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HUMAN HEALTH RISK ASSESSMENT FOR COMMUNITIES EXPOSED TO DUST FROM THE PROPOSED SALDANHA LOGISTICS HUB

PREPARED FOR:

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EXECUTIVE SUMMARY

WSP Group Africa (Pty) Ltd (WSP) has been appointed by ArcelorMittal South Africa (Pty) Ltd (AMSA) to undertake a Basic Assessment (BA) process to meet the requirements under the National Environmental Management Act (Act 107 of 1998) (NEMA), for the proposed new Logistics Hub project in Saldanha Western Cape.

The nearest communities include Bluewater Bay, approximately 4.8 km west-southwest of Saldanha Steel, Langebaan approximately 6.8 km south-southeast, and Vredenburg, approximately 6.1 km north of Saldanha Steel.

The proposed Logistics Hub will receive bulk material ore via road and rail from as far as the Northern Cape, and the operations will entail the receiving, distributing, and handling of various bulk commodities for local and export purposes.

Members of the public expressed concern of possible adverse health effects from exposure to dust emitted from the proposed Logistics Hub, where 5,000,000 tpa bulk material ore may be received, stored and handled before exporting via the port of Saldanha. It was therefore decided to perform a Human Health Risk Assessment (HHRA), to determine the potential for the public to develop adverse health effects from exposure to the dust created at the hub.

Since different types of ore will be handled, the HHRA did not only consider Particulate Matter at or below 2.5 micrometers in diameter ($PM_{2.5}$) as pollutant of concern, but also manganese, lead, and silica.

The HHRA was undertaken according to the US-Environmental Protection Agency's (US-EPA) four step approach (outlined in Section 4), of Hazard identification (can the pollutant cause adverse health effects?), Exposure assessment (what are the concentrations the public may be exposed to?), Dose-response/Concentration-response assessment (what are the concentrations where adverse effects would be unlikely?) and Risk characterisation (considering the previous steps, is there a potential for adverse effects?).

Particulate Matter is statistically significant associated with heart disease, respiratory diseases, and lung cancer in humans. **Manganese** may cause impairment of neurobehavioural function, as was found in occupational studies at relatively high (above 30 $\mu g/m^3$) concentrations, as well as lung irritation. The International Agency for Research on Cancer (IARC) has not classified manganese as a confirmed human carcinogen. **Lead** may cause neurological effects (neuropsychological in adults and affecting the intellect of children). The IARC has not classified lead as a confirmed human carcinogen. **Silica** may cause silicosis (lung fibrosis). However, adverse health effects were not reported from inhalation of large particles or at low levels. The IARC classified crystalline silica inhaled as quartz from occupational sources, as a known human carcinogen.

In this HHRA, the concentrations of pollutants used, were modelled, using CALPUFF, an internationally recognised dispersion model. CALPUFF a Tier 3 model, is recommended for areas of complex meteorological conditions, such as coastal environments. Meteorological conditions over a three-year period (2018 to 2020) were used as input data to model the maximum concentration (as a worst-case) at 24 receptor points and at the Northern Fence Line (depicted in Figure 1), under each of three scenarios. For this exercise it was assumed that all mitigation measures were in place. Maximum short-term (24-h) and long-term (annual) exposure concentrations were modelled under each scenario to determine the potential for acute and chronic health effects. Maximum concentrations are the highest concentrations that can be present at a receptor point, given worst-case conditions, including emissions and meteorological (weather/atmospheric) conditions, which, in real life, may never happen.

These scenarios were:

Scenario 1 – Saldanha Steel operations only

Scenario 2 – Logistics Hub operations only

Scenario 3 – Cumulative (both Saldanha Steel and Logistics Hub) operations

The highest maximum concentrations modelled under each scenario, were at Receptor Point 2 (the main road) and the Northern Fence Line. The second highest concentrations were mostly found at Receptor Point 3 (Southern Fence Line). Concentrations modelled for the residential areas, were well below guidelines and standards under all three scenarios.

The benchmarks (“safe” concentrations) used to quantify the potential for developing adverse health effects under each scenario, were the South African standards for $PM_{2.5,}$ and lead, as these standards may be enforced by law. For comparison, the World Health Organization (WHO) guidelines were used (Table 4). Where South Africa did not have standards (such as for manganese and silica), international standards or guidelines (including those of the WHO) were used (Section 5.3).

In the risk characterisation step of the assessment, risks were quantified by calculating a unitless Hazard Quotient (HQ) (Tables 5 to 7 and Section 5.4).

Under Scenario 1 (Table 5), there is a potential for acute and chronic health effects from $PM_{2.5}$ at the Northern Fence Line, if the South African standards are used, and a potential for acute and chronic effects at Receptor Point 2 (main road) and the Northern Fence Line when using the WHO guidelines. However, all HQs determined for residential areas were below 1, indicating that adverse health effects would be unlikely, even in sensitive individuals.

The HQs calculated under Scenario 2 (Table 6), indicate that it would be unlikely for any individual to develop adverse health effects from exposure to the modelled $PM_{2.5,}$ manganese or lead when using South African standards, WHO guidelines, IRIS guidelines (from the US-EPA) or the Centres for Disease Control (CDC) guideline.

Concentrations for silica were not modelled. However, as a worst-case scenario it was assumed that 100% of the modelled annual $PM_{2.5}$ concentrations under Scenario 2, consisted of crystalline silica. These modelled concentrations and a guideline from the California-EPA were used to quantify the potential for adverse health effects. All HQs calculated indicated that it would be unlikely for any individual to develop chronic health effects such as silicosis.

The HQs calculated under Scenario 3 (Table 7), were similar to the situation under Scenario 1. There is a potential for acute and chronic health effects from $PM_{2.5}$ at the Northern Fence Line, when the South African standards were used, and a potential for acute and chronic effects at Receptor Point 2 (main road) and the Northern Fence Line when using the WHO guidelines. However, all HQs calculated for residential areas, using the modelled data, showed it will be unlikely for any individual, even sensitive individuals, to develop adverse effects.

The risk of exposure to dust created during transport is qualitatively assessed in Appendix II, as no exposure concentrations are available.

In terms of impact significance criteria used in environmental impact assessments (considering magnitude/intensity, extent, reversibility, duration, and probability of occurrence), the Human Health Risk Assessment may be rated as below.

Scenario (onsite processes and operations)	**Rating and Significance before mitigation**	**Summary of mitigation measures**	**Rating and Significance after mitigation**
Scenario 1 – Steelmaking only	N2 – 48, Moderate	Implement existing Fugitive Dust Management Plan (FDMP) with onsite mitigation measures and controls.	N2 – 32, Medium
Scenario 2– Logistics Hub only	N2 – 34, Moderate	Tarring of the service/entrance road, chemical and water spraying of dust and roads, covered conveyors, truckloads covered with tarpaulin, controls and monitoring as per the updated FDMP.	N1 – 16, Low
Scenario 3 – Cumulative (Steelmaking and Logistics Hub operating)	N2 – 48, Moderate	Tarring of the service/entrance road, chemical and water spraying of dust and roads, covered conveyors, truckloads covered with tarpaulin, controls and monitoring as per the updated FDMP.	N2 – 32, Medium

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1. Background

WSP Group Africa (Pty) Ltd (WSP) has been appointed by ArcelorMittal South Africa (Pty) Ltd (AMSA) to undertake a Basic Assessment (BA) process to meet the requirements under the National Environmental Management Act (Act 107 of 1998) (NEMA), for the proposed new Logistics Hub project and associated reconfiguration of existing infrastructure at AMSA Saldanha Steel Works facility, Saldanha, Western Cape. Saldanha Steel is situated in the Western Cape, approximately 120 km north-northwest of Cape Town, along the West Coast of South Africa, 2 km off the coast of Saldanha Bay.

The nearest communities include Bluewater Bay, approximately 4.8 km west-southwest of Saldanha Steel, Langebaan approximately 6.8 km south-southeast, and Vredenburg, approximately 6.1 km north of Saldanha Steel.

The proposed Logistics Hub will receive bulk material ore via road and rail from as far as the Northern Cape, and the operations will entail the receiving, distributing, and handling of various bulk commodities for local and export purposes.

Members of the public expressed concern of possible adverse health effects from exposure to dust emitted from the proposed Saldanha Logistics Hub, where 5,000,000 tpa bulk material ore may be received, stored and handled before exporting via the port of Saldanha. The actual total quantities of the commodities stored in the warehouse at any one time may fluctuate, depending on the bulk commodity required for export, although importantly, the total quantity of cumulative material handled, when operations are underway, will not exceed the threshold stipulated of 5,000,000 tpa. A maximum tonnage of each of the separate proposed commodities that could be stored within the warehouse is provided below.

Commodity	**Maximum Annual Tonnage not to be exceeded**
Manganese Ore (Mn)	4 million tons
Phosphate concentrate (PO_4)	1.2 million tons
Garnet Sands	0.5 million tons
Zircon Sands ($ZrSiO_4$)	0.5 million tons
Lead concentrate (Pb)	0.25 million tons
Copper concentrate (Cu)	0.25 million tons
Zinc concentrate (Zn)	0.25 million tons
Total Maximum Bulk Commodities Handled	**5 million tons**

The existing facility is already permitted to handle 2,832,000 tpa iron ore for the purposes of steelmaking. However, the facility is currently not in operation, as the ironmaking operations ceased in 2020 due to challenges in the global steel market. This situation may change, which will then significantly increase the dust emitted at the facility, considering the extra 2,832,000 tpa iron ore that will be handled.

