

Appendix D16  
Kareerand Expansion Project: Human Health Risk and Impact  
Assessment  
- EnviroSim Consulting, 2020





**EnviroSim**  
CONSULTING



**Kareerand Expansion Project:  
Human Health Risk and Impact  
Assessment**



Nardus@envirosim.co.za

### Report Details

Report Title: Kareerand Expansion Project: Human Health Risk and Impact Assessment

Report Reference Number: AQS01-2020

Revision: 1.0

Date: May 2020

Compiled by	Prepared For
<p>N Potgieter (EnviroSim Consulting) Msc. Chemistry UP Pr.Sci.Nat (Environmental Science)</p> 	<p>AquiSim Consulting (Pty) Ltd P.O. Box 51777 Wierda Park CENTURION 0149</p> <p>Attention: Japie van Blerk</p>

#### Statement of Experience

Nardus Potgieter is registered as a Professional Natural Scientist with the South African Council for Natural Scientific Professionals in Environmental Science field of practice. He has more than 14 years' experience in the assessment of impacts on human health and the environment from hazardous substances in air, water and the terrestrial food chain. His experience includes human health risk assessment, radiological public safety assessment and contaminated land remediation.

#### Declaration

EnviroSim Consulting is an independent consulting firm with no interest in the project other than to fulfil the contract between the environmental impact practitioner and the consultant for delivery of specialised services as stipulated in the terms of reference.

#### Disclaimer

EnviroSim Consulting exercises due care and diligence in rendering services and preparing documents. EnviroSim Consulting accepts no liability, and the client, by receiving this document, indemnifies EnviroSim Consulting and its manager, associates and employees against all actions, claims, demands, losses, liabilities, costs, damages and expenses arising from or in connection with services rendered, directly or indirectly by EnviroSim Consulting, and by the use of the information contained in this document.

## CROSS REFERENCE TABLE

Table prepared in accordance with National Environmental Management Act (Act 107 of 1998) (NEMA) Regulation (2014), Appendix 6

<b>Requirement</b>	<b>Relevant section in report</b>
Details of the specialist who prepared the report.	Report details (page ii)
The expertise of that person to compile a specialist report.	Report details (page ii)
A declaration that the person is independent in a form as may be specified by the competent authority.	Declaration (page v)
An indication of the scope of, and the purpose for which, the report was prepared.	Introduction Section 1.1 and 1.3
The date and season of the site investigation and the relevance of the season to the outcome of the assessment.	Not applicable
A description of the methodology adopted in preparing the report or carrying out the specialised process.	Section 1.2
The specific identified sensitivity of the site related to the activity and its associated structures and infrastructure.	Section 1.3
An identification of any areas to be avoided, including buffers.	Not applicable
A map superimposing the activity including the associated structures and infrastructure on the environmental sensitivities of the site including areas to be avoided, including buffers.	Section 1.1
A description of any assumptions made and any uncertainties or gaps in knowledge.	Section 6.4
A description of the findings and potential implications of such findings on the impact of the proposed activity, including identified alternatives, on the environment.	Section 5.6
Any mitigation measures for inclusion in the environmental management programme report	Section 5.6.4
Any conditions for inclusion in the environmental authorisation	Section 5.6.4
Any monitoring requirements for inclusion in the environmental management programme report or environmental authorisation.	Section 5.6.4
A reasoned opinion as to whether the proposed activity or portions thereof should be authorised.	Section 7
If the opinion is that the proposed activity or portions thereof should be authorised, any avoidance, management and mitigation measures that should be included in the environmental management programme report, and where applicable, the closure plan.	Section 7
A description of any consultation process that was undertaken during the course of carrying out the study.	Not applicable.
A summary and copies if any comments that were received during any consultation process.	No comments received.
Any other information requested by the competent authority.	Not applicable.

## EXECUTIVE SUMMARY

---

Mine Waste Solutions (MWS) is in the process of applying for the expansion of the Kareerand Tailings Storage Facility (TSF), with due considering of revised tailings production forecast rates and land ownership constraints.

The Kareerand TSF was designed with an operating life of 14 years, taking the operation of the facility to the year 2025, and having a total design capacity of 352 million tonnes. After commissioning of the TSF, MWS was acquired by AngloGold Ashanti (AGA) and the tailings production target has increased by an additional 485 million tonnes, which will require operations to continue until the year 2042. The additional tailings, therefore, require an extension of the design life of the Kareerand TSF.

The proposed Extension Project TSF will be located to the west of the existing Kareerand TSF and will cover an additional area of approximately 380 ha.

AquiSim Consulting (Pty) Ltd (AquiSim) approached EnviroSim Consulting on behalf of AGA with a request to perform an assessment of the potential impact on the health of communities, living in the vicinity of the proposed Project, with regard to exposure to airborne pollutants as well as contaminants identified as potentially relevant to groundwater and surface water resources in the area. The human health risk and impact assessment (HHRIA), is aimed at specifically addressing these concerns.

The health risks posed to members of the public by the activities planned as part of the proposed Extension Project, was evaluated using a source-pathway-receptor analysis approach. Information from specialist study reports were incorporated with toxicology data and population statistics to quantify the human health risks associated with the proposed Extension Project.

Information presented indicate that a complete source-pathway-receptor linkage exists for the atmospheric exposure pathway. Information on the aquatic environment, both surface- and groundwater, indicated that complete source-pathway-receptor linkage for this pathway may be possible, if proposed mitigative measures are not implemented. The aquatic pathway was therefore included in the further assessment. The potential for impacts relating to both the atmospheric and aquatic pathways was evaluated for the operational life of the proposed Extension Project. Impacts relating to construction and post-closure phases of the project are also addressed, albeit only qualitatively, as effluents and emissions associated with these phases specifically were not quantified through either the atmospheric dispersion modelling (Airshed, 2020) or contaminant transport modelling (GCS, 2020).

Using approaches developed by the United States Environmental Protection Agency (USEPA) and the World Health Organisation (WHO), the predicted airborne concentrations of air pollutants were assessed and the potential environmental human health risks associated with the proposed project was quantified. The following conclusions were reached:

- Based on the modelled air pollutant concentrations the proposed Extension Project TSF is shown to make a quantifiable but insignificant contribution to daily personal risks of health effects in members of the public.
- Annual risks of health effects from long term exposure to air pollutants were also evaluated. The evaluation similarly indicated a quantifiable but insignificant contribution to daily personal risks of health effects in members of the public.
- It is recommended that, in accordance with the findings presented in the Air Quality Specialist report (Airshed, 2020), that dust mitigation measures be implemented and airborne concentrations of PM<sub>10</sub> and PM<sub>2.5</sub> be monitored.
- The probability of non-cancer and cancer health effects occurring at any of the receptor locations as a result of exposure to airborne particulates is low and no mitigation or monitoring of these substances is considered necessary.
- Evaluation of measured baseline concentrations of ions and elements present in groundwater samples from the project area, indicate that ingestion exposure to the existing groundwater and is unlikely to result in adverse health effects to chronic water users. However, unless mitigation measures are implemented, deteriorating seepage water quality may contaminate water resources to a point where it is no longer fit for human consumption. It is recommended that seepage and runoff from the Kareerand and Extension Project TSFs be collected and contained in accordance with recommendations of the Hydrogeological Specialist report (GCS, 2020). It is recommended that regular groundwater and surface water quality monitoring be maintained in the areas potentially affected by seepage and runoff from these facilities.

In accordance with the requirements of the impact assessment process the potential impacts to human health, identified as part of the HHRIA, were evaluated to determine the significance and risk of each impact. The potential health impacts identified were evaluated using a set of qualitative evaluation variables. This qualitative evaluation concluded that the risk of the impact associated with either long-term or short-term exposure to airborne particulates is Moderate. The potential impacts from exposure to hazardous constituents of the airborne particulates is also Moderate.

The risk of impact to human health from ingestion of contaminated water dispersed from the Extension Project TSF is ranked as Moderate for unmitigated conditions and Low for mitigated conditions.

## DECLARATION OF INDEPENDENCE

---

**I, Nardus Potgieter declare that:**

- All work relating to the proposed Project was undertaken as an independent consultant;
- I have the necessary required expertise to conduct human health risk impact assessments, including the required knowledge and understanding of internationally accepted best practice, guidelines and policies that are relevant to the activity;
- I have undertaken all the work and associated studies in an objective manner, even if the findings of these studies were not favourable to the project proponent;
- I have no vested financial interest in the proposed project or the outcome thereof, apart from remuneration for the work undertaken under the auspices of the project manager, AquiSim Consulting (Pty) Ltd.;
- I have no vested interest, including any conflicts of interest, in either the proposed project or the studies conducted in respect of the proposed project, other than providing an objective evaluation of the identified impacts and complying with the requirements of the impact assessment process;
- I have disclosed any material factors that may have the potential to influence the competent authority's decision and/or objectivity in terms of any reports, plans or documents related to the proposed project as required by the relevant regulations.

# TABLE OF CONTENTS

---

---

CROSS REFERENCE TABLE .....	iii
EXECUTIVE SUMMARY .....	iv
DECLARATION OF INDEPENDENCE .....	vi
TABLE OF CONTENTS.....	vii
LIST OF TABLES.....	ix
LIST OF FIGURES.....	x
TERMS AND ABBREVIATIONS.....	xii
<b>1 BACKGROUND AND SCOPE OF THE STUDY.....</b>	<b>14</b>
<b>1.1 Introduction .....</b>	<b>14</b>
<b>1.2 Study Framework.....</b>	<b>16</b>
1.2.1 Risk Based Approach.....	16
1.2.2 Health Risk Assessment Paradigm .....	16
<b>1.3 Scope of the Assessment.....</b>	<b>17</b>
1.3.1 Pathways, and Receptors of Concern .....	17
<b>2 HAZARD ASSESSMENT .....</b>	<b>19</b>
<b>2.1 Introduction .....</b>	<b>19</b>
<b>2.2 Project Description .....</b>	<b>19</b>
<b>2.3 Sources of Contamination .....</b>	<b>20</b>
2.3.1 General.....	20
2.3.2 Atmospheric Pollution Sources.....	20
2.3.3 Aquatic Pollution Sources .....	21
<b>2.4 Contaminants of Potential Concern .....</b>	<b>22</b>
2.4.1 Atmospheric Pathway.....	22
2.4.1.1 Baseline Air Quality.....	22
2.4.1.2 Predicted Impacts from the Expansion Project .....	22
2.4.2 Aquatic Pathway .....	24
2.4.2.1 General.....	24
2.4.2.2 Acid Generating Potential .....	25
2.4.2.3 Predicted Water Quality Impacts.....	25
2.4.2.4 Baseline Water Quality .....	27
2.4.2.5 Summary .....	30
<b>2.5 Health Significance of Contaminants .....</b>	<b>31</b>

---



2.5.1	General.....	31
2.5.2	Environmental Health Significance of Particulate Matter .....	31
2.5.3	Environmental Health Significance of Arsenic .....	33
2.5.1	Environmental Health Significance of Aluminium .....	32
2.5.2	Environmental Health Significance of Iron .....	33
2.5.3	Environmental Health Significance of Lead .....	<b>Error! Bookmark not defined.</b>
2.5.4	Environmental Health Significance of Manganese .....	34
2.5.5	Environmental Health Significance of Uranium.....	35
2.5.6	Environmental Health Significance of Sulphate.....	36
<b>2.6</b>	<b>Summary.....</b>	<b>36</b>
<b>3</b>	<b>DOSE RESPONSE ASSESSMENT.....</b>	<b>37</b>
<b>3.1</b>	<b>Principles of Dose-Response Assessment.....</b>	<b>37</b>
<b>3.2</b>	<b>Particulate Matter .....</b>	<b>37</b>
3.2.1	Introduction .....	37
3.2.2	Short-term exposure to particulates .....	39
3.2.3	Long-term exposure to particulates .....	39
<b>3.3</b>	<b>Toxicity of Airborne Manganese .....</b>	<b>40</b>
<b>3.4</b>	<b>Toxicity of Airborne and Water Bourne Uranium .....</b>	<b>41</b>
<b>3.5</b>	<b>Toxicity of Arsenic in Drinking Water .....</b>	<b>43</b>
<b>3.6</b>	<b>Toxicity of Lead in Drinking Water .....</b>	<b>44</b>
<b>4</b>	<b>EXPOSURE ASSESSMENT.....</b>	<b>46</b>
<b>4.1</b>	<b>Introduction .....</b>	<b>46</b>
<b>4.2</b>	<b>Atmospheric Pathway .....</b>	<b>46</b>
4.2.1	Contaminant Dispersion in the Environment .....	46
4.2.2	Receptors .....	47
4.2.3	Results.....	49
<b>4.3</b>	<b>Aquatic Pathway.....</b>	<b>51</b>
<b>5</b>	<b>RISK CHARACTERISATION .....</b>	<b>53</b>
<b>5.1</b>	<b>Introduction .....</b>	<b>53</b>
<b>5.2</b>	<b>Methodology of Quantifying Impact.....</b>	<b>53</b>
<b>5.3</b>	<b>Results.....</b>	<b>56</b>
5.3.1	Daily (short term) Risks Associated with exposure to Airborne Particulates .....	56
5.3.2	Annual (long term) Risks Associated with Exposure to Particulates.....	60
5.3.3	Discussion of Results.....	66
5.3.3.1	General.....	66
5.3.3.2	Short-Term Risks .....	66
5.3.3.3	Long-Term Risks .....	66
5.3.3.4	Evaluation.....	67
<b>5.4</b>	<b>Health Risks Associated with Exposure to Particle Associated ContaminantsM .....</b>	<b>67</b>

5.4.1	Calculation of Non-cancer Risk Associated with Inhalation Exposure to Airborne Contaminants.....	67
<b>5.5</b>	<b>Health Risks Associated with Contaminant Concentrations in Water .....</b>	<b>69</b>
5.5.1	Calculation of Non-cancer Risk Associated with Ingestion Exposure to Contaminants in Groundwater and Surface Water Resources. ....	69
5.5.2	Lead and Sulphate in Drinking Water .....	71
5.5.3	Cancer Risk Assessment .....	71
<b>5.6</b>	<b>Conclusions and Recommendations.....</b>	<b>71</b>
5.6.1	Criteria Pollutants .....	<b>Error! Bookmark not defined.</b>
5.6.2	Hazardous Elements Associated with Airborne Particulates .....	72
5.6.3	Contaminants in Water Resources .....	72
5.6.4	Recommendations .....	72
<b>6</b>	<b>UNCERTAINTY ANALYSIS .....</b>	<b>74</b>
6.1	Assumptions and Uncertainty in the Assessment of Health Risks.....	74
6.2	Vulnerability in the South African population .....	75
6.4	Uncertainty in Assumptions .....	76
<b>7</b>	<b>IMPACT ASSESSMENT.....</b>	<b>77</b>
7.1	Impact Identification.....	77
7.2	Impact Assessment Methodology .....	78
7.3	Evaluation and Ranking of the Impacts .....	79
7.3.1	HHRIA01-Human health impact from inhalation exposure to particulates.....	79
7.3.2	HHRIA02- Non-cancer (systemic) health effects from inhalation exposure to particle associated contaminants .....	80
7.3.3	HHRIA03- Risk of systemic health effects and cancer in humans as a result of ingestion of contaminated water. ....	81
<b>8</b>	<b>REFERENCES .....</b>	<b>84</b>

## LIST OF TABLES

---

Table 2.1:	Summarised results from the elemental analysis of tailings samples representative of the AngloGold Ashanti TSFs, reported in parts per million (ppm).....	23
Table 2.2	Screening of elements present in the dust dispersed from the proposed Project. ....	24
Table 2.3:	Results from geochemical seepage modelling for Kareerand tailings. ....	25
Table 2.4:	Summary of gold tailings seepage water analytical results (GCS, 2020). ....	26
Table 3.1	Short-term PM <sub>10</sub> risk factors for mortality. ....	39

---

Table 3.2 Long-term PM <sub>2.5</sub> risk factors for mortality (COMEAP, 2009; Pope, et al., 2002).....	40
Table 3.3 – Minimum risk levels for oral and inhalation exposure to uranium.....	42
Table 4.1: Simulated ground level concentrations of contaminants of concern at affected potential receptors identified for the proposed Project. ....	49
Table 4.2: Estimated annual average ground level concentrations of particle associated manganese and uranium (see Section 2.4.1 for discussion on derivation of element specific concentrations). ....	50
Table 5.1: Mortality data for the Dr Kenneth Kaunda District Municipality, for the year 2017. ....	55
Table 5.2: Potential daily increase in personal risk of non-accidental mortality associated with short-term exposure to PM <sub>10</sub> . ....	56
Table 5.3: Potential daily increase in personal risk of cardiovascular mortality associated with short-term exposure to PM <sub>10</sub> emissions. ....	57
Table 5.4: Potential annual increase in personal risk of non-accidental mortality associated with long-term exposure to modelled PM <sub>2.5</sub> . ....	60
Table 5.5: Potential annual increase in personal risk of cardiopulmonary mortality associated with long-term exposure to modelled PM <sub>2.5</sub> . ....	61
Table 5.6: Potential annual increase in personal risk of lung cancer mortality associated with long-term exposure to modelled PM <sub>2.5</sub> concentrations. ....	62
Table 5.7: HQs associated with exposure to particle associated contaminants.....	68
Table 5.9 Exposure parameters used in estimation of target concentrations.....	69
Table 5.10 Estimated dose values and calculated HQs from exposure to aquatic pathway contaminants.....	70

## **LIST OF FIGURES**

---

Figure 1.1 Locality map showing the Kareerand TSF and expansion project with associated infrastructure.....	15
Figure 4.1 Example of particulate dispersion modelling results for the proposed Expansion Project (AquiSim, 2020). ....	47
Figure 4.2 Locations of potential receptors identified by Airshed (2020). ....	48
Figure 4.3 Simulated potential sulphate migration plume from the Kareerand and Expansion Project TSFs.....	52

Figure 5.1: Comparison of estimated individual risks of non-accidental mortality associated with short-term exposure to PM<sub>10</sub> from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations. .... 58

Figure 5.2: Comparison of estimated individual risks of cardiovascular mortality associated with short-term exposure to PM<sub>10</sub> from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations. .... 59

Figure 5.3: Comparison of estimated individual risks of non-accidental mortality associated with long-term exposure to PM<sub>2.5</sub> from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations. .... 63

Figure 5.4: Comparison of estimated individual risks of cardiopulmonary mortality associated with long-term exposure to PM<sub>2.5</sub> from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations. .... 64

Figure 5.5: Comparison of estimated individual risks of lung cancer mortality associated with long-term exposure to PM<sub>2.5</sub> from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations. .... 65

## TERMS AND ABBREVIATIONS

---

ATSDR	Agency for Toxic Substances and Disease Registry
Cardiopulmonary	Relating to or involving the heart and the lungs.
Cardiovascular system	An organ system that circulates blood throughout the human body. The cardiovascular system consists of the heart, arteries and veins.
COMEAP	UK Committee on the Medical Effects of Air Pollutants
COPD	Chronic Obstructive Pulmonary Disease: disease of the lungs in which the airways become narrowed. This leads to a limitation of the flow of air to and from the lungs causing shortness of breath.
Criteria pollutants	A term used internationally to describe air pollutants that have been regulated and are used as indicators of air quality
EIA	Environmental Impact Assessment
Epidemiological	Scientific studies of factors affecting the health and illness of populations.
HHRIA	Human Health Risk and Impact Assessment
IARC	International Agency for Research on Cancer
LOAEL	Lowest Observed Effect Level
Lung function	Lung function tests determine the lung capacity (volume of air the lungs can hold), the efficiency with which air is moved in and out of the lungs, and the efficiency of carbon monoxide and oxygen exchange. The tests aid in the diagnosis of lung diseases, and measure the severity of lung problems.
Morbidity	The state of being diseased (from Latin morbidus: sick, unhealthy), or disability irrespective of cause (e.g., disability caused by accidents).
Mortality	Number of people dying during a given time interval.
MRL	Minimal Risk Levels
NGL	Natural ground level
OEHHA	California State Environmental Protection Agency, Office of Environmental Health Hazard Assessment
PM	Particulate matter air pollution.
Prevalence	Epidemiological term indicating the total number of cases of a given disease in a specified population at a specified time.
REL	Reference Exposure Level
Respiratory system	In humans and other mammals, the respiratory system consists of the airways, the lungs, and the respiratory muscles that mediate the movement of air into and out of the body.

RfC	Reference Concentration
RfD	Reference Dose
RR	Relative risk or risk ratio. A ratio of the probability of an outcome (e.g. the disease under study) occurring in the exposed group versus a non-exposed group.
US EPA	United States Environmental Protection Agency
WHO	World Health Organisation

# 1 BACKGROUND AND SCOPE OF THE STUDY

---

## 1.1 INTRODUCTION

---

EnviroSim Consulting, was appointed by AquoSim Consulting Pty (Ltd) (AquoSim) to prepare a human health risk assessment in support of an Environmental Impact Assessment for the expansion of the Mine Waste Solutions (MWS) Kareerand Tailings Storage Facility (TSF). The Kareerand TSF is located near the town of Stilfontein in the North West Province of South Africa. The Expansion Project falls within the City of Matlosana and JB Marks Local Municipalities, both of which are in the Dr Kenneth Kaunda District Municipality of the North-West Province.

The MWS operations involve the recovery and re-processing of mine tailings from historical tailings storage facilities (TSFs) located over a large area to the east and south of Klerksdorp. The tailings are recovered with high pressure water cannons that are used to slurry the tailings on the source TSFs. This slurry is pumped, via a series of closed pipelines, to the MWS/Chemwes Processing Plant near Stilfontein where the residual gold and uranium value is extracted from the tailings. When a historical TSF (source TSF) has been completely recovered, the footprint is cleared and rehabilitated. Residues from the tailings Processing Plant is pumped to the Kareerand TSF for deposition.

Construction of the Kareerand TSF commenced in 2010 and was commissioned in 2011 with a total capacity of 352 million tonnes and initial design life of 14 years (to 2025). Subsequent to commissioning of the Kareerand TSF, AngloGold Ashanti (AGA) acquired MWS and increased the tailings production target by an additional 485 million tonnes, in order to accommodate the additional TSFs owned by AngloGold Ashanti. The increased rate of deposition on the existing Kareerand facility means that the TSF will reach its limiting Rate of Rise by the end of 2021, with consequent loss of storage capacity for the new arisings. In order to create the additional tailings storage capacity required, an expansion of the existing Kareerand TSF is proposed. This proposed extension (hereafter referred to as the Expansion Project) will be to the west of the current TSF and will cover an additional area of approximately 380 ha (see Figure 1.1).

In general, mineral processing residue management activities are known to be responsible for various environmental disturbances, which have the potential to release a variety of pollutants to the environment. It is therefore necessary to address the concerns of communities living in the vicinity of such activities with regard to potential health risks, by performing a human health risk assessment. The assessment results are intended to serve as a scientific basis for the understanding of potential health risks.

AquoSim, approached EnviroSim Consulting with a request to perform an assessment of the potential impact on the health of communities living in the vicinity of the proposed Expansion Project, with regard to exposure to airborne pollutants as well as contaminants identified as relevant to water resources in the area. The human health risk and impact assessment (HHRIA), is aimed at specifically addressing these concerns, and is thus limited to the quantitative evaluation of potential health risks relating to the inhalation of airborne pollutants and ingestion of waterborne contaminants.

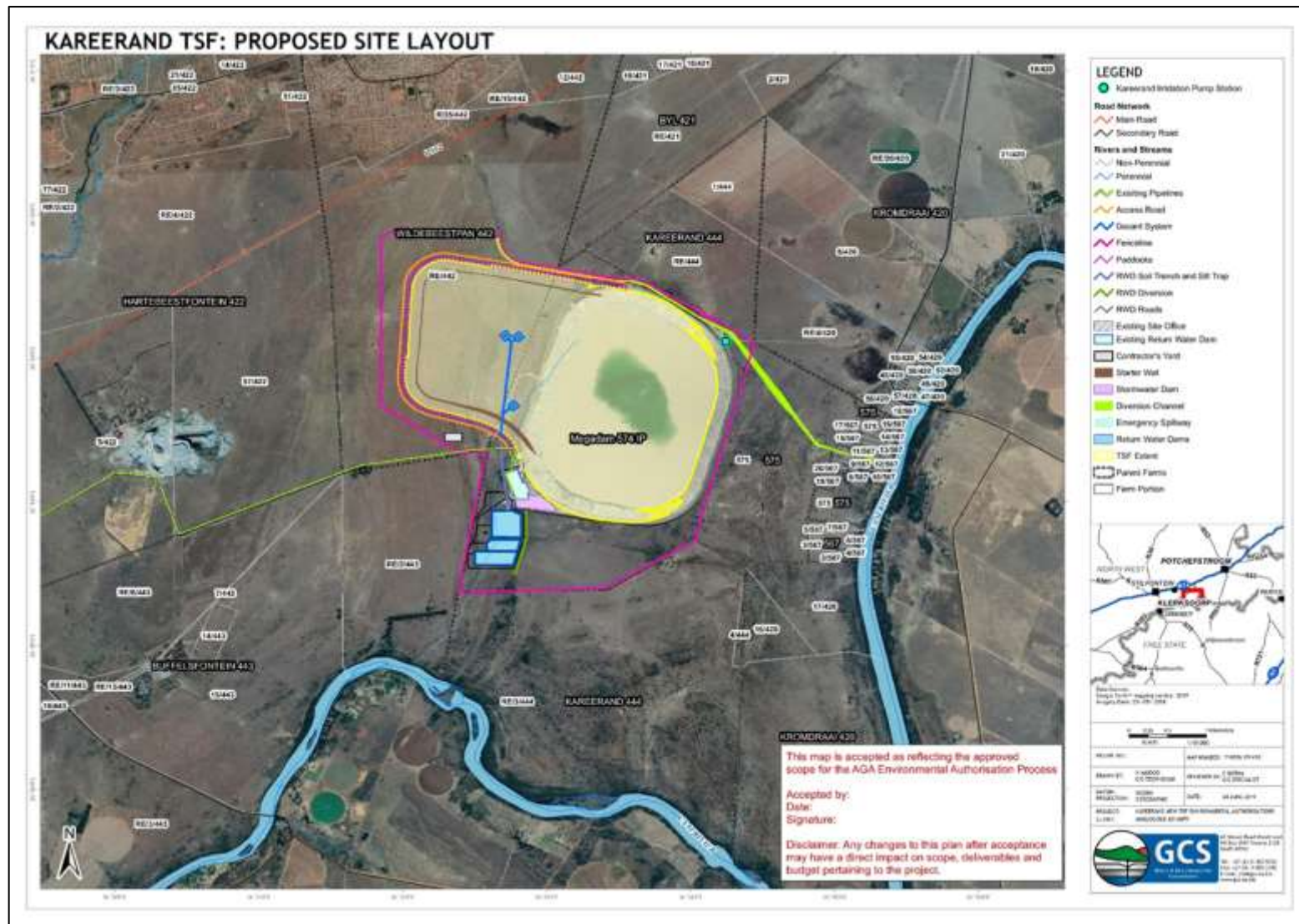


Figure 1.1 Locality map showing the Kareerand TSF and expansion project with associated infrastructure (GCS, 2020).



