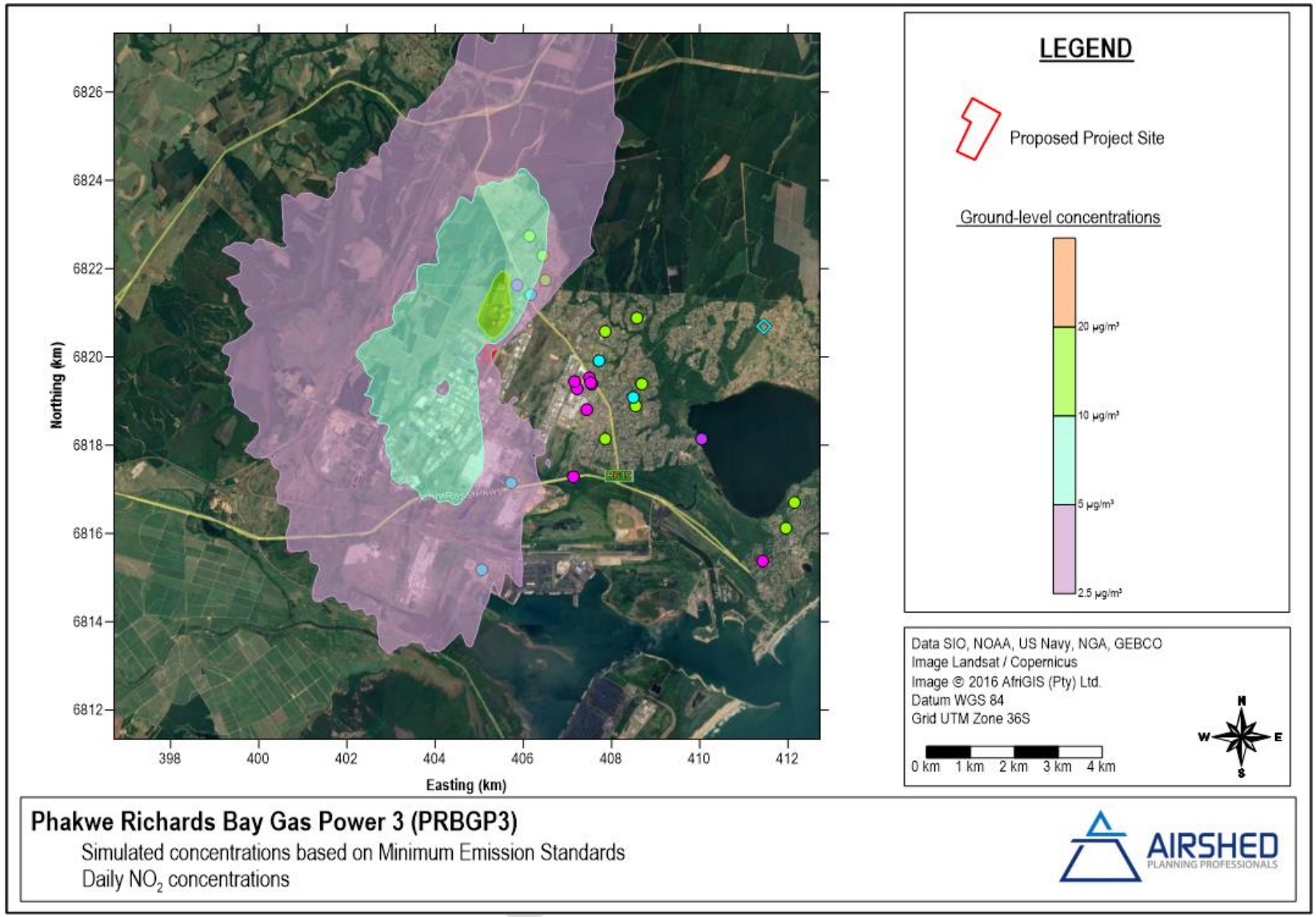

Figure 11.4: Simulated daily NO_2 concentrations in the operational phase.