It was therefore decided to perform a Human Health Risk Assessment (HHRA), to determine the potential for the public to develop adverse health effects from exposure to the dust created at the Saldanha Logistics Hub.

Since different types of ore will be handled, the HHRA did not only consider Particulate Matter at or below 2.5 micrometers in diameter ($PM_{2.5}$) as pollutant of concern, but also manganese, lead, and silica.

2. Terms of Reference

Perform a Human Health Risk Assessment (HHRA) of communities that may be exposed to dust from the proposed Saldanha Logistics Hub, where 5,000,000 tpa bulk material ore will be received, stored and handled before being exported via the port of Saldanha.

3. Scope of Work for Human Health Risk Assessment

An assessment (Human Health Risk Assessment) of the potential risks to human health from exposure to dust emissions from the proposed Saldanha Logistics Hub, was conducted. The HHRA was undertaken according to the US-Environmental Protection Agency's (US-EPA) four step approach (US-EPA, 2023) of Hazard identification, Exposure assessment, Dose-response assessment and Risk characterisation, outlined in Section 4. Occupational health and safety risks.

A report was compiled on the findings.

4. Human Health Risk Assessment (HHRA)

4.1 Approach

In order to manage environmental health, it is important to link human health effects to environmental exposure. One of the linkage methods to use, is a HHRA, as a HHRA identifies the potential for detrimental health effects that could be caused by exposure to a hazard. The hazard may be chemical (gases, particulates, or solutions), physical (radiation, noise and vibration) or biological (bacteria, viruses and pollen). The hazard, exposure potential, population characteristics, magnitude (concentration), frequency (how often) and duration (how long) of exposure, determine risk.

As a HHRA uses existing toxicological and exposure data to predict the potential for health effects, it may be conducted in a much shorter period of time than other methods such as epidemiology studies, which typically lasts two or more years.

In this study, the potential for adverse health effects in the individuals residing in communities around the Logistics Hub, was assessed based on the US-EPA Human Health Risk Assessment Framework (US-EPA, 2023). This approach, also approved by the World Health Organization (WHO) (WHO, 2010/2021), comprises the following steps:

- Hazard identification
- Exposure assessment
- Dose-response assessment or toxicity assessment
- Risk characterisation or risk estimation

4.2 Methodology

4.2.1 Hazard identification.

Hazard identification is aimed at determining whether exposure to a particular substance may result in adverse human health effects. The focus in this first step is on aspects such as:

- Physico-chemical properties relevant to exposure
- Sources, routes and patterns of exposure
- Metabolic and pharmacokinetic properties (how the body absorbs, distributes and eliminates compounds and the effects it may have on the body)
- Short-term *in vivo* (inside the body) and *in vitro* (in a test tube) tests
- Long-term animal studies
- Human exposure studies
- Human epidemiology studies

To identify the abovementioned aspects for the pollutants of concern in this study, reliable databases were accessed, such as the US-EPA, the Centers for Disease Control and Prevention (CDC) in the US, and the World Health Organization (WHO).

4.2.2 Exposure assessment.

Exposure to pollutants takes place when the human body comes into contact with the pollutant. Exposure assessment involves, amongst others, the determination of concentrations of the hazard. Concentrations may be measured (using instrumentation) or may be modelled, using mathematical computer models. These models use parameters such as emissions, climate, topography, as well as fate and transport of pollutants, and deposition, as input data. The output data are then used to estimate the concentration to which populations are or may be exposed to in different media (air, water or soil), through different routes (inhalation, ingestion or dermal contact). Lately satellite observations, chemical transport models and land-use regression models are being used in addition to monitoring, to obtain fine temporal and spatial scales (WHO, 2021).

The duration (how long) of the exposure as well as the frequency (how often) are estimated according to geographic distribution and activity patterns of the populations. In addition to concentrations (that which the body may come into contact with), the dose received (that which

ends up inside the body) may also be calculated when a pollutant is ingested or absorbed through the skin. A dose is expressed as an oral or dermal Average Daily Dose (ADD) for non-carcinogens (a pollutant that does not cause cancer), or a Lifetime Average Daily Dose (LADD) for carcinogens (a pollutant that may cause cancer).

Exposure may further be influenced by behaviour of people, which may vary greatly among countries or regions according to culture, education and climate. When conducting an exposure assessment, time-activity patterns (the time people spend in different microenvironments, such as at home, in the office or in a vehicle), and their activities in those environments, should ideally be evaluated. Important patterns to consider include quantities of food or water consumed and time spent outdoors vs. indoors. Specific behaviour, for example personal hygiene and smoking habits, may also add to, or minimise exposure.

In South Africa, exposure for acute risks is based on short-term (hourly or 24-hourly) monitored or modelled data, and exposure for chronic risks is based on annual data.

4.2.3 Dose-response assessment or concentration-response assessment

Dose-response assessment is the estimation of the relationship between exposure or dose and the human body's response to that exposure or dose. As a HHRA makes use of existing data, the dose-response relationship (in the case of ingestion or dermal contact) or exposure-response relationship (in the case of inhalation) is ascertained from information supplied by:

- Human epidemiological studies
- Human exposure studies
- Animal exposure studies
- Short-term *in vivo* and *in vitro* tests

Although response estimates based on human data are preferable to derive a benchmark value (a "safe" concentration or dose), estimates from animal data are often used when appropriate human studies are limited or not available. In such a case, uncertainty factors are applied to get to a benchmark value. Benchmark values based on health effects are preferred to those incorporating economic or social factors.

Several agencies, such as the US-EPA, the WHO and the CDC in the US, have developed databases for benchmarks that may be used in a HHRA.

The benchmark values most commonly used are:

- **Reference dose (RfD) and reference concentration (RfC)**. These US-EPA benchmark values represent the pollutant levels where no adverse non-cancer health effects are likely to occur if ingested (RfD) or inhaled (RfC) over a specified time period. The Californian EPA's equivalent is known as the Reference Exposure Level (REL) and that of the CDC as the Minimum Risk Level (MRL).

  In the case of criteria pollutants (those pollutants that countries have standards for), the benchmark value is that specific country's standard in air, water or soil, provided the value was based on health and not economy or welfare**.**

- **The oral slope factor and inhalation unit risk values** are used to describe the cancer potency of ingested or inhaled pollutants, respectively. Slope factors generally rely on a linear multistage model, which conservatively assumes that there is no threshold, i.e. a carcinogen may cause cancer at any level of exposure and the likelihood of developing cancer increases as the exposure increases. It must be noted that some scientists are of the opinion that some chemicals have the potential to cause cancer only when a minimum threshold level of exposure has been exceeded.

4.2.4 Risk characterisation

Risk characterisation is the final step in the Human Health Risk Assessment, combining all the information obtained in the previous three steps of the risk assessment to describe whether a risk to health is predicted from exposure to the pollutant(s) of interest. This process may be qualitative or quantitative.

A qualitative risk characterisation is purely a descriptive assessment, whereas the product of a quantitative risk characterisation is a numeric estimate of the public health consequences of exposure to the pollutant. Two types of risk estimates are calculated in a **quantitative** health risk assessment:

The hazard quotient (HQ), which is the ratio of a single substance exposure level over a specified time period to a reference concentration or dose for that substance, derived from a similar exposure period. The HQ describes the potential for developing adverse effects (other than cancer) from exposure to a hazardous substance.

Risk characterisation in a quantitative health risk assessment may vary from a single exposure medium, single exposure pathway through to multi-media and multi-pathway exposure. A multi-pathway, multi-media health risk assessment refers to a health risk assessment in which risk of exposure to pollutants present in multiple environmental media (soil, water, food, air, plants) and all possible routes in which these pollutants may enter the human body (inhalation, ingestion, dermal) are evaluated. The environmental pollutants commonly assessed in a multi-media/multi-pathway health risk assessment, are metals, polycyclic aromatic hydrocarbons, chlorinated hydrocarbons and pesticides.

The **incremental cancer risk**, which is the probability of individuals developing cancer from exposure to a hazardous substance over and above the background cancer risk. For inhalation, the risk is a function of the Inhalation Concentration and the Inhalation Unit Risk and for ingestion a function of the Lifetime Average Daily Dose and the Slope Factor.

The Inhalation Unit Risk (risk for every one µg/m³ of the pollutant) is the unit-less upper bound estimate of the probability of tumour formation per unit concentration of chemical (Mitchell, 2004) and the Slope Factor is an upper bound, approximating a 95% confidence limit, of the increased cancer risk from a lifetime exposure to an agent expressed in units of proportion (of a population) affected per mg/kg/day.

4.2.5 Uncertainties and limitations

The actual risk associated with a hazard can only be assessed and measured once damage from exposure to that hazard or pollutant has occurred. A Human Health Risk Assessment (HHRA) is therefore a *predictive* process that can assess the likelihood of adverse health effects occurring as a result of exposure to a hazardous substance. The risks can thus only be estimations of what could occur, and as such have uncertainty associated with them.

Human Health Risk Assessments are generally quite cautiously done as they include many safety factors that are built into the process. The final risk estimate is therefore likely to overestimate the actual risk.