This HHRIA forms part of the broader environmental authorisation process and includes all aspects relevant to the quantification and assessment of human health risks, as it pertains to the requirements of an Environmental Impact Assessment (EIA).

---

## 1.2 STUDY FRAMEWORK

---

### 1.2.1 Risk Based Approach

Overall, a risk-based approach is followed in development of the HHRIA. This approach is aimed at defining the relationship between cause and effect for the impact under investigation, which is, understanding how a potential hazard occurs, the probability of its occurrence and the consequence if it occurs. The methodology for performing the risk-based assessment is based on defining and understanding the three components of the risk, namely the source of the potential hazard, the pathway along which the hazard propagates and the receptor that experiences the risk.

This Source-Pathway-Receptor analysis methodology is inherently systematic, traceable and transparent and provides the opportunity for iterative evaluation of the system under investigation. Since all three components (source, pathway and receptor) are necessary to demonstrate risk, the Source-Pathway-Receptor methodology allows screening of issues that are not relevant to the investigation.

The Source-Pathway-Receptor methodology is central to the identification and evaluation of potential impacts associated with the proposed Expansion Project. Assessment and quantification of the identified impacts is performed in accordance with the principles of health risk assessment as defined by the health risk assessment paradigm.

### 1.2.2 Health Risk Assessment Paradigm

Human health risk assessment is the qualitative or quantitative characterisation of the probability of potentially adverse health effects in humans from exposure to environmental hazards (Hall, et al., 1997). The outputs of a human health risk assessment, performed for mining activities such as the proposed Project, are necessary for informed regulatory decisions regarding emissions and effluents from the operation and contamination of ambient air, water or the terrestrial food chain to which humans may be exposed.

The original paradigm for regulatory human health risk assessment was developed in the USA by the US National Research Council (NRC, 1983). This model has been adopted and refined by, among other, the US Environmental Protection Agency (US EPA) and is widely used for quantitative health risk assessments (IPCS, 1999).

The risk assessment paradigm essentially divides a human health risk assessment into a number of logical steps, as follows:

- **Hazard identification** involves the identification of substances relevant to the situation under investigation, which have the potential to be released to the environment and are suspected to pose hazards to human health and the environment.
- **Dose-response assessment** addresses the relationship between levels of biological exposure and the manifestation of adverse health effects in humans.
- **Exposure assessment** is a description of the environmental pathways involved in the distribution of hazardous substances and the identification of potentially exposed receptors.
- **Risk characterisation**, which involves the integration of the components described above, with the purpose of determining whether specific exposures to an individual or a community might lead to adverse health effects.
- **Uncertainty analysis** is identifying the nature and magnitude of the uncertainty and variability inherent in the characterisation of risk.

---

## 1.3 SCOPE OF THE ASSESSMENT

---

### 1.3.1 Pathways, and Receptors of Concern

In the preparation of the HHRIA, the following documents and specialist study reports were consulted:

- Feasibility Assessment Report (Knight Piésold, 2019).
- Air Quality Specialist Report for the Kareerand Tailings Storage Facility Extension (Airshed, 2020)
- AngloGold Ashanti dust monitoring project Volume II – Final Report (Annegarn, et al., 2010)
- Radiological Impact Assessment report (AquiSim, 2020).
- Hydrogeological Impact Assessment (GCS, 2020)

Based on the understanding of the proposed activities and the environmental conditions, gleaned from the documents and reports listed above, the atmospheric and aquatic pathways are identified as the most prominent means by which humans may come into contact with potentially hazardous contaminants from the existing Kareerand TSF and the proposed Expansion Project.

These specialist study reports are the primary sources of quantitative information on environmental concentrations of airborne and water borne contaminants originating from the proposed Expansion Project. The scope of the HHRIA is limited by the reported data and findings of specialist studies that describe the atmospheric and aquatic pathways, and the transport and dispersion of potentially hazardous contaminants within these pathways. The information and data obtained from the specialist studies is accepted to be accurate and no verification of the data has been undertaken by EnviroSim.

The HHRIA will only consider non-radiogenic health effects associated with the potential contaminants. Health concerns relating to radioactive contaminants that may be generated from the

proposed operations and the radiological impacts to humans and the environment are addressed in the report by AquiSim Consulting (AquiSim, 2020).

The assessment endpoint of the HHRIA is limited to the evaluation of the risks posed to the health of members of the public residing in the vicinity of the proposed Expansion Project. Potential receptors will be identified from the communities closest to the proposed Expansion Project location, based on information available for these communities. For the purpose of the HHRIA, a sensitive receptor is defined as:

*Any individual or population group whose habits, location or other characteristics could cause them to be exposed to higher concentrations of contaminants than the rest of the exposed population.*



## 2 HAZARD ASSESSMENT

---

### 2.1 INTRODUCTION

---

Hazard assessment is the identification of contaminants suspected to pose a hazard to human health and a description of the type of health hazard they may produce. The hazard assessment step is designed as logical processes for screening the myriad of possible contaminants, as well as the possible circumstances that may lead to human exposure, and so simplify the identification of contaminants of potential concern.

Screening and identifying contaminants of potential concern requires information about the potential sources of health hazards as well as a description of the most likely exposure pathways and receptor populations. The conceptual understanding of the hazard sources, exposure pathways and receptors associated with proposed Expansion Project, was based on the information presented in the documents and specialist reports listed in Section 1.3.1.

The hazard assessment starts with a summary overview of the proposed Expansion Project and associated facilities as well as the environmental disturbances that are expected as part of the proposed Expansion Project. The level of detail presented in the overview is proportionate to the information available and that needed for the identification of potential hazards. That is, the project description is intended to provide a clear representation of the features of the project relevant to the potential impacts under evaluation, and therefore does not necessarily represent a comprehensive, detailed description of all aspects.

The summary project description is followed by an identification of contaminants of potential concern and a description of the environmental health significance of each identified contaminant.

### 2.2 PROJECT DESCRIPTION

---

The proposed Extension Project will add a further 380 ha to the existing Kareerand TSF to cover a total area of 868ha. The extension is expected to become operational in 2021 and is estimated to operate for 21 years. The final tonnages on the existing TSF (including tonnage already deposited) will be 498Mt, while a final tonnage of 354Mt is estimated for the extension.

The existing Kareerand and Extension Project TSFs will be operated as two independent compartments. The variance will be the deposition tonnages at a given time to ensure that a maximum rate of rise is co-ordinated on both the facilities. The aim will be to consolidate the two TSFs at closure and maintain it as one facility with a single central pool. The Expansion Project TSF will be constructed by an upstream construction method using cyclones for tailings deposition.

A bund wall will be constructed around the TSF, next to the access road (see Figure 1.1). The wall will be 6 m at its highest point and 2 m at its lowest with crest width 8 m. The bund wall will also be used as access road on northern side of TSF.

A solution trench, lined with 100 mm thick mesh reinforced concrete will be constructed around the northern, western and southern side of the Expansion Project TSF. Water is decanted from the top of the TSF via a gravity pipe decant system to the solution trench. The lined trench will convey the decant water and storm water from the side slopes, filter discharge (seepage water) from the outer drains and surface runoff from the side slopes to a new return water dam (RWD).

The new RWD will consist of three compartments with a combined capacity of 837 000 m<sup>3</sup> (area of 60 Ha), and will be located south of the Expansion Project and existing RWD complex. The three compartments (one for operation, the other two for dirty water containment) will be lined with a double HDPE liner, consisting of 2 mm geomembrane and 1.5 HDPE geomembrane, and a leakage-detection system. Water from the solution trench will pass through a concrete-lined silt trap between penstock outlet and RWD.

Additional infrastructure required across the operational footprint include new pump stations, slurry launders and connecting slurry and process water pipelines. In total, three new main pump stations and three new satellite pump stations will be built.

---

## 2.3 SOURCES OF CONTAMINATION

---

### 2.3.1 General

All relevant sources of environmental contamination associated with the Expansion Project, must be identified. The sources are characterised in terms of their unique composition and their characteristics, which will determine how contaminants may be distributed in the environment. Based on the available information for the Kareerand TSF and Expansion Project, two types of sources can be identified namely sources of atmospheric pollutants and sources of aquatic contaminants.

Characterisation of the sources relies on the findings of the Air Quality Specialist Report (Airshed, 2020) and Hydrogeological Assessment (GCS, 2020) reports for information.

### 2.3.2 Atmospheric Pollution Sources

The Air Quality Specialist Report (Airshed, 2020) presents an emissions inventory that is compiled by quantifying the contribution to concentrations of ambient air pollution from all potential atmospheric emission sources associated with the Expansion Project. The report indicates that emissions to air during the current activities at the Kareerand TSF, as well as the construction, and operation of the Expansion Project is expected to result from a variety of sources, including bulldozing, scraping, material transfer, wheel entrainment, vehicle exhaust tailpipe and processing activities. Of these emissions Airshed identify airborne particulate matter (PM) as the most significant pollutant.

According to the Airshed (2020) report, particulate matter emissions are expected during the construction, operational, decommissioning and closure phases of the Kareerand TSF and Expansion Project. However, the report indicates that only operational phase air quality impacts were assessed quantitatively as the potential impacts from the construction, decommissioning and closure phase emissions are likely to have a “low” significance (Airshed, 2020).

The operational phase emissions inventory presented in the Airshed report identify several sources as potentially contributing to the concentrations of airborne pollutants. These sources are:

- Particulate emissions from vehicle entrainment along an existing unpaved access road;
- Particulate emissions from vehicle exhaust;
- Particulate emissions from concurrent rehabilitation equipment operating on the TSF area;
- Particulate emissions from concurrent rehabilitation equipment exhaust; and
- Particulate emissions from wind erosion of the existing and additional TSF area.

However, the Airshed report indicates that the impacts quantified through dispersion modelling considered only the contribution of wind erosion of the current Kareerand TSF and Expansion Project areas. The reasons given for this decision is that the areas where the tailings are recovered for reprocessing are not consistent throughout the operational life of the Kareerand TSF or Expansion Project. Dispersion modelling of the rehabilitation is therefore unlikely to be representative of the actual activities and emissions from these activities are estimated to be minimal in comparison to wind erosion (Airshed, 2020).

Airshed (2020) discusses baseline air quality indicating that the main sources likely to contribute to baseline PM emissions in the area include mining operations, industrial operations, vehicle entrained dust from local roads, vehicle exhaust and windblown dust from exposed areas. Other sources of PM include farm activities, occasional biomass burning and household fuel burning in the residential areas of Stilfontein, Klerksdorp, Khuma Township and Village Main Reef Mine. These sources can be expected to contribute to the cumulative concentrations of air pollutants once the proposed expansion project is underway.

However, the report (Airshed, 2020) indicates that that the quantification and subsequent modelling of these sources did not form part of the scope of the current Air Quality Specialist Report. Instead it is noted that simulations for the AGA Vaal River and MWS operations undertaken in 2015, indicate that PM<sub>10</sub> and PM<sub>2.5</sub> concentrations complied to ambient air quality guidelines at all of the sensitive receptor locations considered in the study, over both the short- and long-term. So too were dustfall rates below the National Dust Control Regulations limit for residential areas.

### 2.3.3 Aquatic Pollution Sources

The Hydrogeological Impact Assessment report (GCS, 2020) indicates that the tailings material to be deposited onto the extension project TSF was classified as Type 3 waste in accordance with the South African Waste Classification and Management Regulations. Based on this classification, the extension project TSF is proposed to be constructed with a Class C Barrier containment system to limit seepage into the underlying aquifer. The extension project will further include an under-drain system and will be equipped with large, lined, return water dams.

Consequently, the impacts to water quality that GCS predict through numerical groundwater modelling, relate largely to the existing Kareerand TSF while the contribution from the expansion project is predicted to be low.

The contributions from other sources to baseline water quality is not discussed in the Hydrogeological Impact Assessment report (GCS, 2020).

---

## 2.4 CONTAMINANTS OF POTENTIAL CONCERN

---

### 2.4.1 Atmospheric Pathway

#### 2.4.1.1 Baseline Air Quality

The discussion of baseline air quality presented in the Air Quality Specialist Report (Airshed, 2020) identified only PM as pollutant emitted from other sources in the area. However, baseline airborne concentrations of PM associated with these other sources are not provided. For the purpose of this assessment it is accepted that the contribution to airborne concentrations of PM modelled for the Kareerand TSF is far greater than any of the other baseline sources noted by Airshed and that this contribution alone can be considered a reasonable estimate of baseline PM.

#### 2.4.1.2 Predicted Impacts from the Expansion Project

PM (dust) generated by wind erosion of the Kareerand TSF and Expansion Project surface was identified in the Air Quality Specialist Report (Airshed, 2020) as the primary potential impact to the atmospheric pathway.

PM is normally assessed as different categories, classified by aerodynamic size. The inhalable particulate fraction, PM<sub>10</sub>, refers to PM with an aerodynamic diameter of up to 10 µm, i.e., the fine and coarse particle fractions combined. Fine or respirable particles are up to 2.5 µm in diameter (PM<sub>2.5</sub>) and include the fine and ultrafine fraction, the latter which refers to particles less than 0.1 µm in diameter (PM<sub>0.1</sub>). The full particle size spectrum is normally referred to as Total Suspended Particulates (TSP), which includes all size fractions of PM that are suspended in air. The Air Quality Specialist Report includes emission estimates for PM<sub>10</sub>, PM<sub>2.5</sub> and TSP.

Concerns have also been raised regarding the composition of the PM dispersed from the Expansion Project, and specifically the effects of potentially hazardous constituents of the particulates on the health of potentially affected communities. In order to identify the constituents of the particulate matter which could have an effect on the health of potentially affected communities, a screening assessment is performed using health-risk based guidance values from literature.

As estimate of the composition of the PM that will be generated from the existing Kareerand TSF and the Extension Project, information available for the most likely source material, the tailings material from the historical AngloGold Ashanti and MWS TSFs, is used. The tailings material recovered from these TSFs will be reprocessed to recover residual gold value and the residues will be deposited onto the Kareerand TSF and Expansion Project. The results of a compositional analysis performed on samples of tailings collected from several TSFs in the former AngloGold Ashanti Vaal River operational area, was obtained from a comprehensive study report by the University of Johannesburg Department of Geography (Annegarn, et al., 2010).

The mineralogical composition of the tailings, reported by Annegarn, et al. (2010), indicate that the tailings consist primarily of crystalline silica (61% – 84%) with minor concentrations of other minerals including pyrophyllite, rutile, lepidolite and clinocllore. Silicon, iron and aluminium are the primary elements present, while calcium and potassium are included as major constituents. Several potentially hazardous elements, such as bismuth, lead, uranium and thorium are also present in quantities enriched relative to normal crustal abundance. Table 2.1 presents a summary of the results from the elemental analysis of tailings collected from different AngloGold Ashanti TSFs. The average concentrations of selected elements are presented. The elements selected include major constituents and elements identified as being present at concentrations higher than normally found in the earth's crust (Annegarn, et al., 2010).

**Table 2.1: Summarised results from the elemental analysis of tailings samples representative of the AngloGold Ashanti TSFs, reported in parts per million (ppm).**

Constituent	Concentration
	ppm
Iron	24 100
Magnesium	57 400
Aluminium	26 800
Silicon	312 000
Cadmium	0.11
Chromium (total)	73
Cobalt	44
Copper	49
Lead	87
Manganese	823
Nickel	66
Selenium	0.17
Thorium	27
Uranium	151
Vanadium	24

In order to estimate the concentrations of these elements communities may be exposed to from the proposed extension project, the modelled airborne concentrations of particulate matter reported by Airshed (2020) was used. According to the Airshed report, the highest daily concentrations of PM<sub>10</sub> from the existing Kareerand TSF is estimated at 315.9 µg.m<sup>-3</sup> while the combined Kareerand and extension project contribution is estimated at 339.5 µg.m<sup>-3</sup>. These concentrations represent the absolute highest values modelled and are not representative of concentrations at any of the potential receptors, which will be lower. These maximum modelled particulate concentrations represent the 99<sup>th</sup> percentile of modelled values and thus represent a frequency of approximately 4 days per year. Please see the Airshed (2020) Air Quality Specialist Report (Section 2.3.4) for further information.

Using the concentrations of elements listed in Table 2.1 the reported airborne particulate concentrations are scaled to estimate airborne concentrations of the different elements in air. The concentrations estimated in this way are conservative, as the particulate concentration values used



are actually likely to occur for only a few days a year at a specific point on or very near to the Kareerand TSF and Extension Project. However, the results are appropriate for use in a screening assessment.

The estimated concentrations are compared to health-risk based screening values, where values were available. Table 2.2 present a summary of the screening assessment. The comparison shows that airborne concentrations of manganese and uranium exceed the screening criteria. The guideline value considers chronic exposure (more than a year). Given that the estimated airborne particulate concentrations used are daily maxima, assessment of exposure at this level is very conservative. Nevertheless, both manganese and uranium are evaluated further as pollutants.

**Table 2.2 Screening of elements present in the dust dispersed from the Expansion Project.**

Constituent	Estimated Element Concentration in Air ( $\mu\text{g}\cdot\text{m}^{-3}$ )		Screening Value
	Existing Kareerand TSF	Kareerand TSF & Expansion	$\mu\text{g}\cdot\text{m}^{-3}$
Cadmium	3.47E-05	3.73E-05	5.00E-03 <sup>1</sup>
Chromium (total)	2.30E-02	2.47E-02	1.00E-01 <sup>3</sup>
Cobalt	1.39E-02	1.49E-02	1.00E-01 <sup>3</sup>
Copper	1.54E-02	1.66E-02	1.00E+02 <sup>6</sup>
Lead	2.74E-02	2.95E-02	5.00E-01 <sup>1</sup>
Manganese	2.59E-01	2.79E-01	1.50E-01 <sup>1</sup>
Nickel	2.08E-02	2.24E-02	2.50E-02 <sup>1</sup>
Selenium	5.36E-05	5.76E-05	3.00E+00 <sup>7</sup>
Thorium	8.51E-03	9.15E-03	9.50E+00 <sup>5</sup>
Uranium	4.76E-02	5.12E-02	4.00E-02 <sup>4</sup>
Vanadium	7.56E-03	8.14E-03	1.00E+00 <sup>2</sup>

1. WHO Guidelines ( $\mu\text{g}/\text{m}^3$ ) (2000) chronic guidelines (1 year+)
2. WHO Guidelines ( $\mu\text{g}/\text{m}^3$ ) (2000) acute & Sub- acute guidelines (24hr)
3. US ATSDR Maximum Risk Levels intermediate exposure (up to 1 year)
4. US ATSDR Maximum Risk Levels chronic exposure (up to 1 year)
5. US ATSDR Toxicological Profile for Thorium (ATSDR, 1990)
6. The Californian Office of Environmental Health Hazard Assessment acute Reference Exposure Levels
7. EPA 2001b 40CFR 266, Appendix IV (US EPA, 2002)

## 2.4.2 Aquatic Pathway

### 2.4.2.1 General

The Hydrogeological Impact Assessment report (GCS, 2020) includes results of a geochemistry assessment performed on tailings from the Kareerand TSF. The geochemistry assessment had as its purpose to determine the chemical quality of seepage water expected from the Kareerand TSF by investigating the sulphur content of the TSF, the geochemical composition and acid/base characteristics of the tailings and perform geochemical modelling to predict future behaviour of the Kareerand and Extension Project TSFs.

To determine the geochemical characteristics of the tailings, 6 tailings samples were collected and analysed from 3 sites on the existing Kareerand TSF. Underdrain seepage, return water, and seepage water were also collected from another TSF and analysed.

### 2.4.2.2 Acid Generating Potential

Laboratory acid base accounting tests results reported by (GCS, 2020) show that the neutralisation potential of the tailings is much lower than its acid potential. All tailings therefore have some potential to generate acid drainage. This was confirmed with net acid generation potential (NAG) tests that showed samples of tailings having a high potential for acid generation.

### 2.4.2.3 Predicted Seepage Water Quality

Results of the laboratory tests performed on the tailings samples were used to develop a numeric geochemical reaction model, to predict the composition of seepage from the tailings over time. Where the laboratory tests provide a picture of the instantaneous release of contaminants over a single period of a few hours, the geochemical model attempts to predict the release of contaminants over the long term by taking geochemical processes such as dissolution, precipitation and adsorption into account. Table 2.3 present the findings of the geochemical reaction model

**Table 2.3: Results from geochemical seepage modelling for Kareerand tailings.**

Parameters	Units	Predicted Values Range
pH	pH units	6-7
Total dissolved solids	mg.l <sup>-1</sup>	2500 - 4500
Sulphate		1500 -2500
Sodium		250-1000
Calcium		500-1000
Magnesium		100-300
Potassium		20 - 40
Chloride		500-2000

Conceptually, a TSF can be subdivided, based on the water content of the tailings, into an inner saturated core and an outer layer of unsaturated tailings, which vary in thickness over the outside of the TSF. According to the GCS (2020) report, the water quality in the outer unsaturated layer or shell of the Kareerand TSF and the deeper saturated core will be different. Tailings in the outer shell will include the unsaturated zone and the upper contact zone with the saturated core zone. The water quality in the outer shell will have a much higher SO<sub>4</sub> content and will eventually become acidic, mainly as a result of air diffusing into the unsaturated tailings. Seepage water at the toe of the tailings dam will first be dominated by water from the deeper saturated core but will become more and more representative of the water in the outer shell post-closure.

Similarly, seepage to the underlying aquifer will mostly be that of the inner saturated core for the first few decades. However, this core zone will decrease in size over time until it is limited only to the very central part of the TSF footprint. Over time seepage to the groundwater will become more representative of that of the outer shell.

In the outer shell, water will be slightly acidic during the first 20 - 40 years at pH 4.5. After about 60 years the pH will be below pH 4.5. Several metals including Al, Co, Cr, Cu, Mn, Ni, Pb, Se, U and Zn will be present in acidic seepage from the tailings (GCS, 2020).

In order to better understand the concentrations of metals possible in tailings seepage water, the GCS (2020) report present results from water samples collected from the under-drain outflow pipes of a typical gold mine TSF. Chemical analysis of the seepage water indicates varying pH and metal concentrations. Table 2.4 present a summary of the results, including minimum, maximum and average measured values of different elements.

**Table 2.4: Summary of gold tailings seepage water analytical results (GCS, 2020).**

Parameter	Min	Max	Avg
	mg.l <sup>-1</sup>		
Aluminium	0.060	0.630	0.250
Antimony	0.000	0.000	-
Arsenic	<0.010	0.130	0.053
Barium	0.010	0.030	0.020
Beryllium	<0.01	0.000	-
Boron	<0.01	0.420	0.217
Cadmium	<0.002	<0.002	-
Chromium	<0.01	0.060	0.046
Cobalt	0.840	4.400	1.593
Copper	<0.002	3.500	0.527
Gold	<0.01	0.01	0.01
Iron	0.030	400.0	85.9
Lead	<0.006	0.050	0.038
Lithium	0.040	0.08	0.061
Manganese	2.90	67.00	21.85
Mercury	-	-	-
Molybdenum	<0.01	0.05	0.05
Nickel	<0.02	7.900	2.675
Selenium	-	-	-
Silver	0.000	0.000	-
Sulphide**	-	-	-
Tin	<0.01	0.000	-
Titanium	<0.01	0.000	-
Uranium	<0.006	1.400	0.540
Vanadium	<0.01	0.060	0.033
Zinc	<0.01	1.300	0.278

Comparison of the measured concentrations listed in Table 2.4 with the elements predicted by the geochemical model to increase in the seepage from the Kareerand TSF over time, shows that aside from Selenium, all of the elements have measurable concentrations in typical gold tailings seepage water. If the concentrations of these elements are to increase in the seepage water over time, the

Kareerand TSF may represent a risk to the chemical quality of groundwater in the area, which could be further increased by the addition of seepage from the Extension Project TSF. Considering the values listed in Table 2.4 concentrations of particularly manganese, cobalt, lead, nickel and uranium may be a future concern.