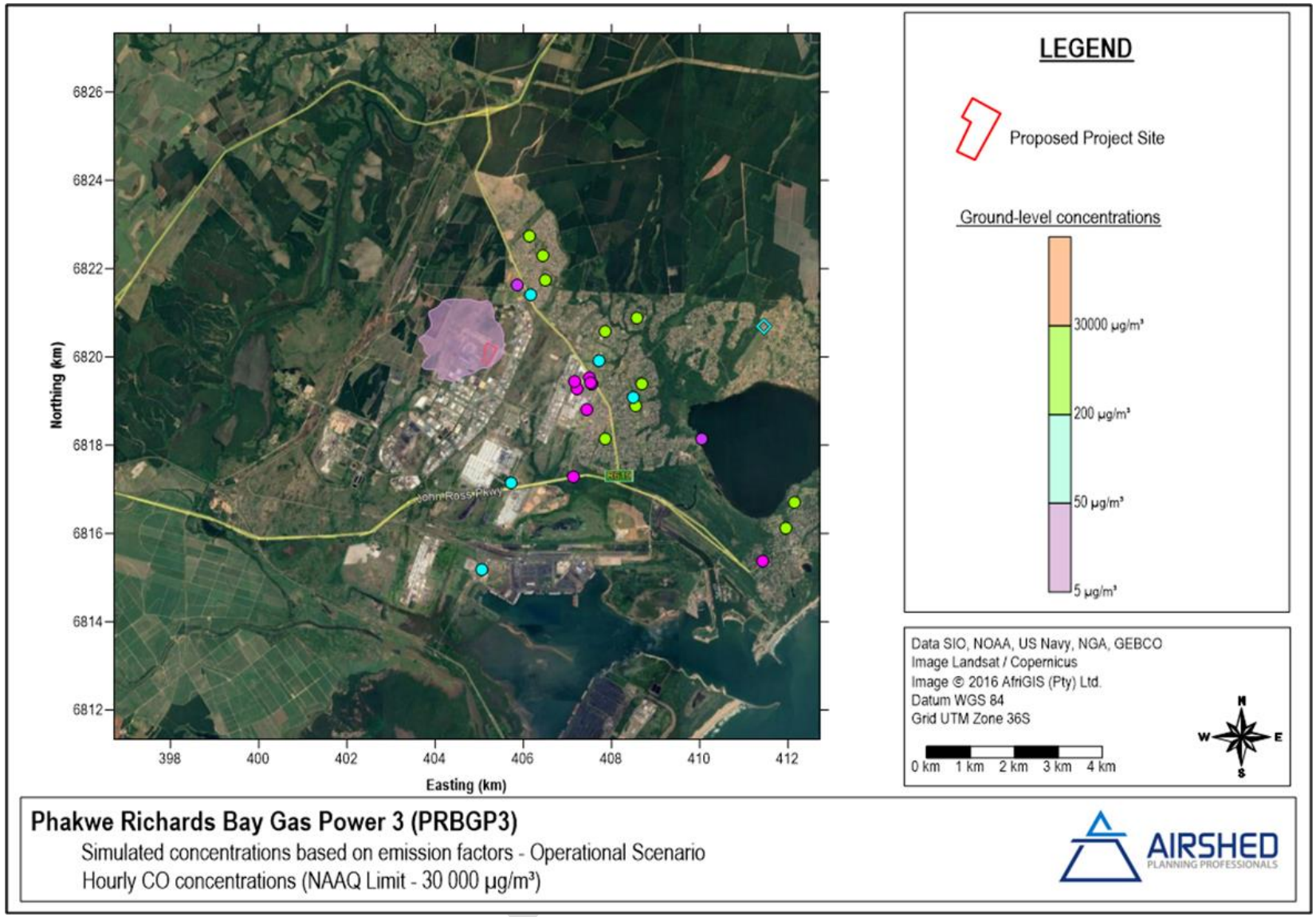

Figure 11.5: Simulated hourly average CO concentrations in the operational phase.