Uncertainty in health risk assessments may be classified into three types:

- Variable uncertainty
- Model uncertainty
- Decision-rule uncertainty

Variable uncertainty occurs when variables appearing in equations cannot be measured precisely or accurately, either due to equipment limitations or spatial or temporal variances in the quantities being measured. Steps in which variable uncertainty may occur include:

- The determination of pollutant emissions for modelling
- The determination of levels (concentrations) of the pollutants from monitoring and/or modelling
- The use of population demographics or statistics
- The determination of activity patterns and health status of individuals

Model uncertainty is associated with all models, and equations, used in all phases of the risk assessment, including:

- Animal models used as surrogates for testing human toxicity and carcinogenicity
- The dose-response models used in extrapolations in the determination of health benchmark values or ambient air quality standards
- The use of computer and other models to quantify exposure and risk

Decision-rule uncertainty is associated with the manner in which the risk assessor conducts the study. This may include:

- The selection of the compounds of potential concern (pollutants) to be included in the risk assessment
- The identification of the most significant exposure pathways applicable in the assessment

- The use of national and international ambient pollutant guidelines/standards as significant values with which health effects may be associated
- The decision as to which exposure pathways are most significant for the specific pollutant(s) assessed

These uncertainties were considered when the HHRA framework was applied in this study.

5. Results and discussion

5.1 Hazard identification

According to the WHO, air pollution is the single biggest environmental threat to human health (WHO, 2021). Since the nineties (1990s), global air pollution has not improved, because although air pollution may have improved in developed countries, in developing countries it mostly deteriorated (WHO, 2021).

Particulate matter (PM) concentrations are often used as a proxy for air pollution, as it is considered as the air pollutant causing the most adverse human health effects (EEA, 2024). PM is defined by the World Health Organization (WHO) (2021) as: “A mixture of solid and liquid particles in the air, that are small enough not to settle out on the earth’s surface under the influence of gravity, classified by aerodynamic diameter”.

Considering the bulk material ore that will be handled at the Logistics Hub, the pollutants of concern identified, were Particulate Matter (PM), Manganese (Mn), Lead (Pb) and Silica (SiO_2). The main route of exposure identified was inhalation. The health effects of these pollutants are briefly mentioned below but are discussed in more detail in Appendix I.

5.1.1 Particulate Matter (PM)

The WHO (2021) suggests that in all cases where concentrations of different sizes of PM are available, preference should be given to $PM_{2.5}$ (particulate matter with an aerodynamic diameter of ≤ 2.5 micrometre). $PM_{2.5}$ particulates are small enough to be inhaled deep into the lungs, whereas Total Suspended Particulates (TSP) include particles of various sizes, a proportion of which are not able to enter the human respiratory tract. Therefore, TSP is not a good indicator of health-related exposure. PM_{10} (particulate matter with an aerodynamic

diameter of ≤ 10 micrometre in diameter) is considered inhalable, but not respirable, as not all of the particulates may reach the air exchange region of the lung.

The WHO did a complete assessment of studies (published up to 2020, including some of 2021), on the short-term and long-term health effects of PM when they updated the air quality guidelines in 2021 (WHO, 2021).

In summary of the studies assessed by the WHO and others, it may be stated that $PM_{2.5}$ in air is statistically significant associated with adverse health effects, including heart disease, respiratory diseases, and lung cancer in humans (WHO, 2021). The chemical composition of the particulates is of course also important in terms of health effects, however, the WHO is of the opinion that there are insufficient data available to base guidelines on different components of PM (WHO, 2021). The International Agency for Research on Cancer (IARC) has classified PM in outdoor air as a confirmed human carcinogen.

5.1.2 Manganese (Mn)

The health effects associated with manganese are also discussed in Appendix I. In summary it can be said that manganese is naturally occurring in the earth's crust and is a trace element necessary for good health (co-factor in some enzymes). The absorption of manganese through inhalation depends on the size of the particles. Only about 3-5% of ingested manganese is absorbed. Exposure to relatively high levels (above 30 µg/m^3) of manganese may cause impairment of neurobehavioural function, as was found in occupational studies (WHO, 2000; IRIS, 1993). The International Agency for Research on Cancer (IARC) has not classified manganese as a confirmed human carcinogen.

5.1.3 Lead (Pb)

The health effects associated with lead exposure are discussed in Appendix I. The general public may be exposed to lead through ingestion of contaminated food and water and inhalation of contaminated air, for example when using lead in hobbies such as soldering or making stained glass objects. Children are mostly exposed to lead through ingestion from hand-mouth-contact with contaminated soil or ingesting flacking lead-based paint. About 60 to 80% of inhaled lead is absorbed, while about 50% of ingested lead is absorbed in children, but only 3 to 10% in adults (CDC, 2020).

Lead has several adverse health effects including effects on the kidneys, on the blood, it may increase blood pressure, neurological effects (neuropsychological in adults and affecting the intellect of children), and may reduce fertility (CDC, 2020). The International Agency for Research on Cancer (IARC) has not classified lead as a confirmed human carcinogen.

5.1.4 Silica

Exposure to silica cannot be avoided, as it may be present in air, soil, food and water as well as consumer products (CDC, 2019).

From the studies investigated, it is evident that adverse effects were associated with particulates in the respirable size range and at relatively high concentrations where workers were exposed for long periods of time. Adverse health effects were not reported from inhalation of large particles or at low levels, or from incidental exposure in the ambient environment (CDC, 2019).

The main health effect from inhalation, is silicosis, a progressive, irreversible, fibrotic lung disease (lung fibrosis). Connective tissue forms as part of normal healing processes in the body, but in lung fibrosis, excess connective tissue forms in the lungs. The International Agency for Research on Cancer (IARC) classified crystalline silica inhaled as quartz from occupational sources, as a known human carcinogen (IARC, 2021).

5.1.5 Summary of main health effects of pollutants of concern

Pollutant of concern	**Main associated health effects**	**Benchmark values**
$PM_{2.5}$	Respiratory diseases; ischemic heart disease; confirmed carcinogen	Refer to Section 5.3, Table 4.
Manganese	Impairment of neurobehavioural function; Lung irritation	Refer to Section 5.3, Table 6.
Lead	Neurological effects (neuropsychological in adults and affecting the intellect of children)	Refer to Section 5.3, Table 6.
Silica	Silicosis; confirmed human carcinogen	Refer to Section 5.3.

5.2 Exposure Assessment

In this HHRA, the concentrations of pollutants used, were modelled, as the activities at the proposed Logistics Hub have not started, and could therefore not be monitored. The model used by the air quality specialists, was CALPUFF, an internationally recognised dispersion model. CALPUFF a Tier 3 model, is recommended for areas of complex meteorological conditions, such as coastal environments (WSP, 2023). Meteorological conditions over a three-year period (2018 to 2020) were used as input data to model the maximum concentration at 24 receptor points and the Northern Fence Line, under each of three scenarios (WSP, 2023). Maximum concentrations are the highest concentrations that can be present at a receptor point given worst-case condition, including meteorological (weather/atmospheric) conditions, which, in real life, may never happen.

Concentrations of pollutants of concern were modelled under the following three scenarios:

Scenario 1 – Saldanha Steel operations only

Scenario 2 – Logistics Hub operations only

Scenario 3 – Cumulative (both Saldanha Steel and Logistics Hub) operations

Short-term (24-h) and long-term (annual) exposure concentrations were modelled under each scenario to determine the potential for acute and chronic health effects.

5.2.1 Communities exposed

The 24 receptor points for which concentrations of pollutants were determined, are depicted in Figure 1.

Figure 1. Receptor points around the Logistics Hub for which concentrations of pollutants were modelled.

5.2.2 Magnitude (concentrations) of exposure

When making assumptions in this study, an approach of maximum protection of the communities of concern were followed.

For each scenario, maximum concentrations were modelled at each of 24 receptor points and the Northern Fence Line, to resemble a worst-case. For this exercise it was assumed that all mitigation measures included in the Fugitive Dust Management Plan, including tarring of the service/entrance road and spraying of dust, were in place.

Modelled concentrations of $PM_{2.5}$ for each scenario, are as in Tables 1, 2 and 3. Modelled concentrations of manganese and lead are as in Table 2, as these pollutants will be emitted under Scenario 2 (operations at the Logistics Hub) only.

Table 1. Maximum 24-h and annual concentrations of $PM_{2.5}$ modelled for Scenario 1 (Steelmaking operation).

Receptor point	Max 24-h$PM_{2.5}$µg/m^3	Max annual$PM_{2.5}$µg/m^3
1	1.42*	0.10*
2	31.11**	8.25**
3	14.36	1.51
4	5.69	0.51
5	2.31	0.22
6	2.42	0.22
7	2.61	0.23
8	3.49	0.29
9	1.62	0.15
10	1.80	0.12
11	1.60	0.11
12	3.03	0.23
13	2.57	0.22
14	1.53	0.12
15	1.53	0.11
16	1.46	0.11
17	3.04	0.29
18	3.14	0.50
19	5.11	0.81
20	5.92	0.86
21	3.35	0.45
22	2.62	0.38
23	2.49	0.38
24	3.84	0.45
Northern Fence Line	66.91**	21.46**

***Lowest concentration modelled **Highest concentration modelled**

From Table 1 it is evident that Under Scenario 1, all maximum modelled 24-h concentrations and all modelled annual concentrations of $PM_{2.5}$ for the 24 receptor points, were below the South African ambient air quality standards of 40 µg/m^3 for the 24-h concentration and 20 µg/m^3 for the annual concentration. However, the maximum concentration modelled for Receptor Point 2 (main road) is already 78% of the 24-h $PM_{2.5}$ standard, just from Saldanha Steelmaking as a single source.