#### 2.4.2.4 Baseline Water Quality

A hydro census was conducted in the project area as part of the Hydrogeological Impact Assessment (GCS, 2020). The census identified 31 boreholes, most of which are not in use. The boreholes that are in use are located to the north east, south east and south west of the Kareerand TSF and are used for stock watering, irrigation or domestic purposes.

According to GCS, water quality data from the hydro census indicate generally, elevated TDS and sulphate concentrations within the direct vicinity of the Kareerand TSF and to the south towards the Vaal River. The laboratory results indicate that generally Ca and Mg are dominant in most of the hydro census samples. Certain parameters (Cl, NO<sub>3</sub>, Na, Fe, Al and Mn) were elevated above the target water quality guidelines (SANS) in some of the boreholes. Manganese occurs above target levels at most of the sites. Neutral pH levels were recorded at all sites, in particular in the area directly west of the Kareerand TSF.

Using water quality monitoring data provided by AGA, EnviroSim was able to evaluate the current (baseline) impacts to water quality that are likely attributable to the Kareerand TSF. Table 2.5 presents a summary of the minimum, maximum and average concentrations of elements and ions measured in monthly samples of groundwater collected from six monitoring points near the Kareerand TSF. The six monitoring points selected are located in the same direction (east and south west) of the Kareerand TSF as the private use boreholes noted by the Hydrogeological Impact Assessment (GCS, 2020) report.

Figure 2.1 present a map with the locations of the water quality monitoring points around the Kareerand TSF indicated. The selected monitoring boreholes (BH07, BH13, BH16, BH21, HC01 and KD04) are located closer to the Kareerand TSF footprint than the private boreholes and are accepted to represent any contamination that may be moving in the direction of the private boreholes and the Vaal River from the TSF. The monitoring data summarised in Table 2.5 represent monthly measurements collected during 2018 and quarterly measurements from 2019. Comparing the average and maximum values to accepted drinking water quality criteria (SANS 241:2015 and Department of Water & Sanitation Standards) indicate that, as noted from the hydro census data, concentrations of aluminium, iron, manganese, nitrate and sulphate are elevated. A summary of the drinking water quality criteria used for the screening is presented in Table 2.6. Although some of the criteria used for screening relate more to aesthetic or operational effects of the water quality, exceedance of these values are taken as an indication of potentially elevated concentrations.

Table 2.5: Summary of water quality monitoring data for boreholes located east and south west of the Kareerand TSF (2018/2019)

Parameter	BH07			BH13			BH16			BH21			HC01			KD04		
	Ave.	Max.	Min.	Ave.	Max.	Min.	Ave.	Max.	Min.	Ave.	Max.	Min.	Ave.	Max.	Min.	Ave.	Max.	Min.
	mg.l <sup>-1</sup>																	
Nitrate	1.22	4.74	0.25	5.12	6.40	0.55	1.03	1.67	0.74	0.74	1.15	0.53	1.08	1.18	0.91	13.87	16.71	10.59
Sulphate	863.58	1187.04	589.26	645.42	1104.91	477.02	1287.88	1692.96	853.24	1001.08	1264.76	800.66	206.71	249.73	186.12	80.63	170.82	20.85
Chloride	180.08	210.21	130.32	132.99	255.20	95.91	211.63	232.42	185.95	214.89	241.38	198.26	71.79	77.32	66.55	50.99	83.82	24.08
Fluoride	-	-	-	-	-	-	-	-	-	-	-	-	0.67	0.77	0.53	-	-	-
Calcium Carbonate	299.42	399.27	165.49	213.70	289.00	146.59	390.70	517.00	226.99	317.99	431.00	187.20	103.29	121.37	61.95	83.74	103.00	66.70
Cyanide Weak acid dissociable	-	-	-	0.02	0.02	0.02	-	-	-	-	-	-	-	-	-	-	-	-
Aluminium	-	-	-	-	-	-	-	-	-	-	-	-	0.38	0.38	0.38	-	-	-
Iron	-	-	-	-	-	-	0.59	0.59	0.59	4.96	12.46	0.79	-	-	-	-	-	-
Lead	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Magnesium	155.72	187.57	115.44	133.31	175.00	105.48	200.15	239.00	160.00	171.94	203.00	147.91	60.26	64.85	56.00	42.96	53.80	31.40
Manganese	-	-	-	-	-	-	0.22	0.23	0.20	0.62	1.36	0.11	-	-	-	-	-	-
Potassium	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Sodium	31.77	36.40	24.88	27.25	43.90	18.05	49.91	65.66	41.50	48.10	54.56	43.72	78.95	99.03	61.46	24.21	27.90	21.40
Arsenic	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-	-	-	-	-	0.52	0.56	0.49	-	-	-
Uranium	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Zinc	-	-	-	-	-	-	-	-	-	-	-	-	0.11	0.11	0.11	1.33	1.33	1.33
Ammonia	-	-	-	-	-	-	-	-	-	0.55	0.60	0.50	-	-	-	-	-	-
Alkalinity, Total	234.27	285.20	210.92	256.80	289.37	225.60	275.32	330.24	209.60	226.25	360.85	42.55	351.24	382.12	298.50	222.18	237.62	209.56
pH	7.41	7.65	7.10	7.33	7.75	7.09	7.48	7.94	7.10	7.05	7.51	6.20	7.84	8.15	7.38	7.81	8.28	7.41



Figure 2.1 Map of Kareerand TSF indication location of water quality monitoring points.

**Table 2.6: Drinking water criteria used for screening of water quality monitoring data.**

Determinant	Unit	SANS 241 Standards Limits		DWS Drinking Standards
pH at 25°C	pH units	≥5 - ≤9.7		
Aluminum	mg Al/ℓ	Operational ≤0.3		
Ammonia	mg N/ℓ	Aesthetic ≤1.5		
Arsenic	mg As/ℓ		Chronic health ≤0.01	
Boron	mg B/ℓ		Chronic health ≤2.4	
Calcium Carbonate	mg Ca/ℓ			No health. Scaling intensifies from 32mg/L
Chloride	mg Cl/ℓ	Aesthetic ≤300		
Cyanide (Total)	µg CN/ℓ		Acute health ≤200	
Fluoride	mg F/ℓ		Chronic health ≤1.5	
Iron	mg Fe/ℓ	Aesthetic ≤0,3	Chronic health ≤2	
Lead	mg Pb/ℓ		Chronic health ≤0.01	
Magnesium	mg Mg/ℓ			Diarrhoea and scaling issues from 70mg/L
Manganese	mg Mn/ℓ	Aesthetic ≤0,1	Chronic health ≤0.4	
Nitrate	mg N/ℓ		Acute health ≤11	
Potassium	mg K/ℓ			No aesthetic or health effects below 50mg/L
Sodium	mg Na/ℓ	Aesthetic ≤200		
Sulfate	mg SO <sub>4</sub> /ℓ	Aesthetic ≤250	Acute health ≤500	
Uranium	mg U/ℓ		Chronic health ≤0.03	
Zinc	mg Zn/ℓ	Aesthetic ≤5		

Important to note is that mainly soluble salt ions like sodium, calcium, magnesium, chloride and sulphate are measured in the baseline groundwater samples. Elements such as arsenic, lead and uranium are not detected.

#### 2.4.2.5 Summary

Information available for the atmospheric pathway indicate particulate matter as the primary pollutant of concern. By scaling the estimated airborne particulate matter emissions from the existing Kareerand and Expansion Project TSFs with the concentrations of elements typically associated with gold mine tailings it was shown that, under extreme conditions of exposure, concentrations of manganese and uranium may exceed health-risk based guidelines.

Further evaluation of the potential risks to human health associated with the Expansion Project will therefore consider airborne particulate matter as well as particulate associated concentrations of manganese and uranium.

A similar evaluation of the information available for the aquatic pathways indicate that several elements and ions are present in groundwater from monitoring boreholes near the Kareerand TSF at concentrations that exceed drinking water quality criteria. Since many of these elements and ions correspond with the elements and ions measured in seepage water from a typical TSF as well as the list of elements predicted by the geochemical study to increase from the Kareerand tailings over time, measured ions in the groundwater is reasoned to be related to seepage from the Kareerand TSF and so is accepted to represent the baseline impact.

As an indication of the impacts most likely to occur during the operational life of a TSF, the measurements of baseline groundwater quality in boreholes near the Kareerand TSF indicate concentrations of aluminium, iron, manganese, nitrate and sulphate exceeding quality criteria.

Based on this available information, further evaluation of the risks posed to human health from the aquatic pathway will focus on the elements measured in typical tailings seepage, which are known to be toxic to humans and are predicted to in the future increase in concentration in seepage from the Kareerand tailings. These elements are arsenic, aluminium, iron and manganese. In addition to these, uranium will also be evaluated as it is a heavy metal as it is known to be present in significant quantities in the tailings.

Further evaluation of the potential risks to human health associated with the Expansion Project will therefore consider aluminium, arsenic iron, manganese, nitrate sulphate and uranium as contaminants of potential concern relating to the aquatic pathway.

---

## 2.5 HEALTH SIGNIFICANCE OF CONTAMINANTS

---

### 2.5.1 General

The sections that follow present a general discussion of the health significance of the contaminants of potential concern identified for the atmospheric and aquatic pathways. The purpose of this discussion is the further screening of the contaminants to identify those relevant to the further assessment for environmental (non-occupational) exposure.

### 2.5.2 Environmental Health Significance of Particulate Matter

Particulate matter was identified as the main atmospheric pollutant of concern for the proposed extension project. Over the past decade, evidence has accumulated indicating that airborne particulate matter (PM), including PM<sub>10</sub> and PM<sub>2.5</sub>, exert a range of adverse health effects. Statistical evidence suggests that the health effects of particulates occur independently of the presence of other pollutants, such as NO<sub>2</sub> and SO<sub>2</sub> (COMEAP, 2006; COMEAP, 2009; WHO, 2005). The identified health effects are diverse in scope, severity, duration, and clinical significance, but there is general agreement that the cardio-respiratory system is the major target of PM effects. A critical review by the UK Committee on the Medical Effects of Air Pollutants (COMEAP, 2006) indicated that long-term exposure to PM (for years or decades) was associated with elevated total, cardiovascular, and infant mortality, and also with respiratory symptoms and effects on lung growth and immune system function. Short-



term studies showed consistent associations of exposure to daily concentrations of PM with mortality and morbidity on the same day or the subsequent days. Patients with asthma, chronic obstructive pulmonary disease (COPD), pneumonia, and other respiratory diseases; with cardio-vascular diseases and with diabetes were especially affected.

The US EPA (2004) concluded that available short-term exposure studies generally showed positive and statistically significant associations of PM<sub>2.5</sub> with excess total non-accidental and cardiopulmonary mortality. The US EPA also noted that a growing body of evidence showed acute cardiovascular disease morbidity effects of PM and co-pollutants and pointed out the possible roles of gaseous co-pollutants (e.g., CO) as potential confounders of the PM effect on cardiovascular disease.

Potential associations between ambient PM and lung cancer were regularly studied. A US EPA Criteria Document (USEPA, 2004) concluded that the evidence for ambient fine particle (PM<sub>2.5</sub>) exposure relationships with increased lung cancer is much clearer and stronger than for PM<sub>10</sub>. The COMEAP (2006) review presented considerable evidence indicating a lack of association, with only one study indicating that PM<sub>2.5</sub> concentrations were statistically significantly related to lung cancer mortality.

The US EPA (2004) concluded that mixed results were available regarding the potential relationship between PM<sub>10</sub> exposures and increased risks of low birth weight or early postnatal mortality, with some studies reporting significant positive relationships, while others found little evidence. It was also pointed out that these results, overall, highlighted the need for more research to elucidate potential ambient PM effects on foetal development, foetal and postnatal mortality and also on postnatal morbidity.

### 2.5.1 Environmental Health Significance of Aluminium

There are numerous studies that have examined aluminium's potential to induce toxic effects in humans exposed via inhalation, oral, or dermal exposure. Occupational exposure studies and animal studies suggest that the lungs and nervous system may be the most sensitive targets of toxicity following inhalation exposure. Respiratory effects, in particular impaired lung function and fibrosis, have been observed in workers exposed to aluminium dust or fumes. With the exception of some isolated cases, inhalation exposure has not been associated with overt symptoms of neurotoxicity (ATSDR, 2008). There is limited information on aluminium toxicity following dermal exposure. Aluminium is not readily absorbed through the skin (ATSDR, 2008).

No information is available regarding the acute toxicity of aluminium in humans. However, animal studies have shown that acute oral toxicity of aluminium (ATSDR, 2008), there is little indication that aluminium is acutely toxic by oral exposure in humans (EFSA, 2008). Most likely due to its low absorption and high rate of excretion. It has been suggested that aluminium is implicated in the development of Alzheimer's disease and associated with other neurodegenerative diseases in humans. These hypotheses remain controversial and based on the available scientific data, it is generally accepted that oral exposure to aluminium *does not* constitute a risk for developing Alzheimer's disease (EFSA, 2008).

There is no evidence of increased cancer risk in non-occupationally exposed persons and the International Agency for Research on Cancer does not list aluminium itself as a human carcinogen (EFSA, 2008). Aluminium is therefore of low environmental health significance through the ingestion route and is not evaluated further as an aquatic pathway contaminant.

### 2.5.2 Environmental Health Significance of Arsenic

Arsenic is widely distributed in the environment from natural sources and is naturally present at low levels in soil, water, and air. Arsenic is classified chemically as a metalloid, having both properties of a metal and a non-metal; however, it is frequently referred to as a metal. Elemental arsenic, also referred to as metallic arsenic, is rarely encountered in the environment (US EPA, 2010). In compounds, arsenic typically exists in one of three oxidation states, -3, +3, and +5. Arsenic compounds can be categorised as inorganic, compounds without an arsenic-carbon bond, and organic, compounds with an arsenic-carbon bond.

In the environment, there are many processes (chemical and biological) that control the overall fate and impact of arsenic. Arsenic does not break down in the environment but can change from inorganic to organic forms through microbial interaction. Most arsenic compounds are soluble in water but do not evaporate. Arsenic can be released into the air when minerals containing arsenic are processed or smelted, or when materials containing arsenic are burned. Airborne particles that contain arsenic, can settle on the ground, surface water, and plants.

Arsenic in soil can exist in various oxidation states and chemical species, but is largely immobile and tends to remain in upper soil layers. However, reducing conditions can form soluble forms of arsenic that can leach from the soil (ATSDR, 2007).

Analysis of the toxic effects of arsenic is complicated by the fact that arsenic can exist in several different oxidation states and many different inorganic and organic compounds. According to the U.S. Agency for Toxic Substances and Disease Registry (ATSDR), most cases of human toxicity from arsenic have been associated with exposure to inorganic arsenic. Organic forms of arsenic are generally considered to be less toxic than inorganic forms (ATSDR, 2007).

Most cases of arsenic-induced toxicity in humans are due to exposure to common arsenic oxides and oxyacids, and there is an extensive database on the human health effects of these compounds. Although there may be some differences in the potency of different chemical forms, these differences are usually minor (ATSDR, 2007).

Non-cancer effects associated with inhalation exposure to airborne arsenic include respiratory irritation, nausea, skin effects, and neurological effects. Arsenic is a known human carcinogen by both the inhalation and oral exposure routes. By the inhalation route, the primary tumour types are respiratory system cancers, although a few reports have noted increased incidence of tumours at other sites, including the liver, skin, and digestive tract (ATSDR, 2007). Arsenic will be evaluated as an aquatic pathway contaminant via the ingestion route of exposure.

### 2.5.3 Environmental Health Significance of Iron

Iron is an essential nutrient; required for maintenance of good health. Available data indicate that to protect against the adverse health effects associated with iron deficiency, the RDA (recommended dietary allowance) should be at least 30 mg.day<sup>-1</sup> for pregnant women. If ingested in larger quantities iron can be toxic, causing effects such as irritability, seizures, abdominal pain, vomiting, diarrhoea, lethargy, and coma. However, apart from accidental or deliberate poisoning, ingestion of sufficient iron to cause these effects is unlikely in most individuals. Combined with oxygen or sulphate, iron particulates are treated as a nuisance dust, causing coughing on inhalation or irritation in contact with the eyes.

There is no evidence that iron can cause cancer. Iron has not been assigned a carcinogenicity weight-of-evidence classification by U.S. Environmental Protection Agency (EPA). Iron, at the concentrations likely in tailings seepage, is of low environmental health significance through the ingestion route and is not evaluated further as an aquatic pathway contaminant.

### 2.5.4 Environmental Health Significance of Manganese

Manganese is a naturally occurring element found in rock, soil, water, and food. In humans and animals, manganese is an essential nutrient that plays a role in bone mineralization, protein and energy metabolism, metabolic regulation, cellular protection from damaging free radical species, and formation of glycosaminoglycans. Manganese is usually found combined with other elements such as oxygen, sulphur, and chlorine. Because manganese occurs naturally in the environment, humans are exposed to low levels of manganese in water, air, soil, and food.

Although manganese is an essential nutrient, exposure to high levels via inhalation or ingestion may cause some adverse health effects. Inhalation of manganese dust at mining or ore processing plants and inhalation of welding fumes may be significant sources of occupational exposure. Following inhalation of manganese dust, absorption into the bloodstream occurs only if particles are sufficiently small to be able to penetrate deeply into the lungs. Long-term inhalation of manganese dust may result in a neurological disorder characterized by irritability, difficulty in walking, and speech disturbances. Impotence and loss of libido also have been reported in men exposed to high levels of manganese in air. Short-term inhalation exposure has been associated with respiratory disease (ATSDR, 2012).

Although the limited human data and extensive animal data clearly identify neurobehavioral changes as the most sensitive effect from oral exposure to excess inorganic manganese, inconsistencies in the dose-response relationship across several of the studies make it difficult to derive oral exposure risk levels. Reports of neurobehavioral effects in children associated with elevated concentrations of manganese in drinking water were evaluated as the possible basis for determining chronic exposure duration toxicity. However, the data was assessed to be unsuitable due to uncertainties about other possible confounding exposures to neurotoxic agents in the drinking water or via food and/or the lack of information about dietary intakes of manganese by the children.

There is no evidence that manganese causes cancer in humans. The U.S. EPA has placed manganese in Group D, not classifiable as to human carcinogenicity (ATSDR, 2012).

Based on the information presented, manganese exposure through the inhalation route is considered the only significant mode of exposure. Data indicates that oral exposure to manganese only present health effects at very high concentrations and chronic exposure, both of which are unlikely to occur from tailings seepage.

#### 2.5.5 Environmental Health Significance of Nitrate

Nitrate ( $\text{NO}_3^-$ ) is a natural inorganic ion of nitrogen that occurs commonly in water, and in some foods and therefore form a natural part of the human diet. Nitrates are part of the nitrogen cycle in nature and naturally occur in soil and water through microbial breakdown of animal and human organic wastes. This breakdown process converts wastes into ammonia, which then oxidizes into nitrite ( $\text{NO}_2^-$ ) and nitrate. Nitrate-containing compounds in the soil are generally soluble, which means they dissolve easily in water and thus flow easily into ground- and surface water (US EPA, 2007).

Nitrates (e.g., potassium nitrate and ammonium nitrate) are common ingredients of fertiliser. Nitrate contamination of drinking water is of special concern in agricultural areas because of fertilisers applied to crops that are converted to nitrate in the soil and then seep into groundwater. Certain leafy and root vegetables are naturally high in nitrates, but nitrates are also added as preservatives to some foods, such as processed meat (US EPA, 2007).

The primary health concern regarding oral exposure to high concentrations of nitrate is the formation of methaemoglobinemia, so-called "blue-baby syndrome". Nitrate is reduced to nitrite in the stomach of infants, and nitrite is able to oxidise haemoglobin so that it becomes unable to transport oxygen around the body, resulting in methaemoglobinemia, which a blue coloration of the skin and, at higher concentrations, severe oxygen deficiency (ATSDR, 2011). Infants younger than 4 months of age exposed to high levels of nitrates or nitrites are especially prone to acute acquired methaemoglobinemia (IRIS, 2002).

#### 2.5.6 Environmental Health Significance of Uranium

Uranium is poorly absorbed following inhalation, oral, or dermal exposure and the amount absorbed is heavily dependent on the solubility of the compound. Only 0.76% to 5% of inhaled uranium and 0.1% and 6% of ingested uranium typically get into the bloodstream following exposure and only a very small quantity of water soluble uranium compounds can be absorbed through the skin.

The health effects of naturally occurring uranium evaluated here are those due to chemical effects of uranium on the human body and is not related to radiation. According to the U.S. Agency for Toxic Substances and Disease Registry (ATSDR, 2011), the main target for non-cancer health effects associated with exposure to uranium is the kidneys. Kidney damage has been observed in humans and animals after inhaling or ingesting uranium compounds. The level of damage depends on the solubility of the uranium with water-soluble uranium compounds resulting in kidney effects at lower doses than following exposure to insoluble uranium compounds.

Inhaled insoluble uranium compounds have been known to damage the respiratory tract, however, the observed effects are likely attributable to the irritation caused by the solid particulates rather than the chemical effect of uranium. No other non-cancer health effects have been consistently found in humans after inhaling or ingesting uranium compounds. Naturally occurring uranium has not been classified with respect to carcinogenicity by either the United States National Toxicology Program, the International Agency for Research on Cancer or the EPA.

The assessment endpoint for uranium is therefore limited to non-cancer health effects and both inhalation and ingestion exposure will be considered.

### 2.5.7 Environmental Health Significance of Sulphate

The presence of sulphate in drinking water at concentrations above 200 mg.l<sup>-1</sup> can cause noticeable taste (DWAf, 1996), and very high levels might have a laxative effect in people not accustomed to high sulphate concentrations. Taste impairment varies with the nature of the associated cation; taste thresholds have been found to range from 250 mg.l<sup>-1</sup> for sodium sulphate to 1 000 mg.l<sup>-1</sup> for calcium sulphate. It is generally considered that taste impairment is minimal at levels below 250 mg.l<sup>-1</sup>.

No health-based guideline value has been derived by the WHO for sulphate. However, because of the gastrointestinal effects resulting from ingestion of drinking water containing high sulphate levels, it was recommended that health authorities be notified of sources of drinking water that contain sulphate concentrations in excess of 500 mg.l<sup>-1</sup> (WHO, 2006). The USEPA has developed a secondary drinking water regulation of 250 mg.l<sup>-1</sup>, which is a non-enforceable guideline regarding cosmetic effects (such as tooth or skin discoloration) or aesthetic effects (such as taste, odour, or colour) of drinking water (US EPA, 2006).

Levels of sulphate measured in the groundwater near the Kareerand TSF is reported as elevated and concentrations above 2 000 mg.l<sup>-1</sup> is predicted for tailings seepage (GCS, 2020). The increase in the concentrations of sulphate estimated as part of the GCS investigation, indicate concentrations of 200 mg.l<sup>-1</sup> reaching the Vaal River, in a scenario where no mitigation is applied. With the already high concentrations of sulphate resulting from the existing Kareerand TSF, it is considered unlikely that the contribution from the proposed Extension Project TSF will significantly increase ambient concentrations of sulphate in groundwater. Furthermore, considering the aesthetic effects of high sulphate concentrations on drinking water, it is considered unlikely that water users will consume affected water in the vicinity of the Kareerand TSF that contain sulphate at a concentration high enough to cause health effects. Based on this sulphate is not considered a contaminant of concern for health effects associated with the aquatic pathway for the Extension Project TSF.

---

## 2.6 SUMMARY

---

Based on the toxicity of the identified contaminants via different routes of exposure, presented above, manganese and uranium will be considered in the evaluation of the atmospheric pathway while evaluation of the aquatic pathway will consider arsenic, nitrate and uranium.

## 3 DOSE RESPONSE ASSESSMENT

---

### 3.1 PRINCIPLES OF DOSE-RESPONSE ASSESSMENT

---

The dose-response assessment (toxicological assessment) is the analysis of the relationship between the total amount of a chemical or substance absorbed by the exposed group and the changes developed in the group in reaction to the substance. Dose-response assessment is therefore the process of quantitatively evaluating the toxicity of a given chemical agent, as a function of the dose of the contaminant administered or received, and the incidence of adverse health effects in the exposed population. From this analysis, toxicity values are derived that describe the numerical relationships between the dose quantity and the severity or probability of the resultant health effect. Examples of toxicity values are reference concentrations, reference doses and slope factors.

Toxicity values can be used to quantitatively estimate the potential for adverse effects or the risk of cancer in an exposed population, based on the numerical value of the administered or received dose. The numerical value of the dose is determined as part of the *Exposure Assessment* (in this report presented in Section 4). The process of quantitative estimation of the potential for adverse effects is referred to as *Risk Quantification* (in this report presented in Section 5).

The contaminants of concern identified through the *Hazard Identification* process (see Section 2.6) for the atmospheric and aquatic exposure pathways are discussed in the sub-sections below and toxicity values relevant to each contaminant, which can be used to quantify the potential effects on the exposed populations, are presented.

---

### 3.2 PARTICULATE MATTER

---

#### 3.2.1 Introduction

Exposure to air pollution has been associated with a variety of adverse health effects (see Section 2.5). The evidence of this association, reported in literature, focuses mainly on respiratory and cardiovascular effects attributed to short- and long-term exposure to criteria air pollutants, but it is important to acknowledge that the total impact of air pollution on the population is likely to be dominated by the less severe health effects such as sore throat, common cold, cough, wheeze and shortness of breath. The proportion of the exposed population affected by less severe health effects is much larger than that affected by more severe events such as admission to hospital and death (WHO, 2005). Nevertheless, effects including increased risk of mortality and reduced life expectancy are most often considered in risk analysis, owing usually to the better availability of routinely collected data on these health outcomes (WHO, 2005).