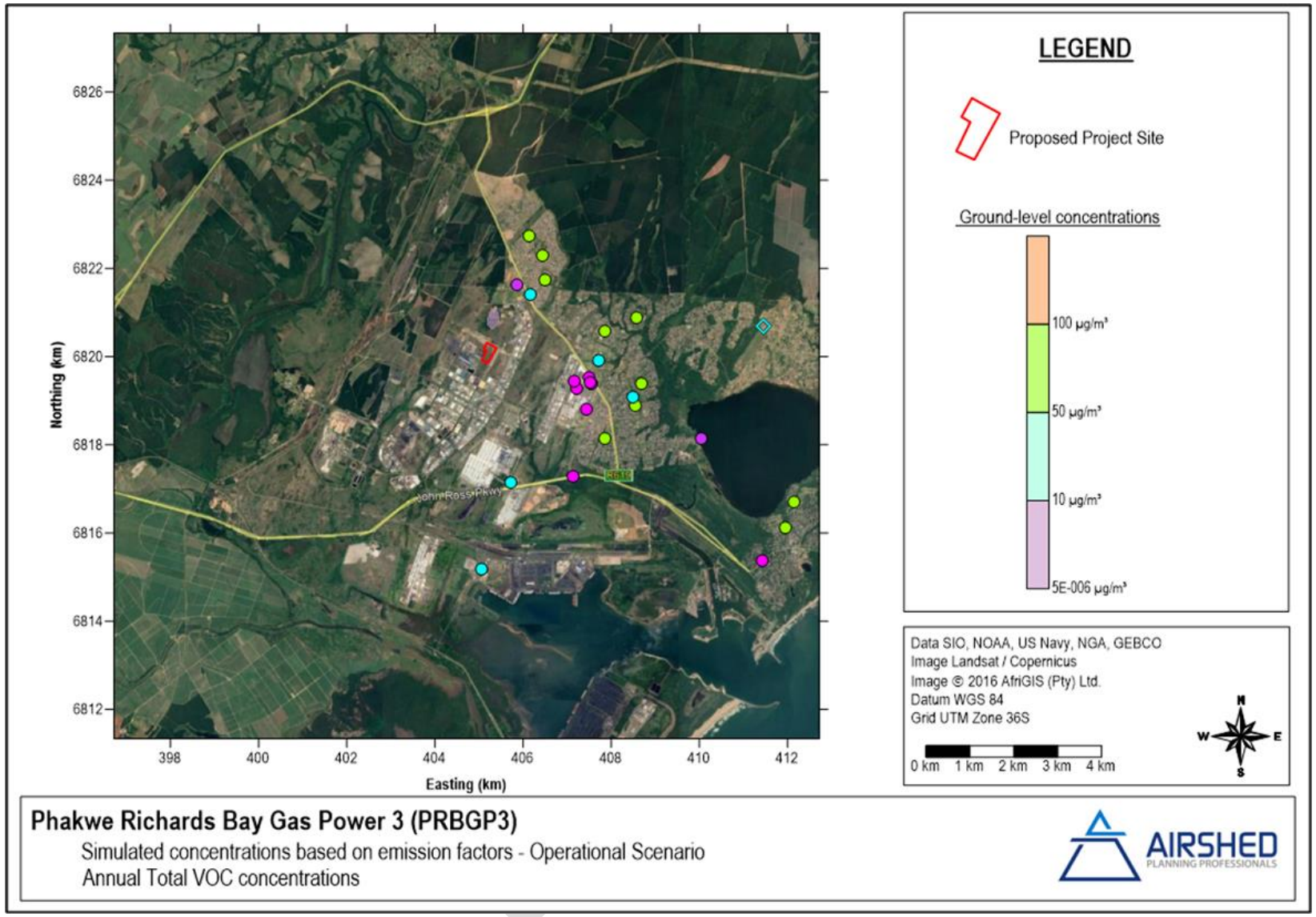

Figure 11.6: Simulated annual average total VOC concentrations - operational phase.

12 Annexure 3: Criteria pollutant health risk calculations results

Table 12.1: AFs of mortality and hospital admissions associated with short-term exposure to PM2.5.