Receptor Point 1 recorded the lowest modelled concentrations and Receptor Point 2 (main road) the highest concentrations. The maximum concentrations modelled for the Northern Fence Line under Scenario 1, exceeded the South African 24-h and annual standards.

Table 2. Maximum 24-h and annual concentrations of $PM_{2.5}$, Mn and Pb modelled for Scenario 2 (Logistics Hub).

Receptor point	Max 24-h $PM_{2.5}$ µg/m³	Max annual $PM_{2.5}$ g/m³	Max Annual Mn µg/m³	Max Annual Pb µg/m³
1	0.03	0.002*	0.000005*	0.00002*
2	0.42	0.10**	0.0005**	0.001**
3	0.71**	0.08	0.0002	0.0009
4	0.29	0.03	0.00006	0.0004
5	0.04	0.004	0.00001	0.00005
6	0.04	0.004	0.00001	0.00005
7	0.04	0.004	0.00001	0.00005
8	0.05	0.005	0.00002	0.00006
9	0.03	0.003	0.000008	0.00003
10	0.03	0.002*	0.000006	0.00002*
11	0.02*	0.002*	0.000005*	0.00002*
12	0.10	0.007	0.00002	0.00009
13	0.07	0.007	0.00002	0.00008
14	0.04	0.003	0.00001	0.00004
15	0.04	0.003	0.000009	0.00003
16	0.04	0.003	0.000008	0.00003
17	0.12	0.01	0.00003	0.0001
18	0.03	0.007	0.00002	0.00008
19	0.04	0.01	0.00003	0.0001
20	0.05	0.01	0.00003	0.0001
21	0.03	0.006	0.00002	0.00007
22	0.03	0.005	0.00002	0.00006
23	0.02*	0.005	0.00001	0.00006
24	0.05	0.006	0.00002	0.00008
Northern Fence Line	14.2**	1.3**	0.009**	0.016**

***Lowest concentration modelled **Highest concentration modelled**

From Table 2 it is evident that Under Scenario 2, all modelled 24-h concentrations and all modelled annual concentrations of $PM_{2.5}$ for the 24 receptor points, as well as the Northern Fence Line, were well below the South African ambient air quality standards. All concentrations were also considerably lower than under Scenario 1 (Saldanha Steelmaking operations only).

The highest 24-h $PM_{2.5}$ concentration for the receptor points, was modelled for Receptor Point 3, and the lowest concentrations for Receptor Points 11 and 23. For the modelled annual $PM_{2.5}$ concentrations, the highest concentration was recorded for Receptor Point 2 and the lowest for Receptor Points 1, 10 and 11. Overall, the highest 24-h and annual concentrations were again modelled for the Northern Fence Line.

All modelled annual manganese (Mn) concentrations at the receptor points, including residential areas, were well below the US-EPA IRIS chronic guideline of 0.05 µg/m^3, the WHO annual average guideline of 0.15 µg/m^3, and the Centres of Diseases Control's Minimal Risk Level for inorganic manganese of 0.3 µg/m^3.

The highest concentration modelled for the 24 receptor points, was for Receptor Point 2 (main road) and the lowest for Receptor Points 1 and 11. The maximum concentration (0.009 µg/m^3) modelled for Mn at the Northern Fence Line, is also below all the international guidelines mentioned above.

All modelled annual lead concentrations at the receptor points were well below the South African annual ambient air quality standard of 0.5 µg/m^3, with the highest concentration modelled for Receptor Point 2 (main road) and the lowest for Receptor Points 1, 10 and 11. The maximum concentration modelled for the Northern Fence Line was again the highest, although still below the South African annual standard.

Table 3. Maximum 24-h and annual concentrations of $PM_{2.5}$ modelled for Scenario 3 (Steelmaking and Logistics Hub).

Receptor point	Max 24-h $PM_{2.5}$ µg/m^3	Max annual $PM_{2.5}$ µg/m^3
1	1.44*	0.10*
2	31.53**	8.35**
3	15.07	1.59
4	5.98	0.55
5	2.36	0.23
6	2.47	0.23
7	2.66	0.23
8	3.54	0.29
9	1.65	0.15
10	1.83	0.12
11	1.63	0.11
12	3.12	0.24
13	2.65	0.23
14	1.57	0.12
15	1.57	0.11
16	1.51	0.11
17	3.16	0.30
18	3.18	0.51
19	5.15	0.83
20	5.97	0.87
21	3.38	0.46
22	2.65	0.39
23	2.51	0.38
24	3.89	0.45
Northern Fence Line	94.15**	23.2**

***Lowest concentration modelled **Highest concentration modelled**

From Table 3 it is clear that Under Scenario 3, all modelled 24-h concentrations and all modelled annual concentrations of $PM_{2.5}$ once again, were lower than the South African ambient air quality standards of 40 µg/m^3 for the 24-h standard and 20 µg/m^3 for the annual

standard at the 24 receptor points. Concentrations were, however, higher than under previous scenarios (Scenarios 1 and 2).

The highest 24-h $PM_{2.5}$ concentration was modelled for Receptor Point 2, and the lowest for Receptor Point 1. The same is true for the modelled annual concentrations. The concentration for Receptor Point 2 (main road) is already 79% of the 24-h $PM_{2.5}$ standard.

Maximum concentrations modelled for the Northern Fence Line exceeded the 24-h and annual South African standards for $PM_{2.5}$. The modelled 24-h concentration were more than double the South African 24-h standard.

Monitored $PM_{2.5}$ and dustfall

Available monitored results (WSP, 2023) of $PM_{2.5}$ were limited to Saldanha Bay and for 2017 and 2018 only. Data capture was poor (51%) and it was decided not to use the data.

Dustfall data were available for a few receptor points for the period July 2018 to March 2019, and for on-site Receptor Points for the period January 2017 and December 2020 (WSP, 2023). As mentioned before (Section 5.1.1) Total Suspended Particulate Matter and dustfall cannot be used to determine health risks, as the particles are normally not respirable and not presented as a concentration.

5.3 Dose-response assessment

In the dose-response assessment step of the human health risk assessment (HHRA), benchmark values ("safe" values) from reliable databases are used. Benchmark values derived from epidemiological and toxicological studies are available for many pollutants. However, when risk assessment is performed for criteria pollutants (those pollutants commonly found in ambient air), ambient air guidelines or standards of the specific country are used as benchmark values. If the specific country does not have guidelines or standards, those from other databases, such as the WHO, Europe or the US-EPA may be used. Standards may be legally enforced, but guidelines not.

5.3.1 Benchmark values for Particulate Matter

As described in the Hazard Identification section (Appendix I), the most recent meta-analyses of short-term and long-term studies on PM indicated there is no threshold and that the concentration-response graph is linear. For every 10 $\mu g/m^3$ increase in concentration, there was an increase in relative risk for a number of mortalities (Chen and Hoek, 2020; Orellano et. al, 2020, WHO, 2021). Due to the fact that no threshold could be demonstrated, even as low as 5 $\mu g/m^3$ guidelines and standards may not offer complete protection against adverse health effects.

Air quality standards set in various countries and based on health effects, are standards that the public may be continuously exposed to (ambient standards) and they are set to protect children (whose physiological systems are still developing), the aged (who's physiological systems are declining) and asthmatics, over a life time. Ambient standards are therefore much lower than occupational standards, where individuals are exposed only for a certain period (about 8 hours) per day. South African standards are stipulated for each of a number of criteria pollutants, together with their averaging periods and the frequency of exceedance allowed. The averaging period refers to the period of time over which an average has to be calculated. The frequency of exceedance refers to the number of times the limit value may be exceeded within one calendar year. If the limit value is exceeded on more occasions than specified, then there is no longer compliance with that standard.

The standards and guidelines for $PM_{2.5}$ applicable in this HHRA, are as in Table 4. The South African standard was used, and those of the US-EPA and the UK as well as the WHO guidelines, are stated for comparison.

Table 4. Guidelines and standards for $PM_{2.5}$ used in the risk assessment.

Pollutant	Time	SA Std[1] Ambient µg/m³	WHO ambient and indoor guidelines[2] µg/m³					US-EPA[3] µg/m³	UK[4] µg/m³
$PM_{2.5}$	24-h		Target 1	Target 2	Target 3	Target 4	Guideline		
		40	75	50	37.5	25	15	35	No 24-h std

SA 2012 (2) WHO 2021 (3) US-EPA, 2024 (4) UK DEFRA, 2023

$PM_{2.5}$ annual		Target 1	Target 2	Target 3	Target 4	Guideline		
	20 15[a]	35	25	15	10	5	9	20

a Effective from 1 January 2030

The South African 24-h standard for $PM_{2.5}$, is 40 µg/m^3, calculated as an average over a 24-hour period, that should not be exceeded more than 4 times per calendar year (SA, 2012). From Table 4 it is evident that the 24-h standard is similar to Interim Target 3 of the WHO. The annual standard, calculated as an annual average, is not allowed to be exceeded for South Africa.