To quantify the impacts on the health of communities from air pollution, figures referred to as *risk factors* that relate an observed change in air concentrations of certain pollutants to hospitalisation or mortality rates, are used. Risk factors for long-term and short-term exposure to various air pollutants are obtained from studies reported in the international scientific literature. These studies use

statistical methods to compare changes in reported hospitalisation or mortality rates with observed changes in air concentrations of specific pollutants and consider large amounts of data collected in several cities all over the world. These risk factors are reported for specific modes of exposure (e.g. short-term or long-term exposure). These exposure modes, in turn, can be related to specific types of air quality information such as hourly maximum, 24 hour or annual average concentrations of pollutants for risk quantification.

As explained in a report by the UK Department of Health Committee on the Medical Effects of Air Pollutants (COMEAP, 2006), the risk factors derived from time-series studies generally refer to the effects of a  $10 \mu\text{g.m}^{-3}$  change in the mean pollutant concentrations. For example, a factor of 1.4% for  $\text{PM}_{2.5}$  and cardiovascular mortality indicates that a  $10 \mu\text{g.m}^{-3}$  increase in the concentration of  $\text{PM}_{2.5}$  is associated with a 1.4% increase in the relevant health outcome, in this case cardiovascular mortality. Thus, if 70 people die each day from all cardiovascular causes, a  $10 \mu\text{g.m}^{-3}$  increase in  $\text{PM}_{2.5}$  will increase the daily deaths due to cardiovascular causes by 1.4%, or about one (1), from 70 to 71 deaths.

It is generally accepted that there is no threshold in particle concentrations below which health would not be jeopardised. Evidence discussed in a report on the long term effects of exposure to air pollution (COMEAP, 2009), indicate a linear relationship between exposure to PM and various health indicators. The data further present no evidence that the line representing the relationship between PM concentration and effect decreases in slope as it approaches low concentrations (COMEAP, 2009). It is therefore accepted that there are health effects for concentration levels from  $0 \mu\text{g.m}^{-3}$ . This implies that even though concentrations of airborne PM may be within ambient air quality guidelines, the occurrence of health effects cannot be excluded.

The studies of correlations between health outcomes and PM concentrations report risk factors for both long- and short-term exposures. Across all studies the results indicate a significant difference in risk factors for short-term exposure as opposed to the risk associated with long-term exposure to the same change in PM concentration. This observed difference is reflected in the risk factors selected for the evaluation of the proposed Expansion Project. All risk factors selected are, where possible, derived from single pollutant models that focus on particulates (i.e. excluding cumulative effects of other pollutants).

The COMEAP (2009) report discusses the question of which index of the air pollution mixture should be considered as the principal metric to be used in quantifying the effects of long-term exposure to air pollution. The report (COMEAP, 2009) suggest, that based on reviews of the studies available at that time on PM exposure and mortality, it is concluded that the association of mortality with the concentrations of fine PM ( $\text{PM}_{2.5}$ ) were clearer and more significant than the association with particle sizes greater than  $\text{PM}_{2.5}$ .  $\text{PM}_{10}$ , on the other hand, appears to have a weaker effect on the relative risk of death from all-causes than  $\text{PM}_{2.5}$ . The evidence as a whole points to  $\text{PM}_{2.5}$  as the most satisfactory index of particulate air pollution for quantitative assessments of long-term exposure.

The assessment of the health effects, specifically from long-term exposure to PM associated with the proposed Expansion Project, will therefore focus on the concentrations of  $\text{PM}_{2.5}$  reported in the Air Quality Specialist Report (Airshed, 2020). Effects relating to short term exposure are evaluated with

risk factors for concentrations of PM<sub>10</sub> as the existing body of epidemiological evidence is insufficient to reach a conclusion on the short term exposure–response relationship to fine particles (PM<sub>2.5</sub>).

The discussions presented in the sections below consider both short- and long-term exposure to particulate matter. Only mortality risk factors are listed for the different exposure modes considered, as the data available for the population of the North West Province, and more specifically the Dr Kenneth Kaunda District Municipality where the proposed Extension Project is located, only report statistics for mortality.

### 3.2.2 Short-term exposure to particulates

In the global update of the WHO Air Quality Guidelines (WHO, 2005), results of short term mortality effects of PM<sub>10</sub> for studies of 29 cities in Europe and 20 cities in the US are presented. These studies reported risk factors of 0.62% and 0.46%, respectively, per 10 µg.m<sup>-3</sup> increase in 24 hour average PM<sub>10</sub> concentrations. An analysis of 29 cities from outside Europe and the US reported an effect of 0.5%, which correlates well with the 0.49% reported for Asian cities. Based on these results the WHO concluded that the risks of mortality associated with PM<sub>10</sub> were likely to be similar in cities in developed and underdeveloped countries at around 0.5% per 10 µg.m<sup>-3</sup> (WHO, 2005).

The risk factor for cardiovascular mortality (0.9% per 10 µg.m<sup>-3</sup>) was derived by COMEAP, based on statistical analysis of 40 epidemiological studies (COMEAP, 2006).

**Table 3.1 Short-term PM<sub>10</sub> risk factors for mortality.**

Health Effect	Percentage increase in risk per 10 µg.m <sup>-3</sup> PM10 increase	Reference
Total (non-accidental) mortality	0.5	WHO 2005
Cardiovascular mortality	0.9	COMEAP 2006

### 3.2.3 Long-term exposure to particulates

COMEAP (2009) conducted a review of the long-term significance of air pollutants and concluded that in terms of particulate matter, the best studied health effects and those recommended for quantification are; all-cause, cardio-pulmonary and lung cancer mortality.

The associations between long-term PM<sub>2.5</sub> exposure and associated health effects reported in long-term exposure studies (Dockery, et al., 1993; Jerrett, et al., 2005; Pope, et al., 1995), were reviewed to identify appropriate risk factors linking long-term exposure to air pollution and mortality. COMEAP concluded from the review that the preferred risk factors are derived from the cohort study by the American Cancer Society (ACS) (Pope, et al., 2002), as it is the most extensive and its data and methods have been the most intensively reviewed by other research groups. Several factors for different health endpoints were reported in the ACS study and COMEAP (2009) indicates that risk factors based on PM, represented as PM<sub>2.5</sub>, for all-cause mortality, supplemented by factors for cardiopulmonary and for lung cancer, are the most appropriate to choose for quantification of health effects from exposure to PM.



A summary of the risk factors for mortality associated with long-term PM<sub>2.5</sub> exposure, which will be used for assessment of annual average particulate concentrations associated with the proposed Project, are presented in Table 3.2.

**Table 3.2 Long-term PM<sub>2.5</sub> risk factors for mortality (COMEAP, 2009; Pope, et al., 2002).**

Health Effect	Percentage increase in risk per 10 µg.m <sup>-3</sup> PM <sub>2.5</sub> increase
Total (non-accidental) mortality	6
Cardiopulmonary mortality	9
Lung cancer mortality	14

### 3.3 TOXICITY OF AIRBORNE MANGANESE

As indicated in Section 2.5.4, adverse effects resulting from manganese exposure in humans are associated primarily with inhalation in occupational settings such as dust in ore processing plants and welding fumes. Inhaled manganese is often transported directly to the brain before it is metabolized by the liver. The symptoms of manganese toxicity may appear slowly over months and years. Manganese toxicity can result in a permanent neurological disorder known as manganism with symptoms that include tremors, difficulty walking, and facial muscle spasms. These symptoms are often preceded by other lesser symptoms, including irritability, aggressiveness, and hallucinations.

Although the workplace is the most common source of excess inhalation of manganese, frequent inhalation of fumes from welding activities in the home can produce a risk of excess manganese exposure leading to neurological symptoms. Environmental exposures to airborne manganese have been associated with similar preclinical neurological effects and mood effects as are seen in occupational studies. Acute or intermediate exposure to excess manganese also affects the respiratory system. Inhalation exposure to high concentrations of manganese dusts (specifically manganese dioxide [MnO<sub>2</sub>] and manganese tetroxide [Mn<sub>3</sub>O<sub>4</sub>]) can cause an inflammatory response in the lung, which, over time, can result in impaired lung function.

A limitation of information gathered from an occupational environment is that it is complicated by the fact that significant oral and dermal exposures are also likely to occur under these conditions and co exposure to other metals and chemicals is also common. Information of this type is therefore subject to some uncertainties. It has to be noted that in the occupation environment, exposure is generally from concentrations of the contaminants in air that are much higher than the concentrations that can be expected in the environment.

Review of the occupational exposure information indicated that many of the studies, especially those dealing with occupational exposures, make the distinction between respirable and total manganese dust. Respirable dust is usually defined by a particular dust particle size that varies from study to study. It is typically defined as those particles ≤5 microns; these smaller dust particles can enter the lower areas of the lungs, including the bronchioles and the alveoli. These particles can be absorbed by the lung and will enter the bloodstream immediately, thus avoiding clearance.

The EPA derived a chronic inhalation RfC of  $5 \times 10^{-5} \text{ mg.m}^{-3}$  for respirable manganese (ATSDR, 2012). This value is based on the LOAEL of  $0.15 \text{ mg.m}^{-3}$  from a study of battery workers exposed to manganese dioxide. The RfC will be used for assessment of potential inhalation exposure to manganese present in the airborne particulate matter associated with the proposed Expansion Project.

---

### 3.4 TOXICITY OF AIRBORNE AND WATER BOURNE URANIUM

---

Most of the information on human exposure to uranium derives from occupational settings such as uranium mining and mineral processing operations. A limitation of information gathered from an occupational environment is that it is often confounded by co exposure to other contaminants. Furthermore, occupational exposure is generally from concentrations that are much higher than those expected in the environment, making it difficult to extrapolate the effects to low concentration environmental exposures. Epidemiological information of this type is therefore subject to some uncertainties.

As indicated in Section 2.5.2, current evidence suggests that the toxicity of uranium is mainly due to its chemical damage to kidney tubular cells following exposure to soluble uranium compounds and the respiratory tract following chronic inhalation exposure to insoluble uranium compounds. Other potential targets of toxicity include the reproductive system and the developing organism.

According to the ATSDR (2011), there are limited data on the renal toxicity of uranium following inhalation exposure in humans. A number of studies report no alterations in mortality due to renal disease in uranium workers, while study of uranium mill workers found evidence of renal dysfunction, the severity and incidence of which appeared to be related to exposure duration.

Several epidemiology studies have found associations between nonspecific parameters of renal dysfunction and elevated uranium levels in drinking water. Although these studies provided information on a large range of exposure levels; the human oral exposure studies do not provide reliable dose-response data (ATSDR, 2011).

Information observed in animal studies following inhalation, oral, or dermal exposures to uranium compounds confirm the effects on the renal system, but also reiterates the difference in observed effect between soluble and insoluble uranium. Renal effects have been observed in animals exposed to aerosols of soluble uranium compounds at concentrations of  $\geq 0.13 \text{ mg U.m}^{-3}$  for intermediate durations, while no renal effects were observed in animals exposed to  $1.0 \text{ mg U.m}^{-3}$  as insoluble compounds.

General damage to pulmonary structures can occur upon inhalation of insoluble, reactive chemicals such as some uranium compounds. However, evidence of studies on uranium mine workers could not conclusively attribute the observed effects to the presence of uranium. The respiratory diseases reported were aggravated by the insoluble aerosol particles (mine dust) to which these miners were exposed and were consistent with toxicity of inhalable dust particles other than uranium. Similarly,

respiratory effects reported in workers acutely exposed to uranium hexafluoride were caused by hydrogen fluoride, a potent lung irritant, and not directly relatable to the uranium.

Similar to human studies, signs of respiratory irritation have been observed in animal studies. Inhalation exposure to insoluble uranium compounds results in very slight pulmonary lesions in rats and dogs exposed to 16 mg U.m<sup>-3</sup> as uranium trioxide for 4 weeks. Interestingly mild to severe renal tubular necrosis was also observed at this concentration. In contrast, chronic exposure to 5.1 mg U.m<sup>-3</sup> as uranium dioxide for at least 3.5 years resulted in lung fibrosis in monkey and dogs, while renal effects were not observed in either species.

Limited data are available regarding reproductive or developmental effects of uranium in humans. reduced fertility, likely due to reductions in spermatozoa counts, was observed in male mice exposed to  $\geq 5.6$  mg U.kg<sup>-1</sup>.day<sup>-1</sup> in drinking water. However, fertility was not significantly affected in another study on mice in which males and females were treated with up to 14 U.kg<sup>-1</sup>.day<sup>-1</sup>.

Since uranium is weakly radioactive, it has been assumed to be potentially carcinogenic at occupational levels. However, the International Agency for Research on Cancer has no classification for uranium. Although significant increases in the occurrence of respiratory tract cancer (predominantly lung cancer) have been found in numerous studies of uranium miners, radon progeny in the mines, and not the uranium, were clearly identified as the carcinogenic agents.

Using the limited epidemiological evidence and extrapolations from animal studies the ATSDR (2011) report estimates of exposure levels posing minimal risk to humans (MRLs) for uranium. An MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of noncarcinogenic adverse effects over a specified duration of exposure. The MRLs for uranium reported are for acute, intermediate, and chronic duration exposures through both inhalation and oral routes and for both soluble and insoluble compounds. Table 3.3 present a summary of MRL values derived for uranium.

**Table 3.3 – Minimum risk levels for oral and inhalation exposure to uranium**

MRL	Route of Exposure	Exposure Duration	Uranium Solubility
1.0E-04 mg U.m <sup>-3</sup>	Inhalation	Intermediate (15–364 days)	Soluble
8.0E-04 mg U.m <sup>-3</sup>	Inhalation	Chronic	Insoluble
4.0E-05 mg U.m <sup>-3</sup>	Inhalation	Chronic	Soluble
2.0E-03 mg U.kg <sup>-1</sup> .day <sup>-1</sup>	Oral	Acute	Soluble
2.0E-04 mg U.kg <sup>-1</sup> .day <sup>-1</sup>	Oral	Intermediate (15–364 days)	Soluble

Values not listed for a particular duration of exposure is due to available toxicological data not being suitable for derivation of the MRL or that no significant differences in health effects are observed for different exposure times.

The long term MRL for inhalation will be used. As it is uncertain whether the particulate associated uranium dispersed from the Expansion Project is likely to be soluble or insoluble in nature, the more conservative MRL for soluble uranium compounds is selected.

In addition to the MRLs, the US EPA has set a chronic oral exposure reference dose (RfD) for uranium at  $3.0 \times 10^{-3}$  mg uranium per kg body weight per day (USEPA, 1989). The RfD is based on renal effects observed in animals following oral exposure to soluble uranium.

---

### 3.5 TOXICITY OF ARSENIC IN DRINKING WATER

---

There are a large number of studies in humans and animals on the toxic effects of ingested arsenic. In humans, most cases of toxicity have resulted from accidental, suicidal, homicidal, or medicinal ingestion of arsenic-containing powders or solutions or by consumption of contaminated food or drinking water. Reports of death in humans due to ingestion of high doses of arsenic note the most immediate effects are vomiting, diarrhoea, and gastrointestinal haemorrhage. Death may ensue from fluid loss and circulatory collapse.

A precise estimate of the ingested dose is usually not available in acute poisonings, so quantitative information on lethal dose in humans is sparse. From a few recorded cases where known amounts were ingested, the lethal doses ranged from 22 to 121 mg As/kg, with one case of a family of eight that died from ingestion of water containing about 110 ppm of arsenic for a week indicating a dose of about 2 mg As/kg/day (ATSDR, 2007).

Older literature estimate the minimum lethal dose range as 70–180 mg (about 1–3 mg/kg) although death due to chronic arsenic exposure has been reported at lower doses of 0.05–0.1 mg As/kg/day in children between the ages of 2 and 7 years (ATSDR, 2007).

Systemic effects of arsenic exposure include respiratory, gastrointestinal, cardiovascular, dermal, and neurological effects. Effects are observed from both acute and chronic exposures and occur over a wide dose range. The ATSDR derived an acute-duration oral MRL for inorganic arsenic of 0.005 mg As/kg/day based on a LOAEL of 0.05 mg As/kg/day for gastrointestinal effects and facial edema. A chronic-duration oral MRL of 0.0003 mg/kg/day was derived for inorganic arsenic by the ATSDR based on a NOAEL of 0.0008 mg As/kg/day for dermal effects. The EPA (IRIS 2007) has derived a chronic oral reference dose (RfD) of 0.0003 mg As/kg/day for inorganic arsenic, based on a NOAEL of 0.0008 mg As/kg/day for dermal effects and possible vascular complications (ATSDR, 2007).

There is convincing evidence from a large number of epidemiological studies and case reports that ingestion of inorganic arsenic increases the risk of developing skin cancer (ATSDR, 2007). Lesions that commonly occur are multiple squamous cell carcinomas and multiple basal cell carcinomas. Although both types of skin cancer can be removed surgically, they may develop into painful lesions that may be fatal if left untreated. In most cases, skin cancer develops only after prolonged exposure, but one study has reported skin cancer in people exposed for <1 year

Reports from Taiwan, Mexico, and Chile have suggested that chronic oral exposure to arsenic may result in the development of respiratory tumours and increased incidence of lung cancer, the development of bladder cancer, as well as internal tumours of the liver and other tissues in patients with arsenic-induced skin cancer. However, studies in U.S. populations exposed to arsenic in drinking water have not yielded the cancer incidences and health effects noted in these other countries.

Furthermore, most studies of animals exposed to arsenate or arsenite by the oral route have not detected any clear evidence for an increased incidence of skin cancer or other cancers. The basis for this lack of tumorigenicity in animals is at this stage unknown. Consequently, the US EPA has determined a drinking water unit risk of  $5 \times 10^{-5} \mu\text{g}\cdot\text{L}^{-1}$  for arsenic based on the observation of induced skin cancer.

---

### 3.6 TOXICITY OF NITRATE IN DRINKING WATER

---

the primary health concern for high concentrations of nitrate in drinking water is a condition known as methaemoglobinemia. Nitrate is reduced to nitrite in the stomach of infants, and nitrite is able to oxidise the iron in haemoglobin from the  $\text{Fe}^{+2}$  form to the  $\text{Fe}^{+3}$  state. The resulting compound (methaemoglobin) is an abnormal form of haemoglobin that is unable to bind oxygen. The result is that blood containing methaemoglobin has a reduced capacity to transport oxygen from the lungs to other tissues in the body (ATSDR, 2011; US EPA, 2007).

Low levels of methemoglobin occur in normal individuals, with typical values usually ranging from 0.5 to 2.0 %. However, due to the large excess capacity of blood to carry oxygen, normal levels of methemoglobin up to around 10 % are not associated with any significant clinical signs. Concentrations above 10 % may cause a bluish colour to skin and lips (cyanosis), while values above 25 % lead to weakness, rapid pulse and rapid breathing. Death may occur if methemoglobin values exceed 50-60 % (IRIS, 2002).

Conversion of nitrate to nitrite is mostly mediated by bacteria in the gastrointestinal tract. Consequently, the risk of methemoglobinemia from ingestion of nitrate depends not only on the dose of nitrate, but also on the number and type of enteric bacteria in the gastrointestinal tract. In healthy adults, available data suggest about 5 % of a dose of nitrate is reduced to nitrite by bacteria in the mouth (IRIS, 2002). Conversion of nitrate to nitrite may also occur in the stomach if the pH of the gastric fluid is sufficiently high (above pH 5) to permit bacterial growth. This is of concern in adults with diseases such as achlorhydria<sup>1</sup> or atrophic gastritis<sup>2</sup> (IRIS, 2002). It is also of concern in infants, since the infant gastrointestinal system normally has a high pH that favours the growth of nitrate-reducing bacteria. For this reason, infants (especially age 0-4 months) are generally recognised as being the subpopulation most susceptible to nitrate-induced methemoglobinemia. Risk is especially high in infants who are exposed to water that is contaminated with bacteria, since this tends to promote high concentrations of bacteria in the stomach and intestines (ATSDR, 2011; IRIS, 2002).

The US EPA has set a RfD for nitrate 1.6 mg nitrate nitrogen per kg body weight per day (equivalent to about 7.0 mg nitrate ion per kg body weight per day) (ATSDR, 2011; US EPA, 2007).

---

### 3.7 SUMMARY

---

The toxicity values that will be used for the assessment of potential non-cancer and cancer effects relating to the proposed Extension Project TSF, are summarised in Table 3.4. Where available, values

---

<sup>1</sup> A lack of hydrochloric acid in the digestive juices in the stomach

<sup>2</sup> Chronic gastritis with atrophy of the mucous membrane and destruction of the peptic glands

for different exposure periods are listed. The effect estimates for short term and long-term exposure to particulates (Sections 3.2.2 and 3.2.3) are not repeated here.

**Note:** Where values are reported that are either very small numbers or very big numbers, scientific notation is used. For example, a number like 0.000134 is written as 1.34E-04, where E-04 denotes the exponent of minus 4 to the base ten ( $10^{-4}$ ).

**Table 3.4: Summary of toxicity values used in the HHRIA.**

Contaminant of Potential Concern	Acute Exposure	Chronic Exposure	Cancer
Atmospheric Pathway			
Manganese	-	5.0E-02 $\mu\text{g}\cdot\text{m}^{-3}$	-
Uranium	1.0E-04 $\text{mg}\cdot\text{m}^{-3}$	4.0E-05 $\text{mg}\cdot\text{m}^{-3}$	-
Aquatic Pathway			
Arsenic	-	3.0E-04 $\text{mg}\cdot\text{kg}\cdot\text{day}^{-1}$	5.0x10-05 $\mu\text{g}\cdot\text{L}^{-1}$
Nitrate	-	1.6E+00 $\text{mg}\cdot\text{kg}\cdot\text{day}^{-1}$	-
Uranium	-	3.0E-03 $\text{mg}\cdot\text{kg}\cdot\text{day}^{-1}$	-

## 4 EXPOSURE ASSESSMENT

---

### 4.1 INTRODUCTION

---

Exposure assessment provides an estimate of the levels and duration of exposure by considering the environmental distribution of hazardous substances, the environmental pathways involved, potentially exposed receptors and the routes of direct and indirect exposure. The assessment of exposure for the proposed Expansion Project relies on information presented as part of the Air Quality (Airshed, 2020) and Hydrogeological (GCS, 2020) specialist reports. The sections following present selected results from these specialist study reports, for ease of reference.

### 4.2 ATMOSPHERIC PATHWAY

---

#### 4.2.1 Contaminant Dispersion in the Environment

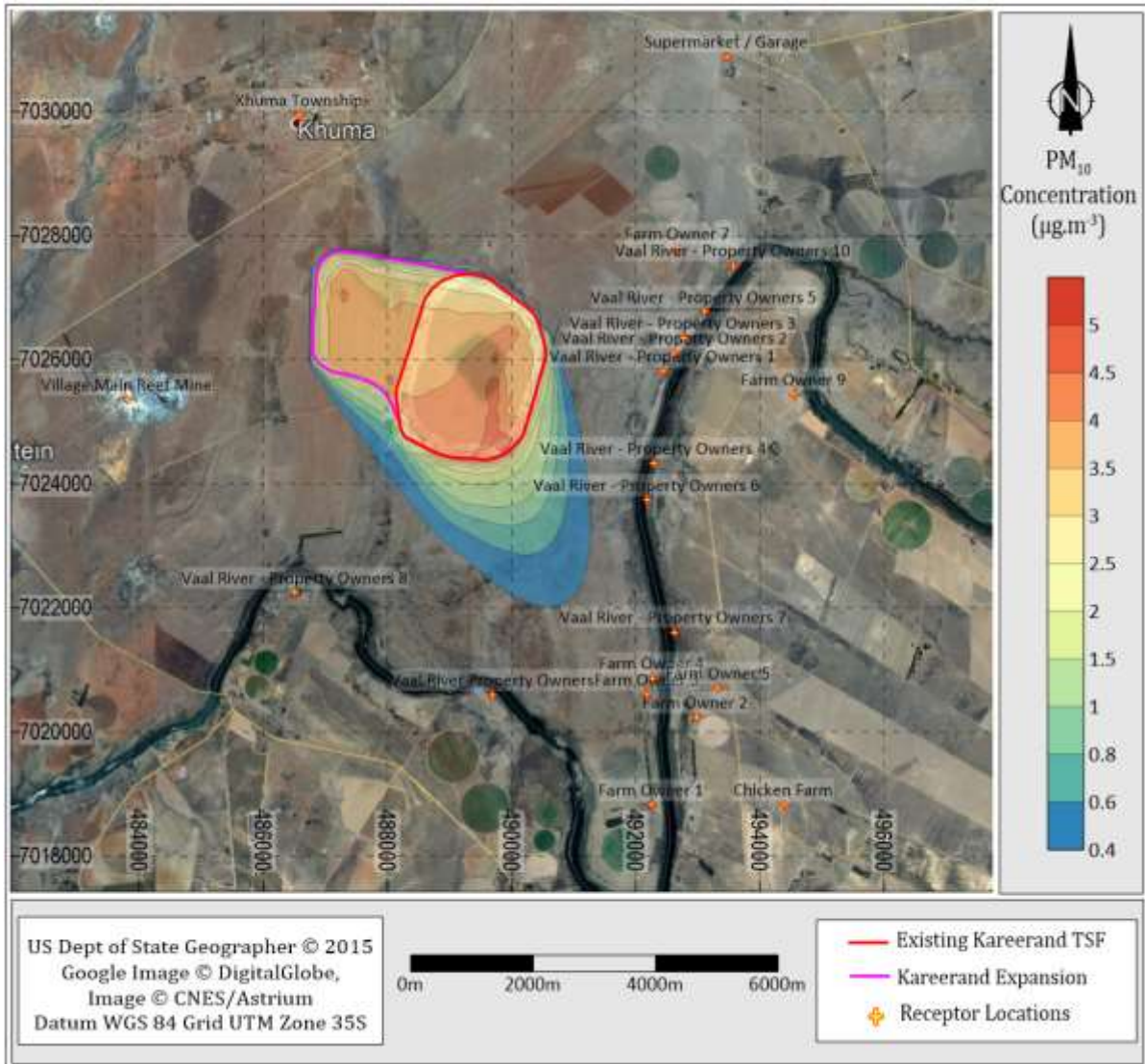
Particulate matter is identified as the primary pollutant of concern for the atmospheric pathway (see Section 2). Based on the information presented in Section 2.6 the contaminants that will be evaluated for the atmospheric pathway are:

- Fine or respirable particulates (PM<sub>2.5</sub>)
- Inhalable particulates (PM<sub>10</sub>)
- Manganese as component of particulates
- Uranium as component of particulates

The Air Quality Specialist Report (Airshed, 2020) includes estimated airborne concentrations of particulates. The airborne concentrations were estimated using a numerical dispersion model. The development of the dispersion model is described in the Air Quality Specialist Report (Airshed, 2020).