Receptor name	Mortality	Cardiovascular hospitalisation	Respiratory hospitalisation
Richards Bay Christian School	7.2E-04	7.1E-04	7.9E-04
Richards Bay Secondary School	5.2E-04	5.2E-04	5.7E-04
Richards Bay Primary School	5.6E-04	5.5E-04	6.1E-04
Richardsbaai Hoërskool	5.9E-04	5.9E-04	6.5E-04
Veldenvlei Primary School	9.0E-04	8.9E-04	9.9E-04
Arboretum Primary School	4.7E-04	4.6E-04	5.1E-04
Bay Primary School	7.5E-04	7.4E-04	8.2E-04
Brackenham Primary School	4.8E-04	4.8E-04	5.3E-04
John Ross College	5.1E-04	5.0E-04	5.6E-04
St Francis Pre-Primary School	7.2E-04	7.1E-04	7.9E-04
Empangeni High School	7.2E-04	7.1E-04	7.9E-04
Phesheya Primary School	4.2E-04	4.1E-04	4.6E-04
Old Mill High School	5.4E-04	5.3E-04	5.9E-04
Pinocchio Pre-Primary School	4.8E-04	4.8E-04	5.3E-04
Empangeni Christian School	5.2E-04	5.2E-04	5.8E-04
St Catherine's High School	5.1E-04	5.0E-04	5.6E-04
Empangeni Preparatory School	4.8E-04	4.7E-04	5.2E-04
Heuwelland Primary School	4.7E-04	4.6E-04	5.1E-04
Thuthukani Special School	5.2E-04	5.2E-04	5.7E-04
Felixton College	5.0E-04	5.0E-04	5.5E-04
Tisand Technical High School	5.1E-04	5.0E-04	5.6E-04
Hlamvana Secondary School	5.2E-04	5.1E-04	5.7E-04
Thanduyise High School	4.4E-04	4.3E-04	4.8E-04
Ilembe Primary School	5.1E-04	5.1E-04	5.6E-04
Imizikayifani Primary School	5.2E-04	5.1E-04	5.7E-04
Khula High School	5.5E-04	5.5E-04	6.1E-04
Umdlamfe Secondary School	5.1E-04	5.1E-04	5.6E-04
Bajabulile Primary School	5.7E-04	5.7E-04	6.3E-04
Thambolini High School	5.6E-04	5.5E-04	6.1E-04
Dlamvuzo Secondary School	5.4E-04	5.4E-04	5.9E-04
University of Zululand	4.3E-04	4.2E-04	4.7E-04
Khandisa Primary School	4.0E-04	4.0E-04	4.4E-04

Receptor name	Mortality	Cardiovascular hospitalisation	Respiratory hospitalisation
Dlangezwa High School	4.1E-04	4.0E-04	4.5E-04
Ongoye Secondary School	4.0E-04	4.0E-04	4.4E-04
Matshangule Primary School	4.1E-04	4.0E-04	4.5E-04
Kwavulindlela Primary School	4.0E-04	3.9E-04	4.4E-04
Qambokwethu Primary School	3.4E-04	3.4E-04	3.8E-04
Mkhobosa Primary School	6.3E-04	6.2E-04	6.9E-04
Tholokuhle Secondary School	5.9E-04	5.9E-04	6.5E-04
Vondlo Primary School	5.8E-04	5.8E-04	6.4E-04
Sinaye Primary School	5.9E-04	5.9E-04	6.5E-04
Kwambonambi Primary School	4.9E-04	4.8E-04	5.4E-04
Nseleni - Community Health Care	6.3E-04	6.2E-04	6.9E-04
Mens Clinic International - Richards Bay	8.6E-04	8.5E-04	9.4E-04
Richards Bay Municipal Clinic	9.3E-04	9.2E-04	1.0E-03
The Bay Hospital	7.7E-04	7.6E-04	8.4E-04
Better2Know Private STD Health Centre Richards Bay	8.6E-04	8.5E-04	9.5E-04
Headache Clinic | Bay Chiropractic | Smile Dent	4.9E-04	4.8E-04	5.4E-04
Umhlathuze Dental	9.0E-04	8.9E-04	9.9E-04
Mandlazini Clinic	7.6E-04	7.5E-04	8.3E-04
Mondi Felixton - Clinic	4.9E-04	4.9E-04	5.4E-04
Pietermaritzburg Medi Clinic	4.6E-04	4.5E-04	5.0E-04
Hope Clinic	4.6E-04	4.6E-04	5.1E-04
Isiboniso Clinic	5.8E-04	5.7E-04	6.3E-04
Better2Know Private STD Health Centre Empangeni	4.5E-04	4.5E-04	5.0E-04
Blue Ladies Clinic	4.7E-04	4.6E-04	5.1E-04
Life Empangeni Garden Clinic	4.5E-04	4.5E-04	5.0E-04
Ngwelezana Hospital	4.6E-04	4.5E-04	5.0E-04
Lower Umfolozi District War Memorial Hospital - Paediatric Ward	4.7E-04	4.6E-04	5.1E-04
Ngwelezana clinic	4.4E-04	4.4E-04	4.8E-04
Richardsbay Medical Institute	7.7E-04	7.6E-04	8.4E-04
Bethlehem recovery centre Empangeni.	4.5E-04	4.5E-04	4.9E-04
Sinalo Cerebral Palsy Centre	4.7E-04	4.7E-04	5.2E-04
Ethembeni Care Centre	5.2E-04	5.1E-04	5.7E-04
Esikhawini	5.4E-04	5.4E-04	6.0E-04
Aquadene	1.3E-03	1.3E-03	1.5E-03
Wild En Weide	7.7E-04	7.6E-04	8.4E-04
Richards Bay Central	9.1E-04	9.0E-04	1.0E-03
Arboretum	7.5E-04	7.4E-04	8.2E-04
Birdswood	6.3E-04	6.2E-04	6.9E-04