The WHO $PM_{2.5}$ guideline is defined as the "99th percentile of the annual distribution of 24-h average concentrations (equivalent to 3-4 exceedance days per year)" (WHO, 2021). This 24-h guideline of 15 µg/m^3, was based on meta-analyses that showed a relative risk (RR) of 1.0065 for all-cause non-accidental mortality for every 10 µg/m^3 increase in $PM_{2.5}$ concentration. Thus, an excess mortality of 0.65% on a day when the $PM_{2.5}$ concentration is 15 µg/m^3 instead of 5 µg/m^3. A linear relationship is assumed and the association persisted to very low levels (WHO, 2021). The long-term and short-term guidelines for different sizes of PM as set by the WHO, are applicable to ambient as well as indoor air (WHO, 2021).

The WHO guideline for $PM_{2.5}$ has interim targets (see Table 4). These interim targets are concentrations associated with a decrease in health risk and are used as steps in the process of reducing air pollution in areas where air pollution is relatively high (WHO, 2021). The South African standard of 40 µg/m^3 is stricter than the WHO interim targets 1 and 2 and is similar to interim target 3.

There is a 1.2% increase in short-term mortality when exposed to the concentration of Interim Target 3, compared to being exposed to the concentration of Interim Target 4 (WHO, 2017). Since the South African 24-h standard is similar to the WHO Interim Target 3, it is expected that the risk of short-term mortality will also increase by 1.2% when exposed to the South African 24-h $PM_{2.5}$ standard of 40 µg/m^3 compared to being exposed to the WHO interim target 4 of 25 µg/m^3 (WHO, 2017).

In 2023, the US-EPA considered the annual standard for $PM_{2.5}$ as the controlling standard, and decided to start a process to reduce it, while at the same time, decided to keep the 24-h standard of 35 µg/m^3 (US-EPA, 2023a). An area complies to the standard if the 98th percentile of 24-hour concentrations in one year, averaged over three years, is ≤ 35 µg/m^3. On 7 February 2024, the US-EPA announced their new annual primary (health-based) $PM_{2.5}$ standard of 9 µg/m^3 (previously 12 µg/m^3) for $PM_{2.5}$ (US-EPA, 2024).

Currently the United Kingdom (UK) has an annual mean standard of 20 µg/m^3, that since the 1st of January 2020, is not allowed to be exceeded (DEFRA, 2023). No 24-h standard/limit value could be located for the UK in the literature searched.

5.3.2 Benchmark values for Manganese, Lead and Silica

It is important to note that the air quality guidelines for pollutants that were not updated by the WHO in 2021, such as manganese and lead, remain the same as in a previous update of the WHO (2000) (WHO, 2021).

Manganese (Mn)

No short-term ambient standards or guidelines could be found for Mn in the literature searched. South Africa does not have an ambient standard for Mn; only an occupational standard for elemental Mn of 0.04 mg/m^3 (40 µg/m^3) as an 8-h Time Weighted Average (TWA) (measured as a respirable fraction). They consider respirable particulates as particulates with a median cut point of 4 µm. (SA, 2021).

The WHO (2000) has an annual average guideline of 0.15 µg/m^3 for Mn, based on studies that showed a No Observed Adverse Effect Level (NOAEL) of 30 µg/m^3.

The US-EPA Integrated Risk Information System (IRIS) has an RfC (Reference Concentration) of 0.05 µg/m^3, based on impairment of neurobehavioural function found in occupational studies. The Lowest Observed Adverse Effect Level (LOAEL) was 50 µg/m^3 (IRIS. 1993). An RfC is for chronic inhalation. Since the US-EPA Framework was used in this assessment, it was decided to use the US-EPA benchmark value as well, together with that of the WHO.

The Centers for Disease Control (CDC) in the US, under their Agency for Toxic Substances and Disease Registry (ATSDR) has a chronic Minimal Risk Level (MRL) of 0.0003 mg/m^3 (0.3 µg/m^3) for chronic-duration (≥1 year) inhalation exposure to inorganic manganese (CDC, 2012).

Lead (Pb)

South Africa does not have an occupational standard for lead in air. The draft lead regulations published in the Government Gazette on 1 March 2024 (SA, 2024), stipulates regular surveillance of workers in terms of lead concentrations in blood and urine, to ensure it remains below certain levels.

South Africa has an ambient air quality standard for lead of 0.5 µg/m^3 as an annual average (SA, 2009). This standard was used in this HHRA, as it may be enforced by law.

The WHO (2000) also has an annual average guideline of 0.5 µg/m^3 for Pb in air. The guideline for lead aims to keep the blood lead levels relatively low. They consider adverse effects (hearing impairment) to start at blood lead levels of 100 µg/L.

The US-EPA has an environmental standard of 0.15 µg/m^3 for Pb in air, measured as a rolling 3-month average that must not be exceeded (US-EPA, 2024).

“Current knowledge of lead pharmacokinetics indicates that risk values derived by standard procedures would not truly indicate the potential risk, because of the difficulty in accounting for pre-existing body burdens of lead. Lead bioaccumulates in the body, primarily in the skeleton. Lead body burdens vary significantly with age, health status, nutritional state,

maternal body burden during gestation and lactation, etc. For this reason, and because of the continued apparent lack of threshold it is still inappropriate to develop reference values for lead".

Silica (Si)

Ambient air standards for silica are not available in most countries. There are many uncertainties as to what the no observed adverse effect level (NOAEL) of respirable crystalline silica would be, in other words, it is uncertain at what level would no adverse health effects be expected (CDC, 2019).

The South African Occupational exposure limit for silica is 0.1 mg/m^3 (100 µg/m^3) (SA, 2021).

One long-term exposure guideline was found that was set to protect sensitive individuals against silicosis, namely the Reference Exposure Level (REL) of the California-EPA (Cal-EPA, 2008), which is 3 µg/m^3 for inhalation of respirable crystalline silica. Respirable in this case was defined as "a 50% cut-point at 4 µm particle aerodynamic diameter" (Cal-EPA, 2008). The REL is defined as "a concentration level at (or below) which no health effects are anticipated" This REL was used as benchmark value in this assessment.

5.4 Risk Characterisation

In the risk characterisation step of the HHRA, the risk is quantified by calculating a Hazard Quotient (HQ). The maximum modelled concentrations and the benchmark values were used to determine the (HQ), which describes the potential for developing detrimental health effects (other than cancer) from exposure to a pollutant. The HQ is the ratio of an air pollutant's concentration over a specified period (short-term or long-term) to a reference concentration for that pollutant for a similar exposure period. The HQ is unitless. The formula is as follows:

HQ (in the case of inhalation) = C/RfC, where:

C = Concentration for a specified period

RfC = Reference Concentration (or benchmark value or standard or guideline) for that pollutant for the same time period.

For acute (short-term) effects the time period will generally be 1-h or 24-hs, and for chronic (long-term) effects, one to several years.

If the determined HQ is below 1, it is an indication that it will be unlikely for individuals, even sensitive individuals, to experience detrimental health effects, but when the HQ is above 1, the potential for a detrimental effect does exist. The potential, however, does not increase linearly. It does therefore not mean that everyone exposed to conditions where the HQ is above 1 will necessarily experience adverse health effects.

In this HHRA, acute non-cancer risks, which are associated with short-term (24-hr) exposure, were quantitatively assessed (as HQs) for $PM_{2.5}$ under each scenario. Chronic non-cancer risks, associated with long-term (annual) exposure, were quantitatively assessed for $PM_{2.5,}$ under all scenarios, and for manganese lead and silica under Scenario 2.

Cancer risks were addressed for PM and silica, the only confirmed human carcinogens (as per the IARC) amongst the pollutants of concern. The risk from inadvertent ingestion of soil, was addressed for lead.

The HQs and applicable benchmarks used to determine risk, are presented under each scenario in Tables 5 to 7.

Table 5. Hazard Quotients (HQs) calculated for $PM_{2.5}$ for Scenario 1 (Steel Operations only), using different benchmarks (SA & WHO).

Receptor point	Max 24-h $PM_{2.5}$ µg/m³	HQ 24-h $PM_{2.5}$ SA (40 µg/m³)	HQ 24-h $PM_{2.5}$ WHO (15 µg/m³)	Max annual $PM_{2.5}$ µg/m³	HQ annual $PM_{2.5}$ SA (20 µg/m³)	HQ annual $PM_{2.5}$ WHO (5 µg/m³)
1	1.42	0.03	0.09	0.10	0.005	0.02
2	31.11	0.77	**2.07**	8.25	0.41	**1.65**
3	14.36	0.36	0.95	1.51	0.08	0.30
4	5.69	0.14	0.38	0.51	0.03	0.10
5	2.31	0.06	0.15	0.22	0.01	0.04
6	2.42	0.06	0.16	0.22	0.01	0.04
7	2.61	0.07	0.17	0.23	0.01	0.05
8	3.49	0.09	0.23	0.29	0.01	0.06
9	1.62	0.04	0.11	0.15	0.008	0.03
10	1.80	0.05	0.12	0.12	0.006	0.02

Receptor point	Max 24-h $PM_{2.5}$ µg/m³	HQ 24-h $PM_{2.5}$ SA (40 µg/m³)	HQ 24-h $PM_{2.5}$ WHO (15 µg/m³)	Max annual $PM_{2.5}$ µg/m³	HQ annual $PM_{2.5}$ SA (20 µg/m³)	HQ annual $PM_{2.5}$ WHO (5 µg/m³)
11	1.60	0.04	0.12	0.11	0.005	0.02
12	3.03	0.08	0.20	0.23	0.01	0.05
13	2.57	0.06	0.17	0.22	0.01	0.04
14	1.53	0.04	0.10	0.12	0.006	0.02
15	1.53	0.04	0.10	0.11	0.005	0.02
16	1.46	0.04	0.10	0.11	0.006	0.02
17	3.04	0.08	0.20	0.29	0.01	0.06
18	3.14	0.08	0.21	0.50	0.02	0.10
19	5.11	0.13	0.34	0.81	0.04	0.16
20	5.92	0.15	0.40	0.86	0.04	0.17
21	3.35	0.08	0.22	0.45	0.02	0.09
22	2.62	0.07	0.17	0.38	0.02	0.08
23	2.49	0.06	0.17	0.38	0.02	0.08
24	3.84	0.10	0.26	0.45	0.02	0.09
Northern Fence Line	66.91	**1.67**	**4.46**	21.46	**1.07**	**4.28**

HQs above 1 in bold

Table 6. Hazard Quotients (HQs) calculated for $PM_{2.5}$, Mn and Pb, for Scenario 2 (Logistics Hub Operations only), using different benchmarks (SA, WHO and IRIS).