As an example of the results obtained from the dispersion model, Figure 4.1 presents a graphical representation of the modelled annual average PM<sub>10</sub> concentrations (including contributions from all sources), as presented by AquSim (2020). The modelled results reported by (Airshed, 2020) for particulates are accepted to represent a reasonable maximum of ambient concentrations associated with the activities at the proposed Expansion Project.

The modelled concentrations are shown as shaded zones with similar concentrations presented by a single colour (concentration isopleths) overlaid on a map of the Project area. The graphical edges of these concentration zones should not be interpreted as concentration boundaries, but rather as a continuum with some overlap between the indicated concentration values. Also, the outside boundary of the concentration isopleths is not a cut-off beyond which there are no more airborne contaminants but is a representation of the extent of the airborne pollutants at the lowest concentration value on the scale. Airborne pollutant concentrations continue beyond this boundary but are all lower than the lowest concentration value on the scale.



**Figure 4.1 Example of particulate dispersion modelling results for the Kareerand and Expansion Project TSFs (AquiSim, 2020).**

#### 4.2.2 Receptors

Airshed (2020) report that the dispersion of pollutants expected to arise from the current Kareerand and Expansion Project TSFs was simulated for an area covering 37.5 km (east-west) by 30.5 km (north-south). Within this area, 31 discrete receptors were identified. The receptors include residential communities such as Stilfontein, Khuma, Village Main Reef and Midvaal Water Company, as well as individual receptors such as farms and properties along the Vaal River. Figure 4.2 present a map of the receptors identified by Airshed.

The dispersion pattern of airborne contaminants, presented in Figure 4.1, indicate that the dispersion is expected to occur in a south-eastern direction from both the source areas. It is assumed that the 25 closest receptor locations selected by Airshed represent the highest exposed individuals and is a true representation of the exposure likely in each of the most exposed areas. It is thus assumed that all 25 the selected receptors conform to the definition of a potential receptor, as presented in Section 1.3.1.



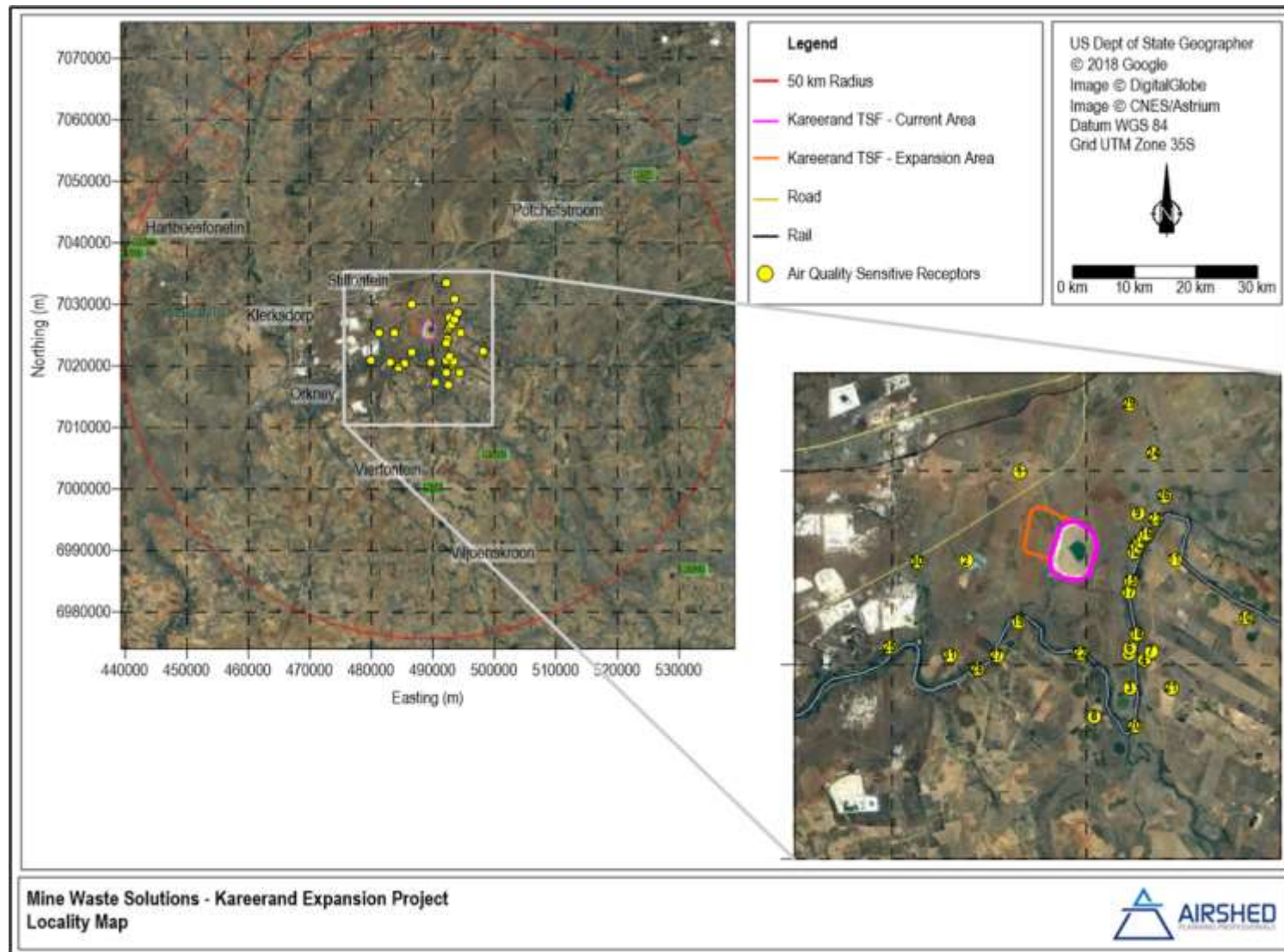


Figure 4.2 Locations of potential receptors identified by Airshed (2020).

### 4.2.3 Results

Airshed (2020) provided the modelled particulate concentrations for different averaging times (i.e. hourly, daily or yearly average concentrations) at the 25 closest receptor locations. The modelled pollutant concentrations that will be used in the HHRIA are selected in accordance with the averaging times of the dose-response data presented in Section 3. The values, as reported by Airshed (2020), are summarised in Table 4.1.

**Table 4.1: Simulated ground level concentrations of contaminants of concern at affected potential receptors identified for the proposed Project.**

Receptor Location		Kareerand TSF		Kareerand + Extension TSF	
		Daily (24-hr) Maximum	Annual Average	Daily (24-hr) Maximum	Annual Average
		PM <sub>10</sub>	PM <sub>2.5</sub>	PM <sub>10</sub>	PM <sub>2.5</sub>
		$\mu\text{g}\cdot\text{m}^{-3}$			
1	Khuma	1.14E-03	5.00E-05	1.70E-04	7.00E-05
2	Village Main Reef Mine	9.36E-02	2.30E-04	1.80E-01	5.40E-04
3	Farm Owner 1	5.92E+00	4.24E-02	1.07E+01	5.80E-02
4	Farm Owner 2	1.32E+01	4.18E-02	1.38E+01	5.32E-02
5	Farm Owner 3	1.58E+01	6.38E-02	2.28E+01	8.09E-02
6	Farm Owner 4	1.75E+01	6.40E-02	2.38E+01	7.98E-02
7	Farm Owner 5	7.83E+00	3.16E-02	9.50E+00	3.94E-02
8	Farm Owner 6	2.07E+00	9.40E-03	3.42E+00	2.08E-02
9	Farm Owner 7	6.48E-01	1.64E-03	1.88E+00	2.94E-03
10	Farm Owner 8	1.77E-01	6.90E-04	2.74E-01	9.70E-04
11	Farm Owner 9	9.18E-01	3.11E-03	1.33E+00	4.13E-03
12	Vaal River - Property Owners 1	2.92E+00	7.35E-03	3.04E+00	9.51E-03
13	Vaal River - Property Owners 2	2.75E+00	5.82E-03	3.00E+00	7.95E-03
14	Vaal River - Property Owners 3	2.64E+00	4.83E-03	3.07E+00	6.97E-03
15	Vaal River - Property Owners 4	3.70E+00	2.15E-02	4.66E+00	2.48E-02
16	Vaal River - Property Owners 5	1.79E+00	3.13E-03	2.94E+00	5.00E-03
17	Vaal River - Property Owners 6	7.28E+00	4.03E-02	8.26E+00	4.55E-02
18	Vaal River - Property Owners 7	1.09E+01	4.91E-02	1.29E+01	5.91E-02
19	Vaal River - Property Owners 8	1.62E+00	1.92E-02	5.56E+00	2.25E-02
20	Vaal River - Property Owner 9	3.65E+00	2.55E-02	6.22E+00	3.87E-02
21	Chicken Farm	5.79E+00	2.02E-02	7.52E+00	2.69E-02
22	Vaal River Property Owners	4.85E+00	2.34E-02	8.74E+00	5.64E-02
23	Vaal River - Property Owners 10	6.53E-01	1.39E-03	1.13E+00	2.70E-03
24	Supermarket / Garage	1.91E-01	2.40E-04	1.99E-01	8.30E-04
25	Midvaal Water Company	1.21E-02	5.00E-05	2.66E-02	1.10E-04

It is assumed that the reported concentration values listed in Table 4.1 include contributions, as relevant to the particular operational area, from all the sources of airborne particulates discussed in Section 2.3.2. Table 4.2 present the concentrations of manganese and uranium estimated in the airborne particulates, based on the elemental composition of the tailings as reported in Table 2.1.

**Table 4.2: Estimated annual average ground level concentrations of particle associated manganese and uranium (see Section 2.4.1 for discussion on derivation of element specific concentrations).**

Receptor Location		Kareerand TSF		Kareerand + Extension TSF	
		Manganese Concentration	Uranium Concentration	Manganese Concentration	Uranium Concentration
		$\mu\text{g}\cdot\text{m}^{-3}$			
1	Khuma	9.38E-07	1.40E-07	1.72E-07	2.57E-08
2	Village Main Reef Mine	7.70E-05	1.48E-04	1.41E-05	2.72E-05
3	Farm Owner 1	4.87E-03	8.81E-03	8.94E-04	1.62E-03
4	Farm Owner 2	1.09E-02	1.14E-02	1.99E-03	2.08E-03
5	Farm Owner 3	1.30E-02	1.88E-02	2.39E-03	3.44E-03
6	Farm Owner 4	1.44E-02	1.96E-02	2.64E-03	3.59E-03
7	Farm Owner 5	6.44E-03	7.82E-03	1.18E-03	1.43E-03
8	Farm Owner 6	1.70E-03	2.81E-03	3.13E-04	5.16E-04
9	Farm Owner 7	5.33E-04	1.55E-03	9.78E-05	2.84E-04
10	Farm Owner 8	1.46E-04	2.26E-04	2.67E-05	4.14E-05
11	Farm Owner 9	7.56E-04	1.09E-03	1.39E-04	2.01E-04
12	Vaal River - Property Owners 1	2.40E-03	2.50E-03	4.41E-04	4.59E-04
13	Vaal River - Property Owners 2	2.26E-03	2.47E-03	4.15E-04	4.53E-04
14	Vaal River - Property Owners 3	2.17E-03	2.53E-03	3.99E-04	4.64E-04
15	Vaal River - Property Owners 4	3.05E-03	3.84E-03	5.59E-04	7.04E-04
16	Vaal River - Property Owners 5	1.47E-03	2.42E-03	2.70E-04	4.44E-04
17	Vaal River - Property Owners 6	5.99E-03	6.80E-03	1.10E-03	1.25E-03
18	Vaal River - Property Owners 7	8.97E-03	1.06E-02	1.65E-03	1.95E-03
19	Vaal River - Property Owners 8	1.33E-03	4.58E-03	2.45E-04	8.40E-04
20	Vaal River - Property Owner 9	3.00E-03	5.12E-03	5.51E-04	9.39E-04
21	Chicken Farm	4.77E-03	6.19E-03	8.74E-04	1.14E-03
22	Vaal River Property Owners	3.99E-03	7.19E-03	7.32E-04	1.32E-03
23	Vaal River - Property Owners 10	5.37E-04	9.30E-04	9.86E-05	1.71E-04
24	Supermarket / Garage	1.57E-04	1.64E-04	2.88E-05	3.00E-05
25	Midvaal Water Company	9.96E-06	2.19E-05	1.83E-06	4.02E-06

---

### 4.3 AQUATIC PATHWAY

---

The Hydrological Impact Assessment report (GCS, 2020) stated as part of its scope, assessment of the risk to groundwater resources and the Vaal River from possible contamination originating from the Kareerand and proposed Extension Project TSFs. In order to do this, information on measured water levels in the area as well as the findings from the geochemistry specialist assessment, were integrated into numerical simulations of contaminant dispersion to groundwater from these source areas.

According to the GCS (2020) report, numerical simulations were used to evaluate the operational and post operational phases of the Kareerand and Extension Project TSFs. Although the construction phase of the Extension Project TSF is discussed, no additional contaminant migration is expected during the construction phase.

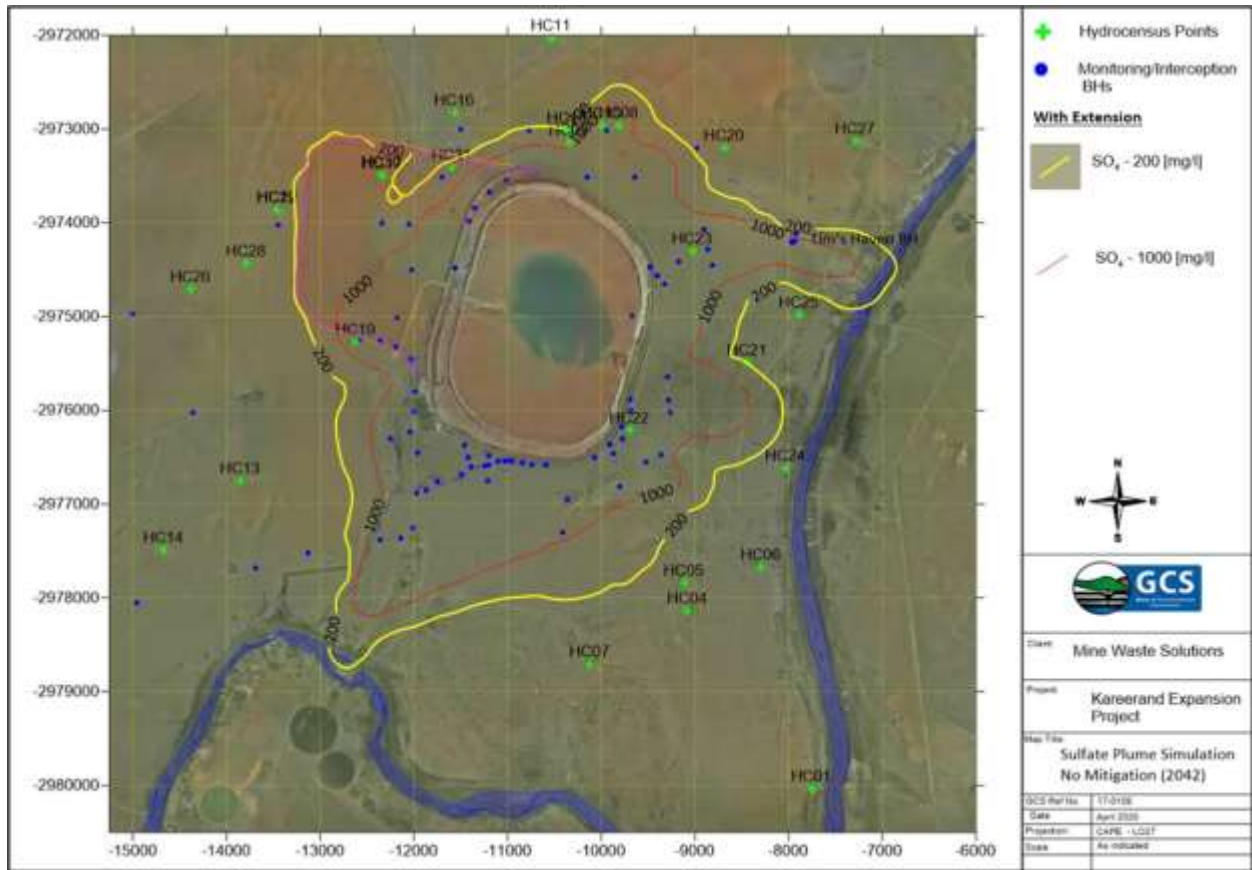
Figure 4.3 is an example of the simulated groundwater contaminant plume predicted for the combined Kareerand and Extension Project TSFs. The numerical simulations were used to evaluate an unmitigated and mitigated scenario, involving the use of groundwater interception boreholes.

According to the GCS (2020) report, the expansion project will have a low impact on the Vaal River and seepage from the existing Kareerand TSF will pose a high to medium risk on the regional aquifer. The results presented in Figure 4.3 indicate the long term predicted contamination plumes for the unmitigated scenario. The results show that contaminated seepage is expected to migrate mainly southwards and eastwards towards the Vaal River and farm boreholes to the north east, east and south east are considered as the primary and most sensitive receivers for the sulphate plume migrating away from the TSF.

The risk on the Vaal River can be managed by interception boreholes. Salt load and concentration increase predictions were made for a mitigated post closure phase. It is predicted that limited TDS increases will occur within the Vaal River if the full mitigation and management is followed, with only 10mg.l<sup>-1</sup> of TDS and approximately 200kg salt load per day expected. This can increase significantly if no or limited mitigation is applied.

Based on the findings of the Geohydrology assessment, the liners and seepage collection integrated into the design of the Extension Project TSF results in it having a negligible effect on contamination of groundwater and surface water resources, compared to the existing Kareerand TSF. Since both the hazard and the pathway of its propagation into the environment is difficult to quantify for the proposed Extension Project, it can reasonably be excluded from further assessment.

However, since the Kareerand and proposed Extension Project are integrated and the impacts to water resources from the Extension Project TSF, however small, will be the same as that observed from the Kareerand TSF. The potential risks associated with the seepage from the Kareerand TSF will therefore be evaluated as 'worst case' estimate of potential impacts associated with the Extension Project TSF.



**Figure 4.3 Simulated potential sulphate migration plume from the Kareerand and Expansion Project TSFs.**

Unfortunately, the Hydrological Impact Assessment report did not explicitly quantify the concentrations of different elements in the groundwater and Vaal River. Although it is possible to use a series of analytical equations to estimate contaminant concentrations based on aquifer characteristics reported by GCS (2020), it is outside the scope of this assessment to do so. The average concentrations as reported from the monitoring measurements (as presented in Table 2.5) as well as values reported for tailings seepage (see Table 2.4), will be used as indicative of a ‘worst case’ water quality impact for the further evaluation of the aquatic pathway.

## 5 RISK CHARACTERISATION

### 5.1 INTRODUCTION

Risk characterisation involves integrating outcomes from the hazard identification, dose-response- and exposure assessments, to determine whether specific exposures to an individual or a community might lead to adverse health effects. The purpose of the risk characterisation presented here is to estimate the probabilities of occurrences of health effects on the communities potentially affected by the proposed Extension Project.

This section describes the methodology used in the quantification of risks associated with exposure to criteria pollutants and hazardous constituents of the airborne particulates, as well as contaminants in the aquatic pathway which are likely to be introduced by activities associated with the Expansion Project. The purpose of these descriptions is to highlight the assumptions and limitations that form part of the results that are presented. Following the descriptions of the approaches followed, the results of the risk characterisation is presented and discussed.

### 5.2 METHODOLOGY OF QUANTIFYING IMPACT

In general, impacts on the health of communities from exposure to airborne particulates is quantified by calculating the potential increase in hospital admissions or in mortality due to specific causes, associated with incremental increases in air concentrations of particulates. These calculations are based on results of 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 concentrations of certain pollutants. Estimates of these effects for environmental exposure to particulates were presented in Section **Error! Reference source not found.** It is important to note that it is not unusual to observe increases in mortality or hospitalisation rates even when the available air concentrations do not exceed environmental air quality guidelines (such as the South African National Ambient Air Quality Standards).

The following equations are used to calculate the potential increase in individual risk associated with increased air concentrations of PM<sub>2.5</sub> and PM<sub>10</sub>. These calculations relate the potential increase in a specific health effect with an incremental increase in pollutant concentration, following the approach of the World Health Organization (Ostro, 1996).

$$E = AF \times P \times B$$

Where:

E	Refers to the potential mortalities per year (or per day) due to exposure to the pollutant
AF	The attributable fraction of mortalities due to exposure to the pollutant
P	Size of the exposed population for this assessment, is set at one (1)
B	The population incidence of mortality (deaths per number of individuals in population)

AF is given by the following equation:

$$AF = \frac{RR - 1}{RR}$$

Where:

RR:	The relative risk of death or illness due to exposure to the pollutant
-----	--

The relative risk of death or illness (RR) is calculated as follows:

$$RR = e^{(\Delta_{deaths} \times \Delta_p)}$$

Where

$\Delta_{deaths}$	Potential proportional change in mortality associated with a 1 $\mu\text{g.m}^{-3}$ change in pollutant concentration
$\Delta_p$	The modelled change in pollutant concentration in $\mu\text{g.m}^{-3}$

The change in the pollutant concentrations ( $\Delta_p$ ) is generally calculated as the difference between concentrations associated with the background (which excludes the contribution of the proposed Project) and concentrations for the background plus the contribution from the proposed Project.

According to the Airshed (2020) report, background pollutant concentrations in the study area have not been quantified and have thus not been included in the modelled values. The ( $\Delta_p$ ) parameter is therefore equal to the modelled pollutant concentrations at the individual receptor locations, as presented in Section 4.2, and represents the incremental contribution from the proposed Project.

In the case of the proposed Kareerand Expansion Project, exposure data were modelled for individual receptor locations in the immediate area. Available information does not include information on the size of the populations residing in the various potentially affected areas. The potential increases in the numbers of mortalities in the populations potentially exposed by the proposed Project could therefore not be directly calculated. The HHRIA thus calculated the potential increases in individual (or personal) risks of mortality experienced at each of the receptor locations, in this case relating to the communities surrounding the Kareerand Tailings Facility. For this reason, the size of the exposed population (P in the equation above), is set at one (1).

This risk assessment relies on the availability of health data and population statistics for quantifying the risk of health effects associated with changes in air concentrations of the criteria pollutants. In the case of the criteria pollutants, mortality or hospitalisation rates for respiratory or cardiovascular causes are the measure of associated illnesses that are mostly referred to in epidemiological studies.

The health data available for the North West Province, and the Dr Kenneth Kaunda District Municipality where the proposed Project is situated, only provides statistics on mortality from different causes. The assessment of the health impact will therefore consider only mortality as endpoint for risk quantification. Data on mortality rates in the Dr Kenneth Kaunda District Municipality were sourced from a Statistics South Africa report on Mortality and the Causes of Death in South Africa for the year 2017 (StatsSA, 2020). Data is available for the years 2006 to 2017. Cause specific mortality data extracted from the 2017 dataset is used for the effect estimate, as it represents the most recent full

set of published mortality data available. Total population numbers for the North West Province and the Dr Kenneth Kaunda District Municipality were found on the StatsSA website (StatsSA, 2016). These data are summarised in Table 5.1. As indicated above, the assessment calculates increases in individual (or personal) risks of mortality. Table 5.1 therefore includes estimates of the baseline mortality rates calculated on a 'per-person' basis.

**Table 5.1: Mortality data for the Dr Kenneth Kaunda District Municipality, for the year 2017.**

Variable	Number of persons		Average incidence of death per person	
	Annual	Average Daily	Average Annual	Average Daily
Total population of South Africa (2013 estimate)	59 308 690			
Total population of North West Province (2016 estimate)	3 597 589			
Total population of Dr Kenneth Kaunda District Municipality (2016 estimate)	398 676			
Person deaths Dr Kenneth Kaunda District Municipality	7 764	21.3	1.95E-02	5.34E-05
Injury deaths Dr Kenneth Kaunda District Municipality	760	2.1	1.91E-03	5.22E-06
Non-injury (non-accidental) deaths Dr Kenneth Kaunda District Municipality	7 004	19.2	1.76E-02	4.81E-05
Cardiovascular deaths Dr Kenneth Kaunda District Municipality	238	0.72	5.97E-04	1.64E-06
Cardiopulmonary deaths Dr Kenneth Kaunda District Municipality	769	2.2	1.93E-03	5.28E-06
Lung cancer deaths (total South Africa)	6 459	17.7	1.09E-04	2.98E-07

The reported incidence of lung cancer mortality is the national statistics reported for 2016. The cause-specific mortality dataset does not report lung cancer mortality figures for the Dr Kenneth Kaunda District Municipality specifically, or for the North West Province. The reason for the omission from the published statistics is because lung cancer is not one of the ten most prominent causes of death in the Province. The national incidence is therefore used with the total South African population to estimate a natural incidence value. Although not ideal, this is a conservative estimate of this effect as statistics from provinces where lung cancer is much more prevalent (i.e. the Western and Northern Cape) is included in this figure.