Receptor name	Mortality	Cardiovascular hospitalisation	Respiratory hospitalisation
Richards bay - New	5.2E-04	5.2E-04	5.7E-04
Meer en See	4.5E-04	4.5E-04	5.0E-04
Ntshingimipisi	4.0E-04	3.9E-04	4.4E-04
Nzalabantu	3.3E-04	3.3E-04	3.6E-04
Vulindela A	4.0E-04	4.0E-04	4.4E-04
Felixton	5.0E-04	4.9E-04	5.5E-04
Eniwe	4.5E-04	4.4E-04	4.9E-04
Hillview	4.4E-04	4.3E-04	4.8E-04
Empangeni	4.8E-04	4.8E-04	5.3E-04
Dondolo	4.3E-04	4.3E-04	4.7E-04
Ngwelezana B	4.3E-04	4.3E-04	4.7E-04
Nseleni A	5.8E-04	5.8E-04	6.4E-04
Matshana	3.9E-04	3.9E-04	4.3E-04
Airport (RBCAA)	6.0E-04	6.0E-04	6.6E-04
Arboretum (RBCAA)	7.2E-04	7.1E-04	7.9E-04
Brackenham (RBCAA)	1.5E-03	1.5E-03	1.6E-03
CBD (RBCAA)	6.5E-04	6.4E-04	7.1E-04
Esikhawini (RBCAA)	5.1E-04	5.0E-04	5.6E-04
Felixton (RBCAA)	5.0E-04	5.0E-04	5.5E-04
Harbour West (RBCAA)	9.9E-04	9.8E-04	1.1E-03
Scorpio (RBCAA)	1.3E-03	1.3E-03	1.4E-03
eNseleni (RBCAA)	5.5E-04	5.4E-04	6.0E-04
Brackenham (uMhlathuze)	2.0E-03	2.0E-03	2.2E-03
Arboretum (uMhlathuze)	5.2E-04	5.1E-04	5.7E-04
eSikhaleni (uMhlathuze)	5.3E-04	5.3E-04	5.8E-04

Table 12.2: AFs of mortality and hospital admissions associated with long-term exposure to PM2.5.

Receptor name	All-cause (natural) mortality age 30+	Asthma incidence, ages 4 to 17
Richards Bay Christian School	2.8E-04	6.9E-05
Richards Bay Secondary School	2.9E-04	7.3E-05
Richards Bay Primary School	3.7E-04	9.2E-05
Richardsbaai Hoërskool	6.6E-04	1.6E-04
Veldenvlei Primary School	3.0E-04	7.5E-05
Arboretum Primary School	3.2E-04	7.9E-05
Bay Primary School	2.5E-04	6.2E-05
Brackenham Primary School	3.1E-04	7.6E-05
John Ross College	3.7E-04	9.2E-05

Receptor name	All-cause (natural) mortality age 30+	Asthma incidence, ages 4 to 17
St Francis Pre-Primary School	2.8E-04	6.9E-05
Empangeni High School	2.8E-04	6.9E-05
Phesheya Primary School	2.6E-04	6.4E-05
Old Mill High School	3.5E-04	8.6E-05
Pinocchio Pre-Primary School	3.1E-04	7.7E-05
Empangeni Christian School	3.3E-04	8.1E-05
St Catherine's High School	3.2E-04	7.9E-05
Empangeni Preparatory School	3.1E-04	7.7E-05
Heuwelland Primary School	3.0E-04	7.4E-05
Thuthukani Special School	2.9E-04	7.3E-05
Felixton College	2.9E-04	7.3E-05
Tisand Technical High School	3.8E-04	9.3E-05
Hlamvana Secondary School	4.0E-04	9.8E-05
Thanduyise High School	2.7E-04	6.6E-05
Ilembe Primary School	3.8E-04	9.5E-05
Imizikayifani Primary School	3.6E-04	8.9E-05
Khula High School	4.0E-04	1.0E-04
Umdlamfe Secondary School	3.5E-04	8.7E-05
Bajabulile Primary School	4.3E-04	1.1E-04
Thambolini High School	3.7E-04	9.2E-05
Dlamvuzo Secondary School	3.4E-04	8.4E-05
University of Zululand	2.4E-04	5.9E-05
Khandisa Primary School	2.3E-04	5.6E-05
Dlangezwa High School	2.3E-04	5.7E-05
Ongoye Secondary School	2.3E-04	5.6E-05
Matshangule Primary School	2.3E-04	5.6E-05
Kwavulindlela Primary School	2.3E-04	5.6E-05
Qambokwethu Primary School	2.0E-04	5.0E-05
Mkhobosa Primary School	5.0E-04	1.2E-04
Tholokuhle Secondary School	6.6E-04	1.6E-04
Vondlo Primary School	6.6E-04	1.6E-04
Sinaye Primary School	6.7E-04	1.7E-04
Kwambonambi Primary School	4.8E-04	1.2E-04
Nseleni - Community Health Care	7.2E-04	1.8E-04
Mens Clinic International - Richards Bay	3.0E-04	7.5E-05
Richards Bay Municipal Clinic	3.3E-04	8.1E-05
The Bay Hospital	2.9E-04	7.3E-05
Better2Know Private STD Health Centre Richards Bay	3.1E-04	7.6E-05