Recep-tor Point	Max 24-h $PM_{2.5}$ µg/m³	HQ 24-h $PM_{2.5}$ SA (40 µg/m³)	HQ 24-h $PM_{2.5}$ WHO (15 µg/m³)	Max annual $PM_{2.5}$ µg/m³	HQ annual $PM_{2.5}$ SA (20 µg/m³)	HQ annual $PM_{2.5}$ WHO (5 µg/m³)	Max Annual Mn µg/m³	HQ Annual Mn IRIS (0.05 µg/m³)	HQ Annual Mn WHO (0.15 µg/m³)	Max Annual Pb µg/m³	HQ Annual Pb SA (0.5 µg/m³)
1	0.03	0.0006	0.002	0.002	0.00008	0.0003	0.000005	0.00009	0.00003	0.00002	0.00004
2	0.42	0.01	0.03	0.10	0.005	0.02	0.0005	0.009	0.003	0.001	0.002
3	0.71	0.02	0.05	0.08	0.004	0.02	0.0002	0.004	0.001	0.0009	0.002
4	0.29	0.007	0.02	0.03	0.002	0.007	0.00006	0.001	0.0004	0.0004	0.0008
5	0.04	0.001	0.003	0.004	0.0002	0.0009	0.00001	0.0002	0.00008	0.00005	0.0001
6	0.04	0.001	0.003	0.004	0.0002	0.0009	0.00001	0.0002	0.00007	0.00005	0.0001
7	0.04	0.001	0.003	0.004	0.0002	0.0009	0.00001	0.0002	0.00008	0.00005	0.0001

Recep-tor Point	Max 24-h $PM_{2.5}$ µg/m³	HQ 24-h $PM_{2.5}$ SA (40 µg/m³)	HQ 24-h $PM_{2.5}$ WHO (15 µg/m³)	Max annual $PM_{2.5}$ µg/m³	HQ annual $PM_{2.5}$ SA (20 µg/m³)	HQ annual $PM_{2.5}$ WHO (5 µg/m³)	Max Annual Mn µg/m³	HQ Annual Mn IRIS (0.05 µg/m³)	HQ Annual Mn WHO (0.15 µg/m³)	Max Annual Pb µg/m³	HQ Annual Pb SA (0.5 µg/m³)
8	0.05	0.001	0.003	0.005	0.0003	0.001	0.00002	0.0003	0.0001	0.00006	0.0001
9	0.03	0.0007	0.002	0.003	0.0001	0.0005	0.000008	0.0002	0.00005	0.00003	0.00006
10	0.03	0.0007	0.002	0.002	0.0001	0.0004	0.000006	0.0001	0.00004	0.00002	0.00005
11	0.02	0.0006	0.002	0.002	0.0001	0.0004	0.000005	0.00009	0.00003	0.00002	0.00004
12	0.10	0.002	0.007	0.007	0.0004	0.001	0.00002	0.0004	0.0001	0.00009	0.0002
13	0.07	0.002	0.005	0.007	0.0003	0.001	0.00002	0.0004	0.0001	0.00008	0.0002
14	0.04	0.001	0.003	0.003	0.0002	0.0006	0.00001	0.0002	0.00007	0.00004	0.00007
15	0.04	0.001	0.003	0.003	0.0001	0.0006	0.000009	0.0002	0.00006	0.00003	0.00007
16	0.04	0.001	0.003	0.003	0.0001	0.0006	0.000008	0.0002	0.00005	0.00003	0.00007
17	0.12	0.003	0.008	0.01	0.0005	0.002	0.00003	0.0006	0.0002	0.0001	0.0002
18	0.03	0.0009	0.002	0.007	0.0003	0.001	0.00002	0.0004	0.0001	0.00008	0.0002
19	0.04	0.001	0.003	0.01	0.0005	0.002	0.00003	0.0007	0.0002	0.0001	0.0002
20	0.05	0.001	0.003	0.01	0.0005	0.002	0.00003	0.0006	0.0002	0.0001	0.0002
21	0.03	0.0007	0.002	0.006	0.0003	0.001	0.00002	0.0003	0.0001	0.00007	0.0001
22	0.03	0.0008	0.002	0.005	0.0003	0.001	0.00002	0.0003	0.0001	0.00006	0.0001
23	0.02	0.0006	0.002	0.005	0.0002	0.001	0.00001	0.0003	0.00009	0.00006	0.0001
24	0.05	0.001	0.003	0.006	0.0003	0.001	0.00002	0.0004	0.0001	0.00008	0.0002
Northern Fence Line	14.17	0.35	0.94	1.33	0.07	0.27	0.009	0.18	0.06	0.016	0.03

Table 7. Hazard Quotients (HQs) calculated for $PM_{2.5}$, for Scenario 3 (Cumulative Operations), using different benchmarks (SA and WHO).

Receptor point	Max 24-h $PM_{2.5}$ µg/m³	HQ 24-h $PM_{2.5}$ SA (40 µg/m³)	HQ 24-h $PM_{2.5}$ WHO (15 µg/m³)	Max annual $PM_{2.5}$ µg/m³	HQ annual $PM_{2.5}$ SA (20 µg/m³)	HQ annual $PM_{2.5}$ WHO (5 µg/m³)
1	1.44	0.04	0.10	0.10	0.005	0.02
2	31.53	0.79	**2.10**	8.35	0.40	**1.67**
3	15.07	0.38	1.00	1.59	0.08	0.32

Receptor point	Max 24-h $PM_{2.5}$ µg/m³	HQ 24-h $PM_{2.5}$ SA (40 µg/m³)	HQ 24-h $PM_{2.5}$ WHO (15 µg/m³)	Max annual $PM_{2.5}$ µg/m³	HQ annual $PM_{2.5}$ SA (20 µg/m³)	HQ annual $PM_{2.5}$ WHO (5 µg/m³)
4	5.98	0.15	0.40	0.55	0.03	0.11
5	2.36	0.06	0.16	0.23	0.01	0.05
6	2.47	0.06	0.16	0.23	0.01	0.05
7	2.66	0.07	0.18	0.23	0.01	0.05
8	3.54	0.09	0.24	0.29	0.01	0.06
9	1.65	0.04	0.11	0.15	0.008	0.03
10	1.83	0.05	0.12	0.12	0.006	0.03
11	1.63	0.04	0.11	0.11	0.006	0.02
12	3.12	0.08	0.21	0.24	0.01	0.05
13	2.65	0.07	0.18	0.23	0.01	0.05
14	1.57	0.04	0.10	0.12	0.006	0.02
15	1.57	0.04	0.11	0.11	0.006	0.02
16	1.51	0.04	0.10	0.11	0.006	0.02
17	3.16	0.08	0.21	0.30	0.01	0.06
18	3.18	0.08	0.21	0.51	0.03	0.10
19	5.15	0.13	0.34	0.83	0.04	0.17
20	5.97	0.15	0.40	0.87	0.04	0.17
21	3.38	0.08	0.23	0.46	0.02	0.09
22	2.65	0.07	0.18	0.39	0.02	0.08
23	2.51	0.06	0.17	0.38	0.02	0.08
24	3.89	0.10	0.26	0.45	0.02	0.09
Northern Fence Line	94.2	**2.36**	**6.28**	23.2	**1.16**	**4.64**

HQs in bold are above 1.

5.4.1 Inhalation risk estimates for Scenario 1 (Steel Operations only)

Acute risks – $PM_{2.5}$

When considering the South African and US-EPA 24-h standards for $PM_{2.5}$, as well as the WHO guidelines up to Interim Target 3, the HQs determined for Receptor Points 1 to 24, indicated that it would be unlikely for individuals to develop adverse effects (see Table 5). Under the South African standard, HQs ranged from 0.03 at Receptor Point 1, to 0.77 at Receptor point 2 (main road). The second highest HQ was calculated for Receptor Point 3 (at the Southern Fence Line), namely 0.36 (see Figure 1 Section 5.2.1).

However, when considering the WHO final 24-h guideline of 15 µg/m^3, then the HQs ranged from 0.09 to 2.07. Receptor Point 2 had the highest HQ (2.07), indicating that individuals exposed at this receptor point were at risk of adverse health effects such as respiratory effects. Receptor Point 2 (main road) was the only receptor point where the HQ was above 1. Receptor Point 2 is situated outside of the fence line, about 500 m North of the northern fence line. The second highest HQ (0.95), was calculated for Receptor Point 3 (Southern Fence Line), with all other HQs including residential areas, well below 1.