The values presented in Table 5.1 are interpreted as statistical probabilities of mortality for different causes. For example, based on the 2016 statistics, the probability of any person living in the Dr Kenneth Kaunda District Municipality to die from a health related (non-accidental) cause in any particular year is taken to be 1.76%. That is approximately one out of every 57 people. Similarly, statistically, any person living in the Dr Kenneth Kaunda District Municipality has a baseline chance of approximately one in 20 800 of dying from a health related cause on any particular day of the year.

The results presented in the sections following, estimate the potential increase in this baseline individual risk of daily and annual mortality that can be attributed to the modelled concentrations of criteria pollutants associated with the proposed Expansion Project.



## 5.3 RESULTS

### 5.3.1 Daily (short term) Risks Associated with exposure to Airborne Particulates

The estimated personal daily short-term risks, attributable to particulate emissions associated with the proposed Expansion Project, are given in Table 5.2 and Table 5.3. Attributable risk is that portion of the personal risk that may be directly attributed to the contribution of specific pollutants emitted from the sources associated with the proposed Project.

**Table 5.2: Potential daily increase in personal risk of non-accidental mortality associated with short-term exposure to PM<sub>10</sub>.**

Receptor location		Baseline risk	Incremental increase in personal risk	
			Kareerand TSF	Kareerand + Extension TSF
1	Khuma	4.81E-05	2.74E-11	4.09E-12
2	Village Main Reef Mine		2.25E-09	4.33E-09
3	Farm Owner 1		1.42E-07	2.57E-07
4	Farm Owner 2		3.17E-07	3.31E-07
5	Farm Owner 3		3.79E-07	5.46E-07
6	Farm Owner 4		4.19E-07	5.69E-07
7	Farm Owner 5		1.88E-07	2.28E-07
8	Farm Owner 6		4.98E-08	8.22E-08
9	Farm Owner 7		1.56E-08	4.52E-08
10	Farm Owner 8		4.26E-09	6.59E-09
11	Farm Owner 9		2.21E-08	3.20E-08
12	Vaal River - Property Owners 1		7.02E-08	7.31E-08
13	Vaal River - Property Owners 2		6.61E-08	7.21E-08
14	Vaal River - Property Owners 3		6.35E-08	7.38E-08
15	Vaal River - Property Owners 4		8.90E-08	1.12E-07
16	Vaal River - Property Owners 5		4.31E-08	7.07E-08
17	Vaal River - Property Owners 6		1.75E-07	1.98E-07
18	Vaal River - Property Owners 7		2.62E-07	3.09E-07
19	Vaal River - Property Owners 8		3.90E-08	1.34E-07
20	Vaal River - Property Owner 9		8.78E-08	1.49E-07
21	Chicken Farm		1.39E-07	1.81E-07
22	Vaal River Property Owners		1.17E-07	2.10E-07
23	Vaal River - Property Owners 10		1.57E-08	2.72E-08
24	Supermarket / Garage		4.60E-09	4.79E-09
25	Midvaal Water Company		2.91E-10	6.40E-10

Table 5.2 presents the incremental risk of non-accidental mortality attributable to particulate emissions from the Existing Kareerand TSF and the Kareerand + Extension Project TSFs, as estimated at each of the potential receptor locations. A graphical representation of the results is presented in Figure 5.1. The incremental increase refers to the increase in the baseline statistical risk, as determined from the published statistics for the Dr Kenneth Kaunda District Municipality (see Table 5.1).

The estimated personal daily short-term risk of cardiovascular mortality attributable to the daily maximum concentrations of PM<sub>10</sub> are presented in Table 5.3. The estimated increase is compared to the natural (statistical) risk of cardiovascular mortality in the Dr Kenneth Kaunda District Municipality. The results are graphically represented in Figure 5.1 and Figure 5.2.

**Table 5.3: Potential daily increase in personal risk of cardiovascular mortality associated with short-term exposure to PM<sub>10</sub> emissions.**

Receptor location		Baseline risk	Incremental increase in personal risk	
			Kareerand TSF	Kareerand + Extension TSF
1	Khuma	1.64E-06	1.68E-12	2.50E-13
2	Village Main Reef Mine		1.38E-10	2.65E-10
3	Farm Owner 1		8.69E-09	1.57E-08
4	Farm Owner 2		1.93E-08	2.02E-08
5	Farm Owner 3		2.31E-08	3.32E-08
6	Farm Owner 4		2.56E-08	3.47E-08
7	Farm Owner 5		1.15E-08	1.39E-08
8	Farm Owner 6		3.04E-09	5.03E-09
9	Farm Owner 7		9.54E-10	2.77E-09
10	Farm Owner 8		2.61E-10	4.03E-10
11	Farm Owner 9		1.35E-09	1.96E-09
12	Vaal River - Property Owners 1		4.29E-09	4.47E-09
13	Vaal River - Property Owners 2		4.04E-09	4.41E-09
14	Vaal River - Property Owners 3		3.88E-09	4.51E-09
15	Vaal River - Property Owners 4		5.44E-09	6.85E-09
16	Vaal River - Property Owners 5		2.63E-09	4.32E-09
17	Vaal River - Property Owners 6		1.07E-08	1.21E-08
18	Vaal River - Property Owners 7		1.60E-08	1.89E-08
19	Vaal River - Property Owners 8		2.38E-09	8.16E-09
20	Vaal River - Property Owner 9		5.36E-09	9.13E-09
21	Chicken Farm		8.50E-09	1.10E-08
22	Vaal River Property Owners		7.12E-09	1.28E-08
23	Vaal River - Property Owners 10		9.61E-10	1.66E-09
24	Supermarket / Garage		2.81E-10	2.93E-10
25	Midvaal Water Company		1.78E-11	3.92E-11

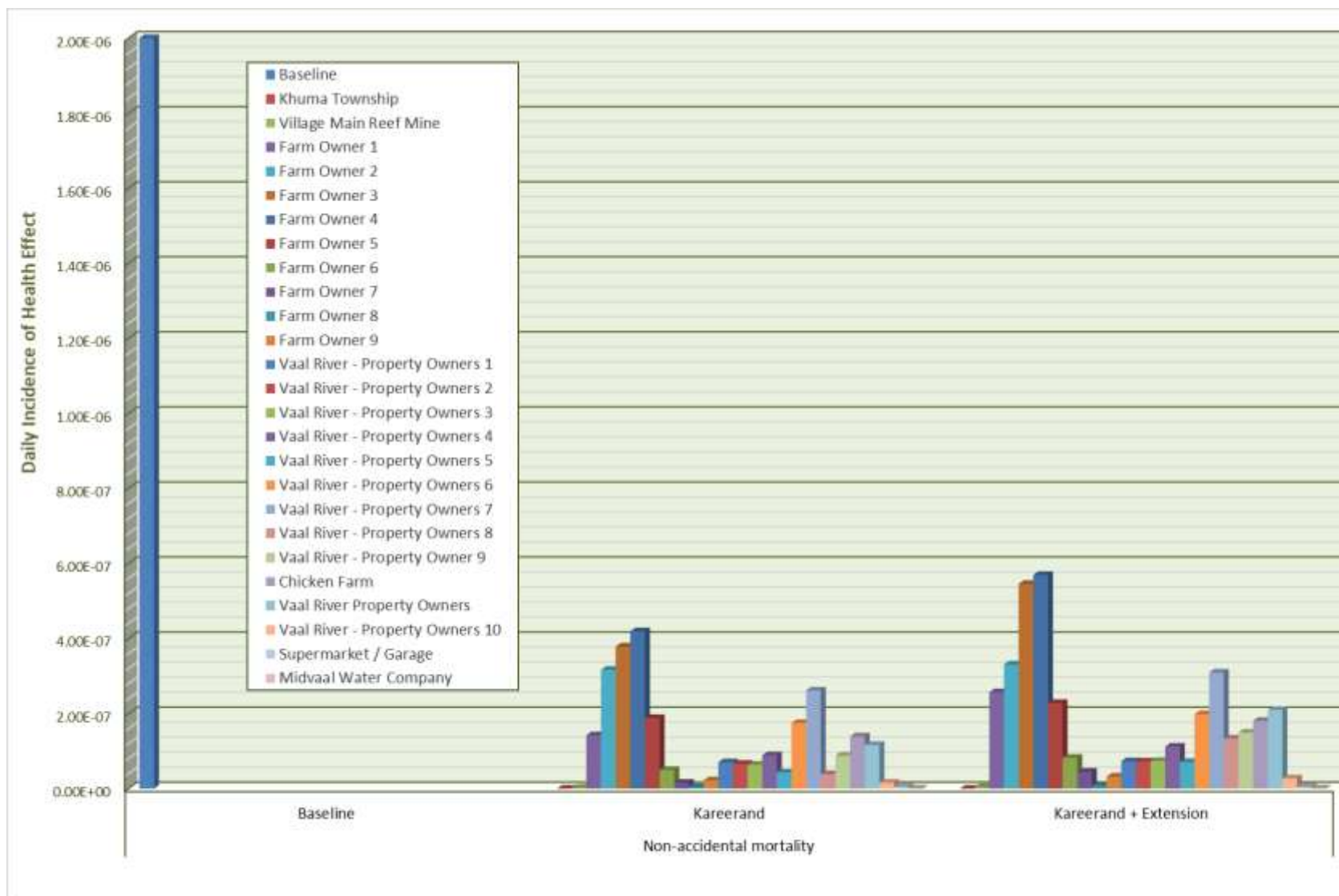


Figure 5.1: Comparison of estimated individual risks of non-accidental mortality associated with short-term exposure to PM<sub>10</sub> from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations.

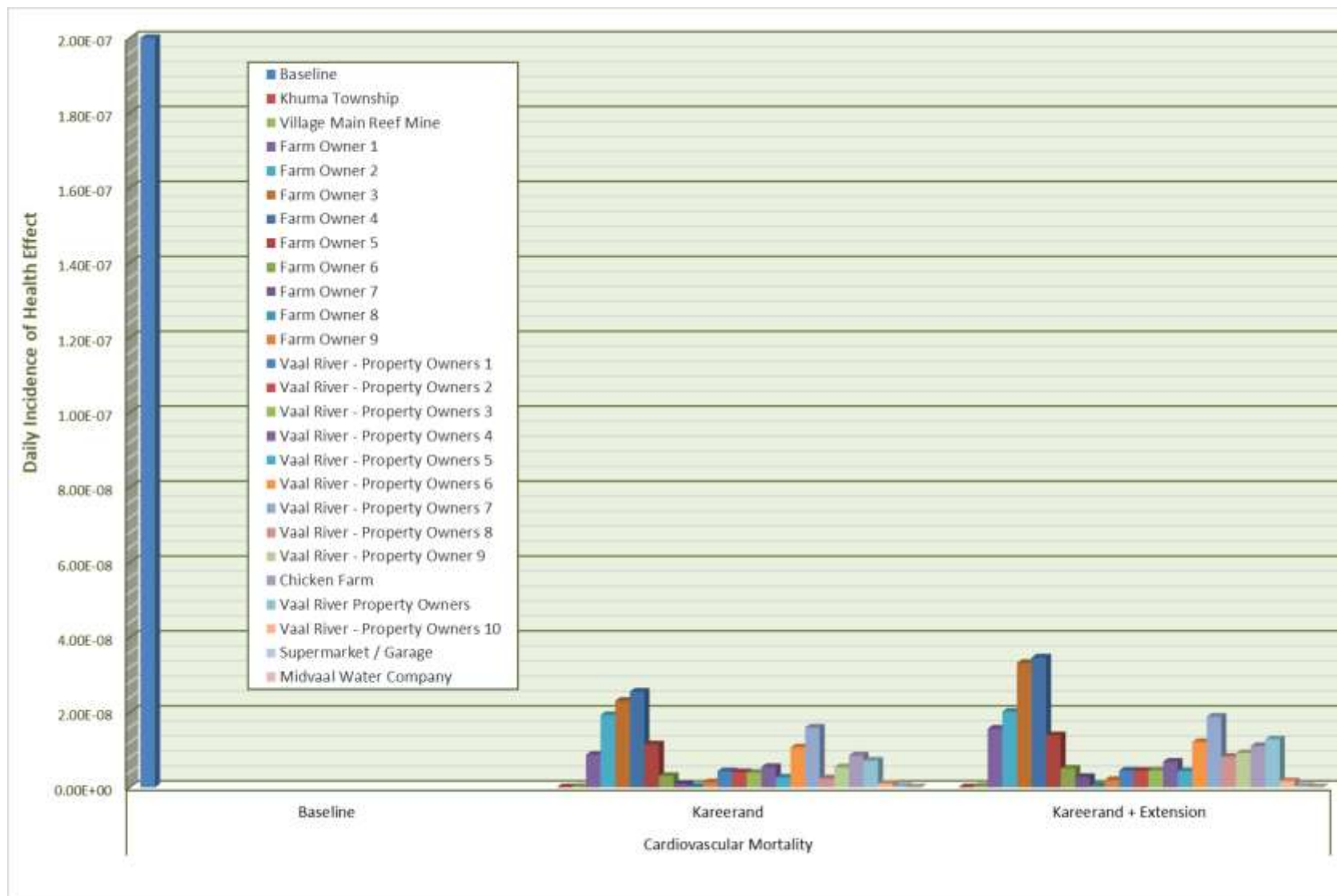


Figure 5.2: Comparison of estimated individual risks of cardiovascular mortality associated with short-term exposure to PM10 from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations.

### 5.3.2 Annual (long term) Risks Associated with Exposure to Particulates

The estimated increase in long-term personal risk of non-accidental mortality, attributable to the concentrations of PM<sub>2.5</sub>, predicted from the Kareerand and Kareerand + Extension TSFs, are listed in Table 5.4. The baseline annual risk of non-accidental mortality for the Dr Kenneth Kaunda District Municipality is also listed for comparison.

**Table 5.4: Potential annual increase in personal risk of non-accidental mortality associated with long-term exposure to modelled PM<sub>2.5</sub>.**

Receptor location		Baseline risk	Incremental increase in personal risk	
			Kareerand TSF	Kareerand + Extension TSF
1	Khuma	1.76E-02	5.27E-09	7.38E-09
2	Village Main Reef Mine		2.42E-08	5.69E-08
3	Farm Owner 1		4.47E-06	6.11E-06
4	Farm Owner 2		4.41E-06	5.61E-06
5	Farm Owner 3		6.72E-06	8.53E-06
6	Farm Owner 4		6.74E-06	8.41E-06
7	Farm Owner 5		3.33E-06	4.15E-06
8	Farm Owner 6		9.91E-07	2.19E-06
9	Farm Owner 7		1.73E-07	3.10E-07
10	Farm Owner 8		7.27E-08	1.02E-07
11	Farm Owner 9		3.28E-07	4.35E-07
12	Vaal River - Property Owners 1		7.75E-07	1.00E-06
13	Vaal River - Property Owners 2		6.13E-07	8.38E-07
14	Vaal River - Property Owners 3		5.09E-07	7.35E-07
15	Vaal River - Property Owners 4		2.27E-06	2.61E-06
16	Vaal River - Property Owners 5		3.30E-07	5.27E-07
17	Vaal River - Property Owners 6		4.25E-06	4.80E-06
18	Vaal River - Property Owners 7		5.17E-06	6.23E-06
19	Vaal River - Property Owners 8		2.02E-06	2.37E-06
20	Vaal River - Property Owner 9		2.69E-06	4.08E-06
21	Chicken Farm		2.13E-06	2.84E-06
22	Vaal River Property Owners		2.47E-06	5.94E-06
23	Vaal River - Property Owners 10		1.47E-07	2.85E-07
24	Supermarket / Garage		2.53E-08	8.75E-08
25	Midvaal Water Company		5.27E-09	1.16E-08

The estimated increases in long-term personal risk of cardiopulmonary mortality attributable to the modelled concentrations of PM<sub>2.5</sub> are given in Table 5.5.

**Table 5.5: Potential annual increase in personal risk of cardiopulmonary mortality associated with long-term exposure to modelled PM<sub>2.5</sub>.**

Receptor location		Baseline risk	Incremental increase in personal risk	
			Kareerand TSF	Kareerand + Extension TSF
1	Khuma	1.93E-03	8.68E-10	1.22E-09
2	Village Main Reef Mine		3.99E-09	9.37E-09
3	Farm Owner 1		7.36E-07	1.01E-06
4	Farm Owner 2		7.26E-07	9.23E-07
5	Farm Owner 3		1.11E-06	1.40E-06
6	Farm Owner 4		1.11E-06	1.38E-06
7	Farm Owner 5		5.48E-07	6.84E-07
8	Farm Owner 6		1.63E-07	3.61E-07
9	Farm Owner 7		2.85E-08	5.10E-08
10	Farm Owner 8		1.20E-08	1.68E-08
11	Farm Owner 9		5.40E-08	7.17E-08
12	Vaal River - Property Owners 1		1.28E-07	1.65E-07
13	Vaal River - Property Owners 2		1.01E-07	1.38E-07
14	Vaal River - Property Owners 3		8.38E-08	1.21E-07
15	Vaal River - Property Owners 4		3.73E-07	4.30E-07
16	Vaal River - Property Owners 5		5.43E-08	8.68E-08
17	Vaal River - Property Owners 6		6.99E-07	7.90E-07
18	Vaal River - Property Owners 7		8.52E-07	1.03E-06
19	Vaal River - Property Owners 8		3.33E-07	3.91E-07
20	Vaal River - Property Owner 9		4.43E-07	6.72E-07
21	Chicken Farm		3.51E-07	4.67E-07
22	Vaal River Property Owners		4.06E-07	9.79E-07
23	Vaal River - Property Owners 10		2.41E-08	4.69E-08
24	Supermarket / Garage		4.17E-09	1.44E-08
25	Midvaal Water Company		8.68E-10	1.91E-09

Table 5.6 presents the estimated increase in long-term personal risk of lung cancer mortality attributable to the modelled concentrations of PM<sub>2.5</sub>.

**Table 5.6: Potential annual increase in personal risk of lung cancer mortality associated with long-term exposure to modelled PM<sub>2.5</sub> concentrations.**

Receptor location		Baseline risk	Incremental increase in personal risk	
			Kareerand TSF	Kareerand + Extension TSF
1	Khuma	1.09E-04	7.62E-11	1.07E-10
2	Village Main Reef Mine		3.51E-10	8.23E-10
3	Farm Owner 1		6.46E-08	8.84E-08
4	Farm Owner 2		6.37E-08	8.11E-08
5	Farm Owner 3		9.72E-08	1.23E-07
6	Farm Owner 4		9.75E-08	1.22E-07
7	Farm Owner 5		4.82E-08	6.01E-08
8	Farm Owner 6		1.43E-08	3.17E-08
9	Farm Owner 7		2.50E-09	4.48E-09
10	Farm Owner 8		1.05E-09	1.48E-09
11	Farm Owner 9		4.74E-09	6.30E-09
12	Vaal River - Property Owners 1		1.12E-08	1.45E-08
13	Vaal River - Property Owners 2		8.87E-09	1.21E-08
14	Vaal River - Property Owners 3		7.36E-09	1.06E-08
15	Vaal River - Property Owners 4		3.28E-08	3.78E-08
16	Vaal River - Property Owners 5		4.77E-09	7.62E-09
17	Vaal River - Property Owners 6		6.14E-08	6.94E-08
18	Vaal River - Property Owners 7		7.48E-08	9.01E-08
19	Vaal River - Property Owners 8		2.93E-08	3.43E-08
20	Vaal River - Property Owner 9		3.89E-08	5.90E-08
21	Chicken Farm		3.08E-08	4.10E-08
22	Vaal River Property Owners		3.57E-08	8.60E-08
23	Vaal River - Property Owners 10		2.12E-09	4.12E-09
24	Supermarket / Garage		3.66E-10	1.27E-09
25	Midvaal Water Company		7.62E-11	1.68E-10

Graphical representations of the results showing comparisons of the incremental increase in health effects associated with long-term exposure to criteria pollutants is presented in Figure 5.3.

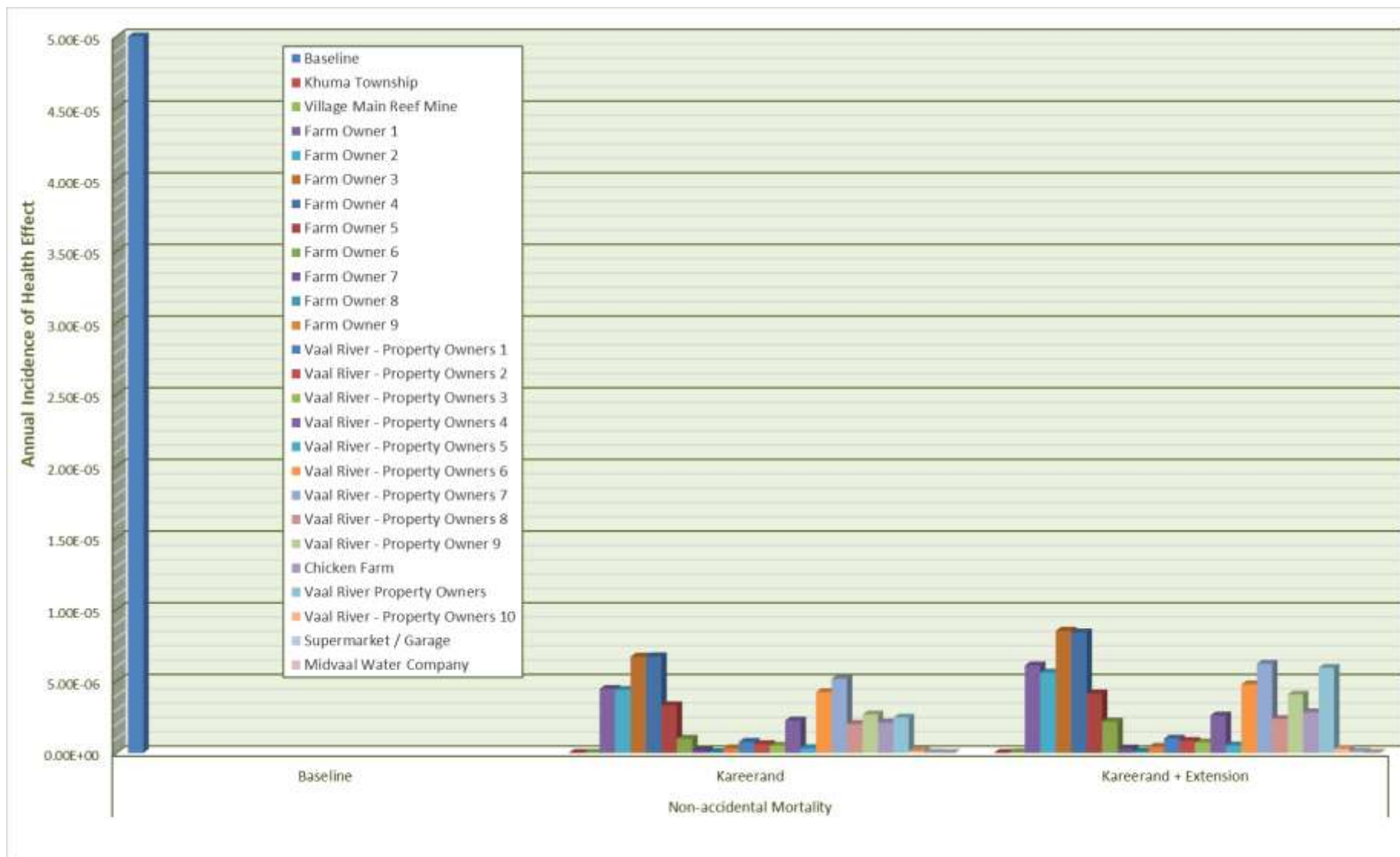


Figure 5.3: Comparison of estimated individual risks of non-accidental mortality associated with long-term exposure to PM<sub>2.5</sub> from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations.