Receptor name	All-cause (natural) mortality age 30+	Asthma incidence, ages 4 to 17
Headache Clinic | Bay Chiropractic | Smile Dent	1.8E-04	4.5E-05
Umhlathuze Dental	3.0E-04	7.5E-05
Mandlazini Clinic	3.0E-04	7.4E-05
Mondi Felixton - Clinic	2.9E-04	7.2E-05
Pietermaritzburg Medi Clinic	3.1E-04	7.6E-05
Hope Clinic	3.2E-04	7.9E-05
Isiboniso Clinic	4.8E-04	1.2E-04
Better2Know Private STD Health Centre Empangeni	3.1E-04	7.7E-05
Blue Ladies Clinic	3.2E-04	7.8E-05
Life Empangeni Garden Clinic	3.1E-04	7.8E-05
Ngwelezana Hospital	2.8E-04	6.9E-05
Lower Umfolozi District War Memorial Hospital - Paediatric Ward	3.2E-04	7.9E-05
Ngwelezana clinic	2.7E-04	6.7E-05
Richardsbay Medical Institute	3.0E-04	7.3E-05
Bethlehem recovery centre Empangeni.	3.1E-04	7.6E-05
Sinalo Cerebral Palsy Centre	3.1E-04	7.7E-05
Ethembeni Care Centre	4.8E-04	1.2E-04
Esikhawini	4.0E-04	9.9E-05
Aquadene	1.7E-03	4.3E-04
Wild En Weide	4.1E-04	1.0E-04
Richards Bay Central	3.3E-04	8.2E-05
Arboretum	2.5E-04	6.2E-05
Birdswood	2.3E-04	5.7E-05
Richards bay - New	1.8E-04	4.4E-05
Meer en See	1.5E-04	3.8E-05
Ntshingimipisi	1.5E-04	3.7E-05
Nzalabantu	1.3E-04	3.2E-05
Vulindela A	2.3E-04	5.6E-05
Felixton	2.9E-04	7.2E-05
Eniwe	2.7E-04	6.6E-05
Hillview	2.9E-04	7.2E-05
Empangeni	3.1E-04	7.6E-05
Dondolo	2.7E-04	6.6E-05
Ngwelezana B	2.7E-04	6.6E-05
Nseleni A	6.7E-04	1.7E-04
Matshana	2.4E-04	5.8E-05
Airport (RBCAA)	2.1E-04	5.1E-05
Arboretum (RBCAA)	1.1E-03	2.8E-04

Receptor name	All-cause (natural) mortality age 30+	Asthma incidence, ages 4 to 17
Brackenham (RBCAA)	1.2E-03	3.0E-04
CBD (RBCAA)	3.1E-04	7.6E-05
Esikhawini (RBCAA)	3.7E-04	9.2E-05
Felixton (RBCAA)	2.9E-04	7.2E-05
Harbour West (RBCAA)	1.9E-04	4.7E-05
Scorpio (RBCAA)	6.4E-04	1.6E-04
eNseleni (RBCAA)	6.1E-04	1.5E-04
Brackenham (uMhlathuze)	2.2E-03	5.5E-04
Arboretum (uMhlathuze)	2.0E-04	4.9E-05
eSikhaleni (uMhlathuze)	3.7E-04	9.2E-05

Table 12.3: AFs of asthma exacerbation associated with short-term exposure to SO_2.