When considering the short-term maximum concentrations modelled for the Northern Fence Line site, the calculated HQs indicate a potential for acute health effects, such as respiratory effects, regardless of the benchmark used. HQs were above 1 using either the South African standard of 40 µg/m^3 or the WHO final guideline of 15 µg/m^3.

The short-term Relative Risk of $PM_{2.5}$ for all-cause (non-accidental) mortality (death) is 1,0065 for every 10 µg/m^3 increase in concentration with a departure point of 5 µg/m^3. This indicates that at a daily concentration of 15 µg/m^3 a 0.65% increase in all-cause (non-accidental) mortality is expected for that day.

Chronic risks $PM_{2.5}$

The HQs calculated from long-term exposure to the modelled annual concentrations under Scenario 1 (see Table 5), show that it will be unlikely for any individual to develop adverse health effects when considering the South African, US-EPA and UK standards, as well as the

WHO guidelines up to Interim Target 4. Using the South African annual standard, HQs ranged from 0.005 at Receptor Point 1, to 0.41 at Receptor Point 2 (main road). The second highest HQ (0.08) was again calculated for Receptor Point 3 (Southern Fence Line).

There is, however, a potential for adverse effects at Receptor Point 2, as indicated by the HQ of 1.65, calculated, using the WHO strict **final** guideline of 5 µg/m^3. The lowest HQ (0.02) was at Receptor Point 1. The second highest (0.30) was again at Receptor Point 3.

The South African annual standard of 20 µg/m^3 for $PM_{2.5}$ falls between the WHO Interim Targets 2 and 3, which are 25 µg/m^3 and 15 µg/m^3 respectively. Given a linear concentration-response (C-R) function of 1.08 per 10 µg/m^3 for long-term $PM_{2.5}$ exposure, the following assumption can be made: If the all-cause non-accidental mortality in a community is set at 100, then the mortality will be 116 at Interim Target 2 and 108 at Interim Target 3. Considering the South African standard, it will fall between those two figures.

When considering the annual maximum concentrations modelled for the Northern Fence Line site, the calculated HQs indicate a potential for chronic health effects, such as respiratory effects, cardiovascular effects, regardless of the benchmark used. HQs were above 1 using either the South African standard of 20 µg/m^3 or the WHO final guideline of 5 µg/m^3.

All HQs calculated for residential areas were below 1, indicating that it will be unlikely for any individual in these areas to develop adverse health effects as a result of exposure to the modelled $PM_{2.5}$ concentrations.

The International Agency for Research on Cancer (IARC) (2021), classified outdoor Particulate Matter (PM), as a confirmed human carcinogen. However, the incremental cancer risk for the general public could not be determined in this HHRA, as no approved cancer potency factor (inhalation unit risk) for ambient PM could be found in the literature searched.

5.4.2 Inhalation risk estimates for Scenario 2 (Logistics Hub Operations only)

Acute risks – $PM_{2.5}$

When considering the modelled 24-h $PM_{2.5}$ concentrations under Scenario 2, all HQs calculated were well below 1 (see Table 6), indicating that it will be unlikely for any individual, even sensitive individuals, to experience adverse health effects. This was true considering the South African standard (40 µg/m^3), as well as the US-EPA standard (35 µg/m^3) and the final WHO guideline (15 µg/m^3).

Under the South African standard, HQs ranged from 0.0006 at Receptor Points 1, 11 and 23, to 0.02 at Receptor Point 3. Under the WHO guideline, HQs ranged from 0.002 (at Receptor Points 1, 9,10,11,18,21,22 and 23) to 0.05 at Receptor Point 3. Receptor Point 3 is at the Southern Fence Line.

Chronic risks $PM_{2.5}$

The HQs calculated from long-term exposure to the modelled annual concentrations under Scenario 2 (see Table 6), also indicate that it will be unlikely for any individual to develop adverse health effects when considering the South African, US-EPA and UK standards, as well as the WHO final guideline.

Using the South African annual standard (20 µg/m^3), HQs ranged from 0.00008 at Receptor Point 1, to 0.005 at Receptor Point 2. The second highest HQ (0.004) was again calculated for Receptor Point 3. With the WHO final guideline of 5 µg/m^3, HQs ranged from 0.0003 at Receptor Point 1, to 0.02 at Receptor Points 2 (main road) and 3 (Southern Fence Line).

Although outdoor PM is classified as a confirmed human carcinogen, the incremental cancer risk for the general public could not be determined under Scenario 2, as no approved cancer inhalation unit risk for ambient PM could be found in the literature searched.

Chronic inhalation risks for Manganese (Mn)

The modelled annual manganese concentrations were used to calculate the potential for chronic adverse effects. Considering the US-EPA Integrated Risk Information System (IRIS)'s

Reference Concentration (RfC) of 0.05 µg/m^3 as benchmark value, calculated HQs for the various receptor points (Table 6) varied from 0.00009 at Receptor Points 1 and 11, to 0.009 at Receptor Point 2. The second highest HQ (0.004) was again found at Receptor Point 3 (Southern Fence Line). The HQs indicated that adverse health effects such as neurological effects from Mn exposure would be unlikely.

When using the WHO guideline, all HQs were also below 1. HQs ranged from 0.00003 at Receptor Points 1 and 11, to 0.003 at Receptor Point 2 (main road).

The HQs calculated from the maximum modelled annual concentration for Mn at the Northern Fence Line were the highest, namely 0.18 with the IRIS guideline and 0.06 with the WHO guideline. However, both these HQs were below 1, indicating that adverse health effects would be unlikely. As Mn is not classified as a confirmed human carcinogen by the IARC, no carcinogenic risks were determined for Mn.

Chronic inhalation risks for Lead (Pd)

The modelled annual lead concentrations were used to calculate the potential for chronic adverse effects. Considering the South African ambient annual standard of 0.5 µg/m^3 as benchmark value, calculated HQs (Table 6) varied from 0.00004 at Receptor Points 1 and 11, to 0.002 at Receptor Points 2 and 3. The HQs indicated that adverse health effects such as neurological effects from Pb exposure would be unlikely. This was also true when the maximum modelled annual concentration of Pb at the Northern Fence Line was considered.

As Pb is not classified as a confirmed human carcinogen by the IARC, no carcinogenic risks were determined for Pb.

Risk for ingestion of Pb

The total dust-fall and the lead dust-fall were modelled for Scenario 2. From these two values, the percentage lead in the dust was calculated and was found to be 4.5%. As children are susceptible to the effects of lead and hand-mouth contact is the main route of children's exposure to lead, it was decided to create situations in which the potential for adverse effects would be determined for a 6-year-old child.

The risk from inadvertent ingestion of soil and wind-blown dust was determined, assuming that 4.5% of the soil individuals will ingest, will be lead. It was further assumed that the intake rate was 400 mg/day, and the body weight 15 kg, as recommended by the SA Framework for Management of Contaminated Land (DEA, 2010) for a child in an **informal** residential area. In another situation the risk was determined at an intake rate of 200 mg/day as recommended by the same framework for a child in a **formal** residential area.

The US-EPA published an update for soil and dust ingestion as part of Chapter 5 of the Exposure Factors Handbook, in 2017 (US-EPA, 2017), where the soil and dust ingestion rate for a child between 6 and 12 years of age, is stated as 60 mg/day. Therefore, in the third situation, the risk to a 6-year-old child was determined, using this updated soil and dust ingestion rate and a bodyweight of 31.8 kg as stated in Chapter 8 of the 2011 issue of the Exposure Factors Handbook (US-EPA, 2011).

An Average Daily Dose (ADD) was determined, using the following formula:

ADD (Average Daily Dose) in mg/kg bodyweight/day = (C X IR X EF X ED) / (BW X T)

Where C = Concentration of pollutant in the specific medium (mg/kg)

IR = Intake Rate in kg/day

EF = Exposure Frequency in days per year

ED = Exposure Duration in years

BW = Body Weight in kg

T = Time in days (years X 365)

The calculated average daily dose (ADD) for a 6-year-old child using South African criteria for an informal residential area (ingesting 400 mg/day), was 1.20 mg/kg bodyweight/day. For an ingestion rate of 200 mg/day (formal residential area), the ADD was 0.60 mg/kg bodyweight/day. The ADD calculated using the EPA criteria, was 0.08 mg/kg bodyweight/day.

To determine an HQ, these average daily doses have to be divided by a reference dose (RfD). However, no RfD for lead could be found in the databases searched, such as the US-EPA Integrated Risk Information System (where the RfD is currently under review) and others.

An expert committee of the WHO evaluated publications on lead in food, and came to the conclusion that there is no safe dose for lead. Even at a chronic dietary exposure of 0.6 µg/kg body weight/day, a decrease of 1 IQ point was estimated (WHO 2011). This dose is considerably lower than those calculated above, using the modelled lead data in dustfall. Modelled lead in dustfall was used, as it is not possible to predict what the concentration of lead in soil will be once the Hub is in operation. The uncertainty is high in the assumption that the soil will contain 4.5% lead, because that was modelled to be the percentage lead in the dustfall in Scenario 2.