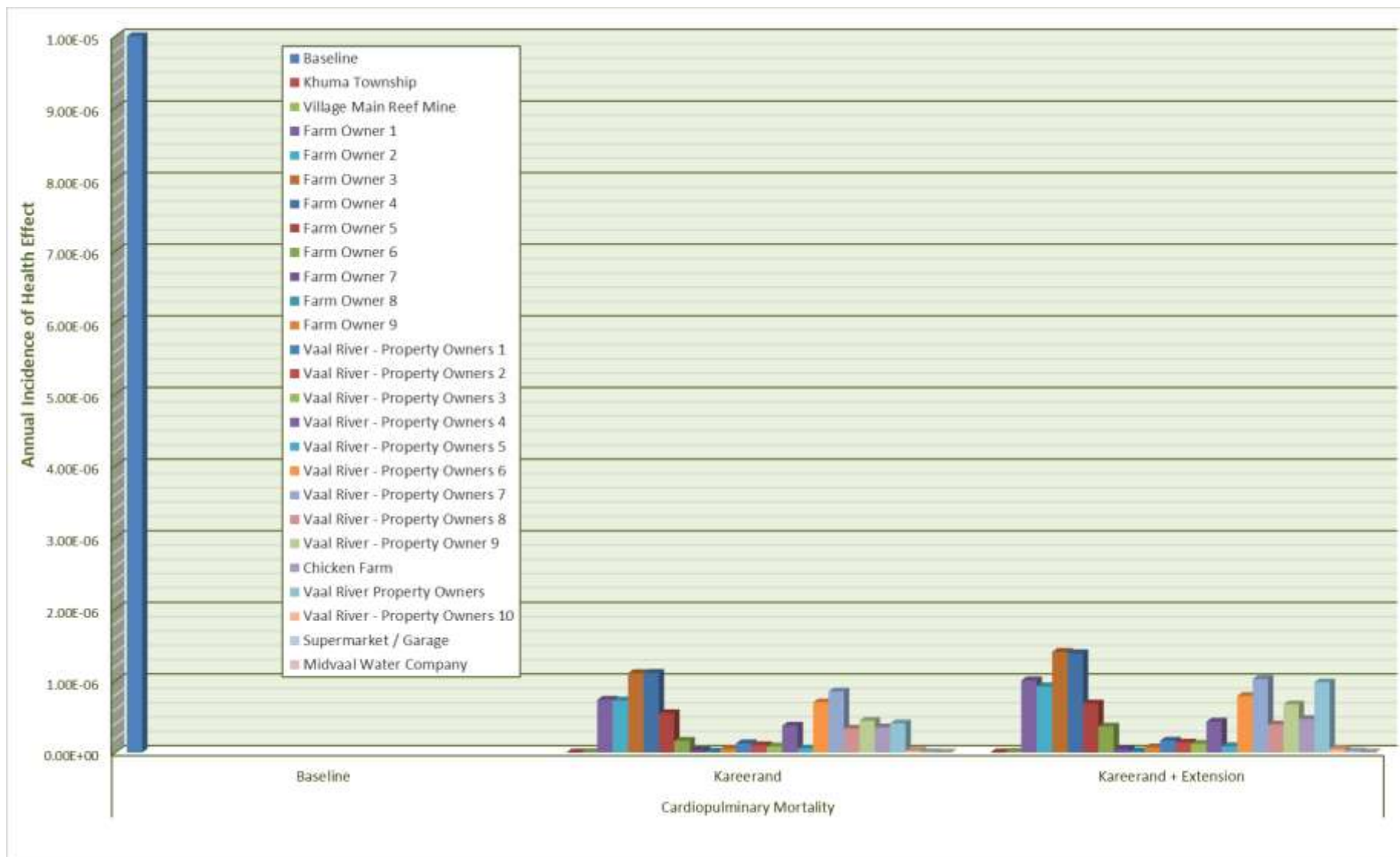


Figure 5.4: Comparison of estimated individual risks of cardiopulmonary mortality associated with long-term exposure to PM2.5 from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations.

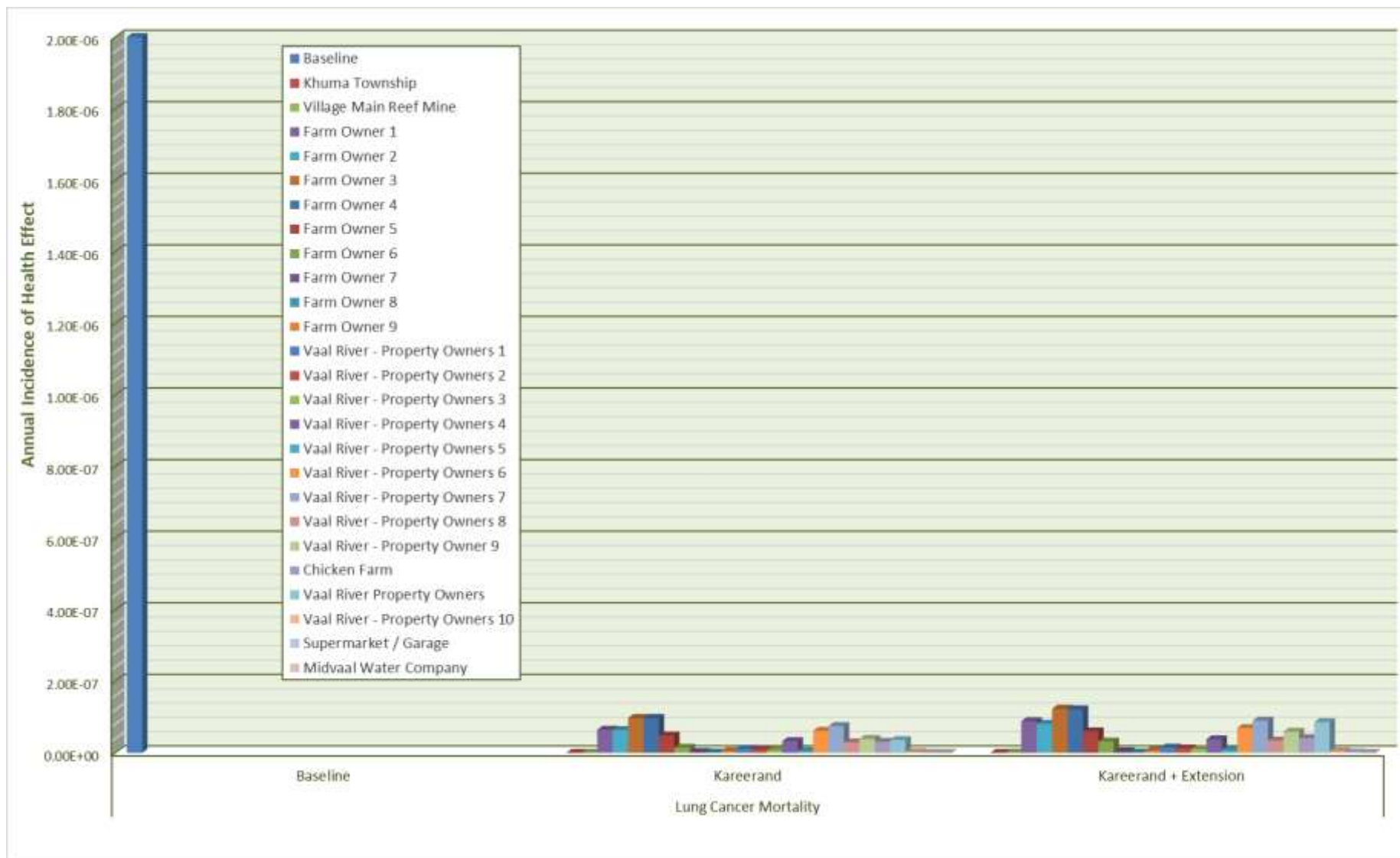


Figure 5.5: Comparison of estimated individual risks of lung cancer mortality associated with long-term exposure to PM2.5 from the Kareerand TSF and the Kareerand + Expansion Project TSFs, at different receptor locations.

### 5.3.3 Discussion of Results

#### 5.3.3.1 General

The values reported in Section 5.3.1 and 5.3.2 indicate the portion of the baseline population risk of health effects attributable to the exposure to modelled concentrations of PM<sub>2.5</sub> and PM<sub>10</sub> predicted for the existing Kareerand TSF and the Kareerand + Extension TSFs.

The results presented indicate that, in general, that short term exposure to particulates has the greatest effect on personal risks experienced at receptor locations

#### 5.3.3.2 Short-Term Risks

In the case of daily risks, the estimated personal risk of total non-accidental mortality from exposure to a single pollutant shows an increase of just over 1% for the combined Kareerand and Expansion TSFs, while personal risks of cardiovascular mortality indicate increases of almost 2.1% from exposure to the daily maximum concentrations of PM<sub>10</sub>, at the same receptor. The receptors where these increases are the clearest are at two farmhouses on the Vaal River, indicated as receptor location 5 and 6 in Figure 4.2.

There is a clear increase in the risk of short-term health effects associated with the addition of the Extension TSF. At receptors located to the south and east of the Extension TSF (receptor location 9 and 19 in Figure 4.2) risk estimates are three times as high compared to the risks associated with the Kareerand TSF alone.

#### 5.3.3.3 Long-Term Risks

Evaluation of long-term exposure to criteria pollutants, showed the increase in personal risk of total non-accidental mortality and cardiopulmonary mortality is very small compared to baseline. The highest estimated increase in baseline risk from a single pollutant is only 0.7% for cardiopulmonary mortality from long-term exposure to PM<sub>2.5</sub> emitted from both the Kareerand and Extension Project TSFs. This highest increase is again observed to the south east of the two TSFs at the number 5 and 6 receptor location (see Figure 4.2)

This much lower increase in the annual personal risk compared to daily risks is due to the much lower quantity of small particulates (<2.5 µm) present in the tailings material, which result in a lower concentration of airborne PM<sub>2.5</sub>.

Similar to the short term risks, the contribution from the Extension Project TSF indicate a three fold increase in annual risks, this time at a different receptor point (indicated as number 24 in Figure 4.2) to the north east of the TSFs.

The estimated increase in the baseline risk of lung cancer mortality is less than 0.12% at all receptor locations, even with the contribution from the Extension TSF included.

### 5.3.3.4 Evaluation

The significance of the increase in personal risk referred to above, is a qualitative statement on the increase estimated as compared to the baseline risk and is a function of the size of the exposed population. As indicated earlier, the baseline risk of non-accidental mortality in the Dr Kenneth Kaunda District Municipality equates to approximately one death per day in every 20 800 people. An incremental increase of for example 20% in the individual risk of non-accidental mortality due to exposure to air pollution, would result in one (1) additional death only if the population exposed to the air pollution includes a minimum of 35 000 people. The population shown to be directly affected by the modelled concentrations of air pollutants from the Kareerand and Extension project TSFs are mostly individual farms or houses. The affected population is therefore smaller than 35 000, which implies that statistically the risk of additional deaths occurring become negligible.

Nevertheless, as qualitative measure of significance a 20% increase in the individual personal risk of a particular effect (as compared with the baseline incidence of that effect), is taken as significant. This is done so that any potential problem areas may be identified. Based on this interpretation, the estimated increases in annual personal risks associated with modelled concentrations of airborne particulates from the proposed Extension Project, is not significant for either short term or annual personal risks at any of the receptors evaluated.

The value of the attributable risk estimates presented above lies in the indication of potential problem areas, rather than in the absolute numbers of the estimated increases in personal risk. The results can therefore be interpreted as pointing to a potential for health impacts to occur at receptor locations in a south easterly direction from the Kareerand TSF, should ambient dust emissions become significantly higher than those estimated in the Air Quality Specialist Report. Also important to note is that a clear increase in baseline health risk is possible at specific receptors located to the south, east and north east of the Kareerand Expansion Project.

---

## 5.4 HEALTH RISKS ASSOCIATED WITH EXPOSURE TO PARTICLE ASSOCIATED CONTAMINANTS

---

### 5.4.1 Calculation of Non-cancer Risk Associated with Inhalation Exposure to Airborne Contaminants

Exposure to non-carcinogenic toxicants through inhalation is normally assessed against a reference concentration (RfC) (USEPA, 2002), if other routes of exposure are not present. A RfC is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a lifetime of exposure. Similarly, a MRL is a measure of exposure levels at which adverse health effects are not expected to occur in humans. MRLs are derived using a modified version of the risk assessment methodology used by the US EPA to derive RfCs for lifetime exposure.

By dividing the estimated concentrations of airborne particle associated pollutants at each of the receptor locations by the RfC or MRL, as appropriate, a hazard quotient (HQ) is calculated. Where a HQ exceeds one, health effects may occur and the situation requires further attention.

The HQs associated with the concentrations of the different contaminants estimated at each receptor location, are presented in Table 5.7. The HQs were estimated using the airborne particulate emission estimates for the combined Kareerand + Extension TSF (see Table 4.1) with the RfC and MRL values reported in Table 3.4.

**Table 5.7: HQs associated with exposure to particle associated contaminants.**

Receptor Location		Airborne Concentration (mg.m <sup>-3</sup> )		Hazard Quotients (HQ)		
		Uranium	Manganese	Soluble U	Insoluble U	Manganese
1	Khuma	2.57E-08	1.40E-07	4.00E-05	8.00E-04	5.00E-05
2	Village Main Reef Mine	2.72E-05	1.48E-04	6.43E-07	3.21E-08	2.80E-06
3	Farm Owner 1	1.62E-03	8.81E-03	6.80E-04	3.40E-05	2.96E-03
4	Farm Owner 2	2.08E-03	1.14E-02	4.05E-02	2.03E-03	1.76E-01
5	Farm Owner 3	3.44E-03	1.88E-02	5.20E-02	2.60E-03	2.28E-01
6	Farm Owner 4	3.59E-03	1.96E-02	8.60E-02	4.30E-03	3.76E-01
7	Farm Owner 5	1.43E-03	7.82E-03	8.98E-02	4.49E-03	3.92E-01
8	Farm Owner 6	5.16E-04	2.81E-03	3.58E-02	1.79E-03	1.56E-01
9	Farm Owner 7	2.84E-04	1.55E-03	1.29E-02	6.45E-04	5.62E-02
10	Farm Owner 8	4.14E-05	2.26E-04	7.10E-03	3.55E-04	3.10E-02
11	Farm Owner 9	2.01E-04	1.09E-03	1.04E-03	5.18E-05	4.52E-03
12	Vaal River - Property Owners 1	4.59E-04	2.50E-03	5.03E-03	2.51E-04	2.18E-02
13	Vaal River - Property Owners 2	4.53E-04	2.47E-03	1.15E-02	5.74E-04	5.00E-02
14	Vaal River - Property Owners 3	4.64E-04	2.53E-03	1.13E-02	5.66E-04	4.94E-02
15	Vaal River - Property Owners 4	7.04E-04	3.84E-03	1.16E-02	5.80E-04	5.06E-02
16	Vaal River - Property Owners 5	4.44E-04	2.42E-03	1.76E-02	8.80E-04	7.68E-02
17	Vaal River - Property Owners 6	1.25E-03	6.80E-03	1.11E-02	5.55E-04	4.84E-02
18	Vaal River - Property Owners 7	1.95E-03	1.06E-02	3.13E-02	1.56E-03	1.36E-01
19	Vaal River - Property Owners 8	8.40E-04	4.58E-03	4.88E-02	2.44E-03	2.12E-01
20	Vaal River - Property Owner 9	9.39E-04	5.12E-03	2.10E-02	1.05E-03	9.16E-02
21	Chicken Farm	1.14E-03	6.19E-03	2.35E-02	1.17E-03	1.02E-01
22	Vaal River Property Owners	1.32E-03	7.19E-03	2.85E-02	1.43E-03	1.24E-01
23	Vaal River - Property Owners 10	1.71E-04	9.30E-04	3.30E-02	1.65E-03	1.44E-01
24	Supermarket / Garage	3.00E-05	1.64E-04	4.28E-03	2.14E-04	1.86E-02
25	Midvaal Water Company	4.02E-06	2.19E-05	7.50E-04	3.75E-05	3.28E-03

The RfC and MRL values used in the calculation of the HQs are for medium term or chronic exposure. However, the airborne contaminant concentrations were estimated using daily maximum PM<sub>10</sub> concentrations. The exposure evaluated therefore represent a very conservative maximum. Nevertheless, the hazard quotients calculated are all well below 1 and indicate the probability of non-

cancer health effects occurring at any of the receptor locations as a result of exposure to either airborne uranium or manganese is very low.

## 5.5 HEALTH RISKS ASSOCIATED WITH CONTAMINANT CONCENTRATIONS IN WATER

### 5.5.1 Calculation of Non-cancer Risk Associated with Ingestion Exposure to Contaminants in Groundwater and Surface Water Resources.

Similar to airborne contaminants, the risk of ingestion exposure to non-carcinogens in groundwater and surface water is evaluated through a hazard quotient (HQ), which is the exposure concentration averaged over the period of exposure divided by the reference dose (RfD) or tolerable daily intake (TDI). Where a HQ exceeds 1, health effects may occur and the situation requires further investigation.

For chronic exposure (2 years+), the estimated average daily dose of a substance received by the ingestion of water can be calculated as follows:

$$CD_{ing} = \frac{C_{water} \times ABS \times IR \times EF \times ED}{BW \times AT}$$

Where:

CD <sub>inges</sub>	Chronic average daily dose of substance in surface water (mg.(kg-day) <sup>-1</sup> )
C <sub>water</sub>	Substance concentration in water (mg.L <sup>-1</sup> )
ABS	Absorption factor (1.0, unitless)
IR	Ingestion rate (litres.event <sup>-1</sup> )
EF	Exposure frequency (events.yr <sup>-1</sup> )
ED	Exposure duration (years)
BW	Body weight (kg)
AT	Averaging time (days)

The exposure parameters required for the equation, intake rates, body weights, exposure frequency and exposure duration, are selected to be representative of adults, as there are no specific sensitive receptor age group identified in the toxicological data. The values of the exposure factors selected for this evaluation are from the higher end of the range of published values and are selected to be conservative and evaluate exposure assuming total reliance on contaminated groundwater as the only source of drinking water. The values selected are listed in Table 5.8 and were obtained from the Exposure Factors Handbook published by the US EPA (2011).

**Table 5.8 Exposure parameters used in estimation of target concentrations.**

Exposure Parameter	Range of Published Values	Selected Value	Unit
IR	0.03 - 2.544 (lower 10 <sup>th</sup> upper 95 <sup>th</sup> percentile) all ages	2.544	L.day <sup>-1</sup>
EF	-	365	days.yr <sup>-1</sup>
ED	-	2	years

Exposure Parameter	Range of Published Values	Selected Value	Unit
BW	51.4 – 63.0 (lower 10 <sup>th</sup> percentile), men and women 21 to 30 years of age	54.7	kg
AT	-	730	days

As discussed in Section 4.3, the impacts to water quality from the Kareerand and Extension Project TSFs will be evaluated using the concentrations of contaminants reported in baseline groundwater and seepage samples collected from the under-drain outflow pipes of a typical gold mine TSF (as reported by GCS and presented in Table 2.4). This is used in the absence of simulated values and are considered as indicative of a 'worst case' water quality impact from the TSFs.

As the values measured in seepage water samples are significantly higher than the concentrations that can be expected in groundwater, a dilution factor of 50 is assumed for the concentrations of arsenic and uranium. The measured concentration of nitrate is used directly from the highest average and maximum values listed in Table 2.5. The estimated dose values for arsenic, nitrate and uranium are listed in Table 5.9. Using the toxicity values listed in Table 3.4, the HQs are calculated and are also listed in Table 5.9.

**Table 5.9: Estimated dose values and calculated HQs from exposure to aquatic pathway contaminants.**

Contaminant		Dose Estimate mg.(kg-day) <sup>-1</sup>	Hazard Quotient
Arsenic	Diluted Ave.	2.46E-04	0.16
	Diluted Max.	6.05E-04	0.4
Nitrate	Measured Ave.	6.45E-01	0.40
	Measured Max.	7.78E-01	0.49
Uranium	Diluted Ave.	2.51E-03	0.17
	Diluted Max.	6.51E-03	0.43

The results indicate that should members of the public consume water with concentrations of either arsenic or uranium at one twentieth the average concentrations measured in seepage from a typical TSF, no health effects are likely to occur. Similarly, no health effects are expected from the baseline concentrations of Nitrate in the groundwater.

A simple evaluation of the effect of dilution indicated that a dilution by a factor of 50 will reduce the risk of health effects well below acceptable levels. However, it has to be kept in mind that the concentrations of arsenic, uranium and nitrate are predicted to increase in seepage water as a result of oxidation and lowering in the pH of the Kareerand TSF. Combining concentrations of these elements in seepage or runoff from the tailings with baseline groundwater and surface water could result in concentrations that have a potential to cause health effects. Care should therefore be taken to avoid seepage from the TSFs entering water resources in the area.

### 5.5.2 Sulphate in Drinking Water

High concentrations of sulphate was identified as potential contaminant for the aquatic pathway. However, no toxicity reference values or risk factors are developed for sulphate which can be used in the evaluation of estimated dose levels.

The issues associated with elevated sulphate concentrations is mainly related to the aesthetic quality of the contaminated water and health effects from sulphate exposure is observed only at high concentrations and in sensitive individuals.

Sulphate can therefore only be evaluated qualitatively by comparison with drinking water guidelines. Concentrations of sulphate associated with gold tailings or specifically modelled for the Kareerand TSF were shown to exceed quality criteria. Based on these exceedances it can be concluded that health effects are possible should drinking water resources be affected by seepage from the Kareerand or Expansion project TSFs.

### 5.5.3 Cancer Risk Assessment

The unit risk values for arsenic is used to evaluate the risk of cancer incidence associate with exposure to contaminated drinking water. The unitless cancer risk value is calculated by multiplication of the estimated concentrations of arsenic by a unit risk factor. A cancer risk in the order of one in a hundred thousand ( $1.0E-5$ ) is usually considered to be acceptable, while one in a million ( $1.0E-6$ ) is usually considered to be negligible.

The cancer risks calculated for the average and maximum concentrations of arsenic measured in tailings seepage are both below  $1.0E-8$ . In spite of the very conservative approach to the calculations, the estimated cancer risk values indicate risks are negligible, with values either well below one in a million. Dilution of the seepage in groundwater will further reduce the concentration of arsenic. The risk of cancer from the ingestion of water potentially contaminated with seepage from the Kareerand TSF or the Extension Project TSF is therefore considered negligible.

---

## 5.6 CONCLUSIONS AND RECOMMENDATIONS

---

### 5.6.1 Airborne Particulates

Based on the estimated increase in the personal risks associated with either short- or long-term exposure to airborne particulates from the Kareerand and Extension Project TSFs combined, the increase in risk of all health endpoints assessed are insignificant. Although the evaluation of short term exposure (highest 24 hour average) to concentrations of  $PM_{10}$  showed a measurable increase in the risk of both non-accidental and cardiovascular mortality, none of these estimated increases are significant.

Long-term exposure to  $PM_{2.5}$  was shown to lead to very low increase in personal risk of total non-accidental mortality and cardiopulmonary mortality, as compared to the baseline risk. It was reasoned



that this is due to the low concentration of smaller (<2.5µm) particles assumed by the air quality specialist for the dispersion model.

### 5.6.2 Hazardous Elements Associated with Airborne Particulates

Exposure to particle associated manganese and uranium was evaluated using a set of conservative assumptions with regard to the quantities that can enter the atmosphere. The estimated airborne concentrations were evaluated assuming long-term chronic exposure, but using short term (daily) maximum airborne particulate concentrations. The resulting hazard quotients indicate that the probability of non-cancer health effects occurring from inhalation exposure to any of the contaminants are low.

### 5.6.3 Contaminants in Water Resources

The potential for health effects associated with the contamination of groundwater or surface water resources from activities or sources related to the proposed Extension Project, could not be evaluated directly due to absence of information on the concentrations of these contaminants likely to be induced in local groundwater and surface water resources.

However, evaluation of baseline water quality data indicates that water resources in the area are not yet severely impacted and it is expected that the contribution from the proposed Extension Project will not significantly impact water quality, provided that proposed mitigation measures are applied. Cancer risk assessment performed on the estimated concentrations of arsenic indicated cancer risks to be negligible.

### 5.6.4 Recommendations

Interpretation of the results leads to the conclusion that the potential for health impacts relate mainly to the residential receptors located on the Vaal River in a south easterly direction from the Kareerand TSF. However, although not necessarily significant, the increase in personal risks associated with the proposed Extension Project TSF demonstrated a small potential increase over baseline risks and those relating to the Kareerand TSF.

In terms of airborne concentrations of particulates, the dust emission rates and particle size distribution assumed by the air quality specialist, had a critical influence on the calculated risks. It is consequently recommended that, as recommended by Airshed (2020), source and ambient air quality monitoring be implemented and that recommended dust abatement measures be applied.

The monitoring results can be compared, not only to the South African National Air Quality Standards, but also to the emission rates used by Airshed for the dispersion modelling. Any exceedances of these emission values can be regarded as an indication of a potential for health effects and measures should be implemented to reduce airborne pollutant emissions.

It has to be noted that the methodology used for the assessment of potential health effects, conservatively assumed that short term (daily) maximum concentrations are representative of long-

term average concentrations. The results from this assessment are therefore considered representative of the highest potential risk of health impacts likely posed to members of the public by any of the different operational phases associated with the proposed Extension Project.

The estimated potential for health risks from exposure to airborne particle associated contaminants was shown to be low for all contaminants and all potential receptor locations. All individuals or residential communities located further away from the Project site, will be subject to lower concentrations of the pollutants, and consequently also to lower risk of health effects.

In accordance with recommendations of the Geohydrological Specialist report, it is recommended that seepage and runoff from the tailings must be contained as far as possible through the implementation of the proposed groundwater interception system for the existing Kareerand TSF, concurrent side wall rehabilitation and the proposed Class C lining system for the extension. It is recommended that regular groundwater and surface water quality monitoring be established and maintained in the areas potentially affected by seepage and runoff from the TSFs. Any groundwater abstraction boreholes in use by members of the neighbouring communities should be closely monitored for deterioration of water quality. Once the trend of baseline water quality variation is understood, any observed increase in the concentrations of elements and ions, especially arsenic, nitrate or uranium, should be immediately investigated and the use of groundwater from the affected borehole must be suspended.



## 6 UNCERTAINTY ANALYSIS

---

### 6.1 ASSUMPTIONS AND UNCERTAINTY IN THE ASSESSMENT OF HEALTH RISKS

---

The health data on which the quantification of health effects depends, are subject to various uncertainties related to the quality and representativity of the health databases used as a basis. As indicated earlier, the health data available for the North West Province, and the Dr Kenneth Kaunda District Municipality where the Kareerand TSF and proposed Extension Project is situated, only provides statistics on mortality from different causes. For the purpose of this assessment it was assumed that the mortality rates, as available for the Dr Kenneth Kaunda District Municipality is representative of the incidence of effect specific mortalities in the communities affected by the proposed Extension Project.