Receptor name	AF (unitless)
Richards Bay Christian School	1.5E-02
Richards Bay Secondary School	8.1E-03
Richards Bay Primary School	7.3E-03
Richardsbaai Hoërskool	2.0E-02
Veldenvlei Primary School	1.8E-02
Arboretum Primary School	9.2E-03
Bay Primary School	1.5E-02
Brackenham Primary School	8.6E-03
John Ross College	7.2E-03
St Francis Pre-Primary School	1.5E-02
Empangeni High School	1.5E-02
Phesheya Primary School	6.2E-03
Old Mill High School	1.1E-02
Pinocchio Pre-Primary School	8.5E-03
Empangeni Christian School	8.5E-03
St Catherine's High School	8.1E-03
Empangeni Preparatory School	7.9E-03
Heuwelland Primary School	7.6E-03
Thuthukani Special School	8.1E-03
Felixton College	7.5E-03
Tisand Technical High School	7.2E-03
Hlamvana Secondary School	7.5E-03
Thanduyise High School	6.7E-03
Ilembe Primary School	7.4E-03
Imizikayifani Primary School	7.0E-03

Receptor name	AF (unitless)
Khula High School	7.6E-03
Umdlamfe Secondary School	6.9E-03
Bajabulile Primary School	7.8E-03
Thambolini High School	7.3E-03
Dlamvuzo Secondary School	7.0E-03
University of Zululand	5.6E-03
Khandisa Primary School	5.1E-03
Dlangezwa High School	5.1E-03
Ongoye Secondary School	5.1E-03
Matshangule Primary School	5.1E-03
Kwavulindlela Primary School	5.1E-03
Qambokwethu Primary School	4.5E-03
Mkhobosa Primary School	9.9E-03
Tholokuhle Secondary School	2.0E-02
Vondlo Primary School	1.8E-02
Sinaye Primary School	2.1E-02
Kwambonambi Primary School	1.2E-02
Nseleni - Community Health Care	2.0E-02
Mens Clinic International - Richards Bay	2.1E-02
Richards Bay Municipal Clinic	2.0E-02
The Bay Hospital	2.1E-02
Better2Know Private STD Health Centre Richards Bay	2.2E-02
Headache Clinic | Bay Chiropractic | Smile Dent	8.1E-03
Umhlathuze Dental	1.8E-02
Mandlazini Clinic	1.9E-02
Mondi Felixton - Clinic	7.3E-03
Pietermaritzburg Medi Clinic	7.7E-03
Hope Clinic	8.0E-03
Isiboniso Clinic	1.1E-02
Better2Know Private STD Health Centre Empangeni	8.7E-03
Blue Ladies Clinic	7.9E-03
Life Empangeni Garden Clinic	8.5E-03
Ngwelezana Hospital	7.3E-03
Lower Umfolozi District War Memorial Hospital - Paediatric Ward	9.2E-03
Ngwelezana clinic	6.5E-03
Richardsbay Medical Institute	2.1E-02
Bethlehem recovery centre Empangeni.	8.5E-03
Sinalo Cerebral Palsy Centre	7.8E-03

Receptor name	AF (unitless)
Ethembeni Care Centre	1.2E-02
Esikhawini	7.5E-03
Aquadene	5.3E-02
Wild En Weide	2.2E-02
Richards Bay Central	2.0E-02
Arboretum	1.5E-02
Birdswood	1.2E-02
Richards bay - New	7.6E-03
Meer en See	6.6E-03
Ntshingimipisi	5.9E-03
Nzalabantu	6.2E-03
Vulindela A	5.0E-03
Felixton	7.3E-03
Eniwe	6.2E-03
Hillview	7.7E-03
Empangeni	8.6E-03
Dondolo	6.6E-03
Ngwelezana B	6.4E-03
Nseleni A	2.1E-02
Matshana	5.4E-03
Airport (RBCAA)	1.0E-02
Arboretum (RBCAA)	1.5E-02
Brackenham (RBCAA)	5.0E-02
CBD (RBCAA)	1.6E-02
Esikhawini (RBCAA)	7.2E-03
Felixton (RBCAA)	7.5E-03
Harbour West (RBCAA)	3.0E-02
Scorpio (RBCAA)	3.7E-02
eNseleni (RBCAA)	1.8E-02
Brackenham (uMhlathuze)	8.4E-02
Arboretum (uMhlathuze)	1.1E-02
eSikhaleni (uMhlathuze)	7.3E-03

Table 12.4: AFs of asthma-related emergency visits and hospitalisation associated with short-term exposure to NO_2.

Receptor name	AF (unitless)
Richards Bay Christian School	2.05E-03
Richards Bay Secondary School	1.19E-03
Richards Bay Primary School	1.00E-03

Receptor name	AF (unitless)
Richardsbaai Hoërskool	3.13E-03
Veldenvlei Primary School	2.70E-03
Arboretum Primary School	1.30E-03
Bay Primary School	2.10E-03
Brackenham Primary School	1.26E-03
John Ross College	1.00E-03
St Francis Pre-Primary School	2.05E-03
Empangeni High School	2.05E-03
Phesheya Primary School	8.25E-04
Old Mill High School	1.58E-03
Pinocchio Pre-Primary School	1.23E-03
Empangeni Christian School	1.28E-03
St Catherine's High School	1.25E-03
Empangeni Preparatory School	1.17E-03
Heuwelland Primary School	1.09E-03
Thuthukani Special School	1.19E-03
Felixton College	1.10E-03
Tisand Technical High School	1.01E-03
Hlamvana Secondary School	1.11E-03
Thanduyise High School	8.57E-04
Ilembe Primary School	1.03E-03
Imizikayifani Primary School	9.60E-04
Khula High School	1.13E-03
Umdlamfe Secondary School	9.54E-04
Bajabulile Primary School	1.16E-03
Thambolini High School	2.55E-03
Dlamvuzo Secondary School	3.95E-03
University of Zululand	5.34E-03
Khandisa Primary School	6.73E-03
Dlangezwa High School	8.12E-03
Ongoye Secondary School	9.51E-03
Matshangule Primary School	1.09E-02
Kwavulindlela Primary School	1.23E-02
Qambokwethu Primary School	1.37E-02
Mkhobosa Primary School	1.50E-02
Tholokuhle Secondary School	1.64E-02
Vondlo Primary School	1.78E-02
Sinaye Primary School	1.92E-02

Receptor name	AF (unitless)
Kwambonambi Primary School	2.05E-02
Nseleni - Community Health Care	2.19E-02
Mens Clinic International - Richards Bay	2.33E-02
Richards Bay Municipal Clinic	2.46E-02
The Bay Hospital	2.60E-02
Better2Know Private STD Health Centre Richards Bay	2.74E-02
Headache Clinic | Bay Chiropractic | Smile Dent	2.87E-02
Umhlathuze Dental	3.01E-02
Mandlazini Clinic	3.15E-02
Mondi Felixton - Clinic	3.28E-02
Pietermaritzburg Medi Clinic	3.42E-02
Hope Clinic	3.55E-02
Isiboniso Clinic	3.69E-02
Better2Know Private STD Health Centre Empangeni	3.82E-02
Blue Ladies Clinic	3.96E-02
Life Empangeni Garden Clinic	4.09E-02
Ngwelezana Hospital	4.22E-02
Lower Umfolozi District War Memorial Hospital - Paediatric Ward	4.36E-02
Ngwelezana clinic	4.49E-02
Richardsbay Medical Institute	4.63E-02
Bethlehem recovery centre Empangeni.	4.76E-02
Sinalo Cerebral Palsy Centre	4.89E-02
Ethembeni Care Centre	5.02E-02
Esikhawini	5.16E-02
Aquadene	5.29E-02
Wild En Weide	5.42E-02
Richards Bay Central	5.56E-02
Arboretum	5.69E-02
Birdswood	5.82E-02
Richards bay - New	5.95E-02
Meer en See	6.08E-02
Ntshingimipisi	6.21E-02
Nzalabantu	6.35E-02
Vulindela A	6.48E-02
Felixton	6.61E-02
Eniwe	6.74E-02
Hillview	6.87E-02
Empangeni	7.00E-02

Receptor name	AF (unitless)
Dondolo	7.13E-02
Ngwelezana B	7.26E-02
Nseleni A	7.39E-02
Matshana	7.52E-02
Airport (RBCAA)	7.65E-02
Arboretum (RBCAA)	7.78E-02
Brackenham (RBCAA)	7.91E-02
CBD (RBCAA)	8.03E-02
Esikhawini (RBCAA)	8.16E-02
Felixton (RBCAA)	8.29E-02
Harbour West (RBCAA)	8.42E-02
Scorpio (RBCAA)	8.55E-02
eNseleni (RBCAA)	8.68E-02
Brackenham (uMhlathuze)	8.80E-02
Arboretum (uMhlathuze)	8.93E-02
eSikhaleni (uMhlathuze)	9.06E-02