Other factors contributing to uncertainty are the quality of the air pollution databases and the reliability of the statistical models used to assess relationships between air pollutant concentrations and health effects. The uncertainties related to the databases include the completeness of data, the impact of measurement error and the limitations of using fixed air monitors to represent the entire population in environmental exposure studies. Lastly, statistical models may be biased and may over- or underestimate the potential magnitude of the predicted mortality rates.

The validity of the projected associations between air pollutant concentrations and mortality reported in the literature, is only as good as the quality of the study that produced those relationships (Ostro, 1996). As a basis for this report, care was taken to select good quality studies and the validity of the conclusions for the populations in which they were conducted, should be high. Unfortunately, these reports never included South African or even African populations. Epidemiological studies conducted in South Africa would have been the ideal basis for an evaluation of health effects associated with the proposed Extension Project, but such studies are not available.

An important source of uncertainty is therefore the validity of applying relationships derived from non-African, mostly developed countries, to the South African, semi-developed country scenario. Since the general South African population is poorer than populations from developed countries, they can be expected to be less healthy, are likely to have poorer access to medical care (which might be of a lower standard) and therefore probably experience increased susceptibility to especially respiratory diseases, resulting in higher baseline morbidity and mortality rates.

In this regard, further uncertainty is introduced by the potential impact of high rates of infectious diseases such as HIV/AIDS and tuberculosis (TB), which may increase susceptibility to diseases, resulting in the potential underestimation of the morbidity impacts of air pollutants. On the other hand, high rates of HIV/AIDS and TB may inflate the mortality rates and may change the value of the risk factor applicable to South African populations. In other words, risk factors calculated for South African populations may be more conservative, due to possible high incidence of HIV/AIDS and TB in the communities.

---

## 6.2 VULNERABILITY IN THE SOUTH AFRICAN POPULATION

---

Vulnerability of a community considers the resilience of a community to recover from the impact of natural or anthropogenic hazards. Understanding of vulnerability at community or population helps to identify and protect sensitive sub-population groups from the effects of air pollution. Risks therefore should be considered within the boundaries of the susceptibility of communities to the risks.

In South Africa, the CSIR has conducted some research into community vulnerability, with the aim of developing vulnerability factors specific to the South African population. Juanette John and her colleagues (John, et al., 2008) has identified examples of aspects that are especially important in the South African context, resulting in people being less resilient to and therefore less able to cope with adverse effects of environmental exposures, including air pollution. These are:

- Presence of existing diseases
- Gender distribution of the household
- Presence of certain nutrients in the diet
- Source of household energy (fire or electricity).

John et al. (2008) concluded that the integration of vulnerability assessments and the traditional risk assessment process in South Africa face several challenges. Vulnerability factors specific to the South African situation are, as yet, not available for integration into the health risk assessment process.

---

## 6.3 LIMITATIONS OF HEALTH DATA AND POPULATION STATISTICS

---

The South African health- and population data presented in this report are not as detailed as ideally required to perform the possible health effect estimations for which risk factors are available. In this study only mortality as effect was considered as there are no condition specific data available for rates of hospital admissions in either the Dr Kenneth Kaunda District Municipality or North West Province. The assessment could have included estimates for effects on hospitalisation rates but data would have to be adapted, which means that the assessment could not have been performed with a high degree of confidence.

The provincial data used for assessment of the effect on mortality rates is approximately two years old, but is considered to be an adequate representation of cause specific mortality in the municipal area where the proposed Extension Project is located. The quality of the mortality data used therefore does not detract from the level of confidence in the results obtained from the health risk assessment.

Due to limitations in the available population statistics specific to the study area, the risk factors could not be used to predict potential numbers of deaths (absolute risks). Risk was therefore presented as relative risks, which may be difficult to interpret by the community and is therefore not the method of choice. However, the results are nevertheless useful to indicate areas where modelled concentrations of pollutants may result in proportionally high effects.

---

## 6.4 UNCERTAINTY IN ASSUMPTIONS

---

The concentrations of potentially hazardous elements (arsenic, manganese, lead and uranium) in the tailings, obtained from an earlier study by the University of Johannesburg (Annegarn, et al., 2010) is assumed to be representative of the materials that will generate airborne particulates from the proposed Extension Project.

Analytical data from samples collected in the environment typically varies over time and space, even for samples collected from one source area. The concentrations of potentially hazardous elements used in the evaluation of health impacts from these elements can therefore be expected to vary from the different materials (e.g. tailings from different source TSFs) associated with the proposed Extension Project. Although the concentrations of these elements may be higher in certain materials, the risks calculated are low enough to allow for some increase in concentration without the risk of significant health impacts occurring.

The lack of background air monitoring data for the area, and particularly the receptor areas surrounding the proposed Extension Project area, necessitated the assumption that the modelled concentrations represent the total pollutant concentrations in the area as a result of the proposed Extension Project. This assumption has the potential for misinterpretation of actual risks. However, in this case the assumption is regarded as valid, because the health data used in the evaluation of effects relating to particulate exposures are relatively recent and effects associated with the concentrations of airborne particulates from the existing Kareerand TSF are likely to be accounted for in the natural incidence derived from the available statistics.

Another source of uncertainty in the assessment is the quality and accuracy of the predicted pollutant and contaminant concentrations in environmental media that were used in the calculation of health risks. EnviroSim cannot verify input values and results obtained from groundwater and atmospheric dispersion models and therefore assume that the results, as presented by specialists are correct and a true representation of exposure.

To that effect, for the purpose of this assessment, it was assumed that the Geohydrological and associated Geochemistry Specialist Investigations are correct in accepting that the tailings deposited onto the Kareerand and Extension Project TSFs will be the only potential source of contamination to groundwater and surface water resources in the area, whether it be through seepage or from runoff.

With regard to air quality, it is assumed that the potential receptor locations selected by Airshed represent the highest exposed individuals and is a true representation of the exposure likely in each of the most exposed areas.

## 7 IMPACT ASSESSMENT

---

### 7.1 IMPACT IDENTIFICATION

---

The HHRIA presented here is one of many specialist components to a broader Environmental Impact Assessment process undertaken by GCS on behalf of MWS. The EIA process requires the assessment of all potential impacts (negative or positive) associated with the proposed Extension Project.

The health risks posed to members of the public by the activities planned as part of the proposed Extension Project, was evaluated using a source-pathway-receptor analysis approach. Information from specialist study reports were incorporated with toxicology data and population statistics to quantify the human health risks associated with the proposed Extension Project.

Information presented indicate that a complete source-pathway-receptor linkage exists for the atmospheric exposure pathway. Information on the aquatic environment, both surface- and groundwater, indicated that complete source-pathway-receptor linkage for this pathway may be possible, if proposed mitigative measures are not implemented. The aquatic pathway was therefore included in the further assessment. The potential for impacts relating to both the atmospheric and aquatic pathways was evaluated for the operational life of the proposed Extension Project. Impacts relating to construction and post-closure phases of the project are also addressed, albeit only qualitatively, as effluents and emissions associated with these phases specifically were not quantified through either the atmospheric dispersion modelling (Airshed, 2020) or contaminant transport modelling (GCS, 2020).

The impacts associated with the proposed Extension Project that are under evaluation for this study are defined as follows:

- |         |   |
|---------|---|
| HHRIA01 | Impact to human health associated with inhalation exposure to airborne particulates (PM <sub>2.5</sub> and PM <sub>10</sub> ) emitted from the surface of the Extension Project TSF.              |
| HHRIA02 | Non-cancer (systemic) health effects in humans as a result of inhalation exposure to manganese and uranium present in particulate matter emanating from the surface of the Extension Project TSF, |
| HHRIA03 | Risk of systemic health effects and cancer in humans as a result of ingestion of water contaminated through seepage from the Extension Project TSF.   |

The potential for occurrence of the impacts are evaluated based on the risks quantified in Section 5 of this report. For the purpose of this assessment, the nature of the impacts is rated as **negative** as any detrimental health effects associated with exposure to airborne pollutants and contaminated water is an anomalous occurrence that is entirely due to the proposed activity.

---

## 7.2 IMPACT ASSESSMENT METHODOLOGY

---

In accordance with the requirements of the EIA process, the potential impacts to human health, identified as part of the HHRIA, must be evaluated to determine the significance of each impact. This significance is as a function of the likelihood and consequence of the impact, which is determined according to the following variables (evaluation components), as provided by GCS:

- Duration (time scale),
- Scale (physical and spatial size of the impact)
- Magnitude (severity)
- Frequency of activity
- Frequency of impact
- Legal issues, and
- Detection

The evaluation proceeds by ranking identified impacts in terms of each evaluation component, according to a number scale. Using the assigned numeric rankings, the consequence and likelihood of each identified impact is determined by adding numbers for the relevant evaluation components.

The significance or 'risk' of each impact is then determined as the product of the consequence and likelihood and is interpreted as follows:

- High Risk- Rating 170-600.
- Moderate Risk – Rating 56-169.
- Low Risk – Rating 1-55.

---

## 7.3 CONSTRUCTION PHASE

---

Establishment of the Expansion Project TSF will involve the construction of new infrastructure to facilitate the deposition of tailings during the operational phase, as well as water management facilities to contain seepage and process water effluents.

During construction, airborne pollutants are expected to be generated from a variety of sources (e.g. earth-works, materials loading and off-loading, vehicle movement and vehicle exhaust emissions) associated with the construction activities. Although the contribution of these activities to the ambient concentrations of airborne pollutants is uncertain, it is expected that the duration of the activities will be limited compared to the duration of the operational phase. According to the Air Quality Specialist Report (Airshed, 2020) the potential impact on ambient air quality from the construction phase is expected to be low. Consequently, the potential of impact to health from the construction phase is expected to be low compared to that from the operational phase.

Similarly, during the short construction phase no direct impacts to water quality is expected. Health impacts associated with the ingestion or use of contaminated water is therefore accepted as negligible.

---

## 7.4 EVALUATION AND RANKING OF OPERATIONAL PHASE IMPACTS

---

### 7.4.1 HHRIA01-Human health impact from inhalation exposure to particulates.

The **Severity** of the impact is used to establish whether the impact is destructive or not and is an indication of whether the potential impact will result in a measurable change in the affected environment. The risks calculated as part of this assessment indicate that for exposure to the airborne particulates, health effects may occur (are probable) but the probability is very low. The Severity of the impact relating to exposure to airborne particulates is therefore ranked **Potentially harmful (2)** as there is a potential for harm should particulate concentrations increase. Even with mitigation, the ranking of this impact remains the same.

The **Scale** of a potential impact considers whether the impact is expected to be restricted to the local environment or whether the impact may extend further afield. Based on the conditions of exposure considered for the assessment, and the dispersion modelling results presented, unmitigated airborne particulate emissions are expected to go beyond the physical project boundary. The scale of the unmitigated potential impact is ranked as **Regional (4)**. However, with mitigation applied the dispersion of airborne particulates can be expected to be significantly decreased. With mitigation the scale of the impact is expected to be reduced to the **Site Only (2)**.

The **Duration** of the potential impact is expected to be for the **Life of the activity (4)**. Although the concentration and distance of airborne particulate dispersion is expected to be reduced by mitigation measures, any exposure that may occur will occur as long as the tailings remain in place.

**Likelihood** of the impact is the synthesis of the frequency of the activity, frequency of the impact as well as how quickly the impact can be observed. The health risks associated with exposure to airborne particulates was evaluated for daily maximum modelled concentrations. As these daily maxima are expected to occur for only a few days in a year, frequency of the activity and impact is assumed to be no more than once a month (both ranked **Monthly, 3**). Although it is expected that this frequency may be reduced as a result of mitigation, it is not as The health effects associated with exposure to increased concentrations of airborne particulates can be observed over both the short and long term, depending on the specific health effects observed. For the assessment, mortality rates were evaluated which would require some effort to obtain and relate back to the increased exposure (ranked **3**).

Another aspect considered is whether the activity is governed by legislation. In the case of impacts to health, there are no regulation or guidelines specifically governing this impact (ranked **1**). The likelihood of impact relating to exposure to airborne particulates is ranked at **10**.

Table 7.1 presents a summary of the of the rankings assigned to the various evaluation components used in the assessment of the impact.



Table 7.1: Assessment of impact HHRIA01.

Impact Mode	Receptor	Evaluation of Impact Risk						Risk
		Nature	Severity	Scale	Duration	Consequence	Likelihood	
Unmitigated	Land users who live closest to operational areas	Negative	2	4	4	10	10	100 Moderate
Mitigated		Negative	2	2	4	8	10	80 Moderate

**Mitigation measures:**

- Mitigation measures (Airshed, 2020):
- Disturbed area reduction – planned through deposition on one area at a time.
- Disturbance frequency reduction – planned through continuous revegetation and rehabilitation.
- Dust spillage prevention and/or removal.
- Disturbed area wind exposure reduction, e.g. vegetation on side slopes, wind fences/nets at source areas.

The risk of impact to human health from inhalation exposure to airborne particulates dispersed from the Extension Project TSF is therefore ranked as **Moderate** for both unmitigated and mitigated conditions.

7.4.2 HHRIA02- Non-cancer (systemic) health effects from inhalation exposure to particle associated contaminants

Although the levels of exposure to particle associated contaminants will be similar to that of criteria pollutants the calculated risk of health effects is much lower. The **Severity** of the impact is therefore ranked **Insignificant (1)** and remains insignificant also for mitigated conditions.

As is the case for the particulates the **Spatial Extent** of the impact relates to the dispersion of the dust and is ranked **Regional (4)** for unmitigated and **Site Only (2)** for mitigated conditions. **Duration** of the potential impact associated with exposure to particle associated contaminants is ranked as **Life of the activity (4)**.

The frequency of the activity, frequency and observability of the impact as well as regulatory issues remain the same for this impact meaning that the **Likelihood** of the impact is ranked at 10. Table 7.2 presents a summary of the of the rankings assigned to the various evaluation components used in the assessment of the impact.

The risk of impact to human health from inhalation exposure to particle associated contaminants dispersed from the Extension Project TSF is therefore ranked as **Moderate** for both unmitigated and mitigated conditions.

**Table 7.2: Assessment of impact HHRIA02.**

Impact Mode	Receptor	Evaluation of Impact Risk						Risk
		Nature	Severity	Scale	Duration	Consequence	Likelihood	
Unmitigated	Land users who live closest to operational areas	Negative	1	4	4	9	10	<b>90 Moderate</b>
Mitigated		Negative	1	2	4	7	10	<b>70 Moderate</b>

**Mitigation measures** (Airshed, 2020):

- Disturbed area reduction – planned through deposition on one area at a time.
- Disturbance frequency reduction – planned through continuous revegetation and rehabilitation.
- Dust spillage prevention and/or removal.
- Disturbed area wind exposure reduction, e.g. vegetation on side slopes, wind fences/nets at source areas.

**7.4.3 HHRIA03- Risk of systemic health effects and cancer in humans as a result of ingestion of contaminated water.**

The risks calculated as part of this assessment indicate that for exposure to drinking water contaminated with arsenic, nitrate or uranium both systemic health and cancer risks are negligible, provided that mitigation is applied. However, should seepage from the Extension Project TSF reach groundwater resources, contaminant transport modelling indicates that dissolved contaminants can reach boreholes from which groundwater is utilised for domestic purposes. **Severity** of the impact is therefore ranked as **Potentially harmful (2)** for unmitigated conditions, while the rating is **Insignificant (1)** if mitigation is applied.

**Spatial Extent** of the impact relates to the dispersion of the contaminated groundwater from the Extension Project TSF specifically. This is expected to be limited to the footprint of the Extension TSF, provided that recommended mitigation is applied, the scale of the potential impact is therefore ranked as **Whole Site (2)** while a case where mitigation is not applied is ranked as **Local (3)**. **Duration** of the

potential impact is however ranked as **Permanent (5)**, as should it occur the effects will last beyond the duration of the project. If mitigation is applied the duration ranking is lowered to **1**.

Although the observability of the impact as well as regulatory issues remain the same for this impact, the frequency of the activity and frequency of impact are both lowered to **1** for mitigated conditions. The **Likelihood** of the impact is therefore ranked at **10** for unmitigated conditions and **6** under mitigated conditions.

**Table 7.3: Assessment of impact HHRIA03.**

Impact Mode	Receptor	Evaluation of Impact Risk						Risk
		Nature	Severity	Scale	Duration	Consequence	Likelihood	
Unmitigated	Land users who live closest to operational areas	Negative	2	3	5	10	10	<b>100 Moderate</b>
Mitigated		Negative	1	2	1	4	6	<b>24 Low</b>

**Mitigation measures:**

- Class C Barrier containment system to limit seepage into the aquifer
- Appropriate under-drain systems
- Larger return water dam system serving both the Kareerand and Extension Project TSFs.
- Interception boreholes for active sulphate plume management

The risk of impact to human health from ingestion of contaminated water dispersed from the Extension Project TSF is therefore ranked as **Moderate** for unmitigated conditions and **Low** for mitigated conditions.

## 7.5 DECOMMISSIONING AND CLOSURE PHASE

The Air Quality Specialist Report (Airshed, 2020) indicates that, although dispersion modelling for dust emissions associated with decommissioning and closure phases could not be undertaken, air quality impacts from these phases are likely insignificant.

Similar to the construction phase, the decommissioning and closure phases are expected to involve various activities that will generate airborne pollutants. However the limited duration of the decommissioning activities would likely reduce the significance of the potential impacts relative to that

of the operational phase. Consequently, the potential of impact to health from the decommissioning and closure phases is expected to be low compared to that from the operational phase.

The decommissioning and closure activities are not expected to make any directly contribution to contaminant concentration groundwater and surface water resources. Decommissioning of the Kareerand and Extension Project TSFs will likely involve cover and vegetation of the TSF surface which will limit the quantity of water infiltrating into the TSF and reduce the quantity of water seeping out of the tailings over time. As indicated in the Hydrogeological Impact Assessment report (GCS, 2020), the quality of seepage from the tailings is expected to deteriorate post closure. However, as the mitigation measures proposed for capturing and containing the contaminated seepage is expected to prevent the contamination of off-site resources, health impacts associated with the ingestion or use of contaminated water is therefore accepted to remain negligible post closure.

---

## 7.6 CUMULATIVE IMPACTS

---

The risk of impact from exposure to airborne and aquatic pathway contaminants were shown to be low, especially if proposed mitigation is applied. The contaminants further do not target the same organs and although simultaneous exposure to airborne and waterborne contaminants may result in cumulative health effects, the health effects are not synergistic.

Cumulative impacts from air pollutants originating from other sources is discussed in the Air Quality Specialist Report (Airshed, 2020). It indicates that It is difficult to predict the contribution of sources such as residences, farming, mining and wilderness to existing air quality, but that it is unlikely these sources will result in a significant increase in pollutant concentrations, at least in the long-term.

It is therefore concluded that the contribution made by the proposed Extension Project TSF to baseline concentrations of air pollutants or waterborne contaminants will most likely not lead to risks of higher significance than the results presented in Section 7.4.



## 8 REFERENCES

- Airshed, 2020. *Air Quality Specialist Report for Mine Waste Solutions Kareerand Extension Project, 18AGA01*, Midrand: Airshed Planning Professionals (Pty) Ltd.
- Annegarn, H. J., OJelede, M. E., Umba-Ndolo, G. & Kneen, M. A., 2010. *Anglogold Ashanti dust monitoring project Volume II – Final Report*, Johannesburg: Department of Geography, Environmental Management & Energy Studies,.
- AquiSim, 2020. *Kareerand TSF Expansion Project: Radiological Public Impact Assessment, ASC 1025G-1*, Centurion: AquisSim Consulting (Pty) Ltd.
- ATSDR, 2007. *Toxicological Profile for Arsenic*, Atlanta, GA: US Department of Health and Human Services, Public Health Service, Agency For Toxic Substances and Disease registry.
- ATSDR, 2008. *Toxicological Profile for Aluminium*, Washington DC: US Department of Health and Human Services, Public Health Service, Agency For Toxic Substances and Disease Registry.
- ATSDR, 2011. *Nitrates and Nitrites, CAS#84145-82-4, 14797-65-0*, Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine.
- ATSDR, 2011. *Toxicological profile for Uranium*, Atlanta, GA: Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services.
- ATSDR, 2012. *Toxicological Profile for Manganese*, Atlanta, GA: Public Health Service, US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry.
- ATSDR, 2019. *Toxicological Profile for Lead*, Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology and Human Health Sciences, Environmental Toxicology Branch..
- Baars, A. J. et al., 2001. *Re-evaluation of Human-Toxicological Maximum Permissible Risk Levels. RIVM Report No. 711701025*, Bilthoven, Netherlands: National Institute of Public Health and the Environment.
- COMEAP, 2006. *Cardiovascular Disease and Air Pollution*, London: UK Department of Health Committee on the Medical Effects of Air Pollutants.
- COMEAP, 2009. *Long term Exposure to Air Pollution: Effect on Mortality*, London: UK Department of Health Committee on the medical Effects of Air Pollution.
- Dockery, D. W. et al., 1993. An association between air pollution and mortality in six US cities. *N. Eng. J. Med.*, Issue 329, pp. 1753-1759.
- DWAF, 1996. *South African Water Quality Guidelines, Volume 1: Domestic Use*, Pretoria: Department of Water Affairs and Forestry.

EFSA, 2008. Scientific Opinion of the Panel on Food Additives, Flavourings, Processing Aids and Food Contact Materials on a request from European commission on Safety of aluminium from dietary intake.. *The European Food Safety Authority Journal*, Volume 754, pp. 1-34.

GCS, 2020. *Hydrogeological Assessment for the Kareerand TSF & Expansion Project, 17-0109.*, Durban, South Africa: GCS (Pty) Ltd.

Hall, S. K., Chakraborty, J. & Rich, R. J., 1997. *Chemical Exposure and Toxic Responses*, Boca Raton, Florida, USA: CRC Press Inc..

IPCS, 1999. *Principles for the Assessment of Risks to Human Health from Exposure to Chemicals, Environmental Health Criteria 210, A cooperative Agreement between UNEP, ILO, FAO, WHO, UNIDO, Unitar and OECD*, s.l.: International Programme on Chemical Safety.

IRIS, 2002. *Nitrate CASRN —14797-55-8, Integrated Risk Information System*, Washington, DC: United States Environmental Protection Agency.

Jerrett, M. et al., 2005. Spatial analysis of air pollution and mortality in Los. *Epidemiology*, Issue 16, pp. 727-736.

John, J., Matoane, M., Oosthuizen, R. & Wright, C., 2008. *Vulnerability to air pollution: To intervene or not to intervene. Science real and relevant*. Pretoria, 2nd CSIR Biennial Conference, CSIR International Convention Centre Pretoria, 17&18 November 2008, pp 6.

Knight Piésold, 2019. *FEASIBILITY STUDY FOR KAREERAND TSF EXTENSION PROJECT, 301-00204/13 Rev 1*, Johannesburg: Knight Piésold (Pty) Ltd.

NRC, 1983. *Narional Research Council, Risk Assessment in the Federal Government: Managing the Process, Committee on the Institutional Means for the Assessment of Risks to Public Health*, Washington, DC: National Academy Press.

Ostro, B., 1996. *A Methodology for Estimating Air Pollution Health Effects.*, Geneva: World Health Organisation.

Pope, C. A. et al., 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA*, Issue 287, pp. 1132-1141.

Pope, C. A. et al., 1995. Particulate air pollution as a predictor of mortality in a prospective study of US adults.. *Am. J. Respir. Crit. Care Med.*, Issue 151, pp. 669-674.

StatsSA, 2016. *Statistics By Place,nStatistics South Africa*. [Online] Available at: [http://www.statssa.gov.za/?page\\_id=964](http://www.statssa.gov.za/?page_id=964) [Accessed 4 May 2020].

StatsSA, 2020. *Mortality and Causes of Death in South Africa 2017: Findings from death notifications, Statistical Release P0309.3*, Pretoria, South Africa: Statistics South Africa.

US EPA, 2002. *Code of Federal Regulations, Title 40 - National Ambient Air Quality Standards for Particulate Matter; Final Rule*, Washington, DC: United States Environmental Protection Agency.

US EPA, 2004. *Fourth External Review Draft of Air Quality Criteria for Particulate Matter, Volume II. EPA/600/P-99/002aD*, Research Triangle Park, NC: U.S. Environmental Protection Agency.

US EPA, 2006. *Drinking Water Standards and Health Advisories*. [Online] Available at: <http://www.epa.gov/waterscience/criteria/drinking/dwstandards.html#inorganics> [Accessed 4 January 2013].

US EPA, 2007. *Nitrates and Nitrites*, Washington, DC: United States Environmental Protection Agency.

US EPA, 2010. *Toxicological Review of Inorganic Arsenic - In support of Summary Information on the Integrated Risk Information System (IRIS), EPA/635/R-10/001*, Washington DC: United States Environmental Protection Agency.

US EPA, 2011. *Exposure Factors Handbook, 2011 Edition, EPA/600/R-09/052F*, Washington, DC: US Environmental Protection Agency, Office of Research and Development, National Centre for Environmental Assessment.

WHO, 2005. *WHO Air Quality Guidelines Global Update. Meeting Report*, Geneva: World Health Organisation.

WHO, 2006. *Guidelines for Drinking Water Quality. First addendum to 3rd Edition, Volume 1*, Geneva: World Health Organization.