Chronic inhalation risks for Silica (Si)

Concentrations for Si were not modelled. As a worst-case scenario it was assumed that 100% of the modelled annual $PM_{2.5}$ concentrations under Scenario 2, consisted of crystalline silica. These modelled concentrations and the long-term Reference Exposure Level (REL) of the California-EPA of 3 µg/m^3 (Cal-EPA, 2005) (for inhalation of respirable crystalline silica), were used to quantify the potential for adverse health effects, such as silicosis. HQs ranged from 0.0006 at Receptor Points 1 and 11, to 0.03 at Receptor Points 2 and 3. It will thus be unlikely that any individual will develop adverse effects such as silicosis, as a result of exposure to the concentrations of crystalline silica calculated for Scenario 2, as all HQs were below 1.

Carcinogenic risk for Si

The International Agency for Research on Cancer (IARC) (2021), classified crystalline silica, inhaled in the form of quartz from occupational sources, as a confirmed human carcinogen. However, the incremental cancer risk for the general public could not be determined in this HHRA, as no approved cancer potency factor (inhalation unit risk) for silica could be found in the literature searched.

Lacasse et al., (2009) and Poinen-Rughooputh et al., (2016) performed meta-analyses on many studies to determine the association between silica exposure and lung cancer. Lacasse et al., came to the conclusion that “increased risk is particularly apparent when the cumulative exposure to silica is well beyond that resulting from exposure to the recommended limit concentration for a prolonged period of time”. Seven years later, Poinen-Rughooputh et al., came to more or less the same conclusion, when they stated the association is “more pronounced at higher levels of exposure, in the presence of silicosis and in the mining industry”.

5.4.3 Inhalation risk estimates for Scenario 3 (Cumulative Operations)

Acute risks – $PM_{2.5}$

When considering the South African and US-EPA 24-h standards for $PM_{2.5}$, as well as the WHO guidelines up to Interim Target 3, the HQs determined under Scenario 3, indicated that it would be unlikely for individuals to develop adverse effects. From Table 7, it is evident that under the South African 24-h standard, HQs ranged from 0.04 at Receptor Points 1, 9, 11, 14, 15 and 16 to 0.79 at Receptor point 2. The second highest HQ (0.38) was calculated for Receptor Point 3 (Southern Fence Line) (see Figure 1 Section 5.2.1).

However, when considering the WHO final 24-h guideline of 15 µg/m^3, then the HQs ranged from 0.10 (at Receptor Points 1, 14 and 16) to 2.10 at Receptor Point 2 (main road). The HQ at Receptor Point 2 is an indication that individuals at this receptor point may develop adverse effects, such as respiratory effects. Receptor Point 2, which is situated outside of the fence line, about 500 m North of the Northern Fence Line, was the only site where the HQ was above 1. The second highest HQ (1.00), was calculated for Receptor Point 3 (Southern Fence Line), with all other HQs well below 1.

Considering the HQs calculated from the maximum modelled 24-h $PM_{2.5}$ concentrations for the Northern Fence Line site, it is evident that there is a potential for developing acute adverse health effects regardless of the standard (SA) or guideline (WHO) used in the HQ calculations.

Again, it must be noted that for every 10 µg/m^3 increase in concentration of $PM_{2.5,}$ the short-term all-cause (non-accidental) mortality (death) is expected to increase by 0.65% (WHO, 2021).

Chronic risks $PM_{2.5}$

The HQs calculated from long-term exposure to the modelled annual concentrations under Scenario 3 (Table 7), show that it will be unlikely for any individual to develop adverse health effects when considering the South African, US-EPA and UK standards, as well as the WHO guidelines up to Interim Target 4. Using the South African annual standard of 20 µg/m^3, HQs

ranged from 0.005 at Receptor Point 1, to 0.42 at Receptor Point 2. The second highest HQ (0.08) was again calculated for Receptor Point 3 (Southern Fence Line).

When considering the WHO strict **final** guideline of 5 µg/m^3, the HQ of 1.67 at Receptor Point 2 (main road), indicates a potential for adverse effects. The lowest HQ under Scenario 3 (0.02) was at Receptor Points 1, 10, 11, 14, 15 and 16. The second highest (0.32) was again at Receptor Point 3.

The HQs calculated for the Northern Fence Line from the maximum annual concentrations modelled for the site, were all above 1, indicating a potential for chronic adverse health effects, such as respiratory effects and cardio-vascular effects. However, all HQs calculated for residential areas were below 1.

The South African annual standard of 20 µg/m^3 for $PM_{2.5}$ falls between the WHO Interim Targets 2 and 3, which are 25 µg/m^3 and 15 µg/m^3 respectively. Given a linear concentration-response (C-R) function of 1.08 per 10 µg/m^3 for long-term $PM_{2.5}$ exposure, the following assumption can be made: If the all-cause non-accidental mortality in a community is set at 100, then the mortality will be 116 at Interim Target 2 and 108 at Interim Target 3 (WHO, 2021). Considering the South African standard, the all-cause non-accidental mortality will then fall between those two figures.

In terms of impact significance criteria used in environmental impact assessments (considering magnitude/intensity, extent, reversibility, duration, and probability of occurrence), the Human Health Risk Assessment for each Scenario may be rated as below.

Receptor	Summary of mitigation measures	Rating and Significance
Scenario 1 – Steelmaking	Tarring/paving of the service road and spraying of dust	N2 - Medium
Scenario 2– Logistics Hub	Tarring/paving of the service road and spraying of dust	N1 - Low
Scenario 2 - Cumulative	Tarring/paving of the service road and spraying of dust	N2 - Medium

Impact Nr	Receptor	Mitigation Measure Description	Stage	Character	Ease of Mitigation	Pre-Mitigation							Post-Mitigation						
						(M+	E+	R+	D)x	P=	S	Rating	(M+	E+	R+	D)x	P=	S	Rating
Scenario 1:	HHRA: $PM_{2.5}$ Scenario 1	—Concentrations of $PM_{2.5}$ for the HHRA were modelled for the operational phase only and assuming all measures to mitigate dust were in place, such as water and or chemical spraying of dust and roads, covered conveyor belts, truckloads covered with tarpaulin, etc. Therefore, pre-mitigation concentrations of $PM_{2.5}$ were not available to assess the situation, but according to the scoring system, it has to be assumed that no mitigation measures are in place — After mitigation, the max 24-h $PM_{2.5}$ concentration at fenceline (where the public	Operation	Negative		4	3	5	4	3	**48**	**N2**	4	3	5	4	2	**32**	**N2**

Impact Nr	Receptor	Mitigation Measure Description	Stage	Character	Ease of Mitigation	Pre-Mitigation							Post-Mitigation						
						(M+	E+	R+	D)x	P=	S	Rating	(M+	E+	R+	D)x	P=	S	Rating
		may be exposed) for Scenario 1 (Saldanha Steel as the only source) was 67 ug/m^3, which is still well above the SA 24-h standard of 40 ug/m^3.																	
					Significance	**N2 - Medium**							**N2 - Medium**						
Scenario 2:	HHRA: Scenario 2 (All pollutants emissions)	—Concentrations of $PM_{2.5}$, Mn, Pb and Si modelled for Scenario 2 (Logistics Hub only), also assumed all mitigation measures (as described in Scenario 1) for dust generation were in place. —Pre-mitigation situation can thus not be classified, but assumed no measures in place. Post- mitigation the probability of developing adverse health effects is low. Concentrations at fence line so low that impact is seen as local (score a 2).	Operation	Negative		5	3	5	4	2	**34**	**N2**	5	2	5	4	1	**16**	**N1**
					Significance	**N2 - Medium**							**N1 - Low**						
Scenario 15:	HHRA: PM2.5 Scenario 3	—Concentrations for Scenario 3 (Cumulative Operations) were once again modelled assuming all mitigation measures for dust control were in place. —The pre-mitigation situation could	Operation	Negative		4	3	5	4	3	**48**	**N2**	4	3	5	4	2	**32**	**N2**

Impact Nr	Receptor	Mitigation Measure Description	Stage	Character	Ease of Mitigation	Pre-Mitigation							Post-Mitigation						
						(M+	E+	R+	D)x	P=	S	Rating	(M+	E+	R+	D)x	P=	S	Rating
		therefore not be evaluated, but was assumed not to be in place. The post-mitigation situation was worse than Scenario 1. The 24-h $PM_{2.5}$ concentration was at 94.2 ug/m^3, more than double the SA standard of 40 ug/m^3 (due to one source (Saldanha Steel) only).																	
					Significance	N2 - Medium							N2 - Medium						

5.5 Uncertainties, Limitations, and Variability

5.5.1 Assumptions for the human health risk assessment

- Valid modelled concentrations of the pollutants were provided to the health risk assessor.
- The study was limited to $PM_{2.5,}$ under all three scenarios and manganese, lead and silica under Scenario 2.
- Occupational health and safety fell outside the scope of this assessment.

5.5.2 Limitations of the study

As the Logistics Hub is not operational yet, pollutants could not be monitored and had to be modelled. However, the model used is an internationally recognised dispersion model, recommended for areas of complex meteorological conditions, such as coastal environments

Available monitored results (WSP, 2023) of $PM_{2.5}$ were limited to Saldanha Bay and for 2017 and 2018 only. Data capture was poor (51%) and it was decided not to use the data.

Dustfall data were available for a few receptor points for the period July 2018 to March 2019, and for on-site Receptor Points for the period January 2017 and December 2020 (WSP, 2023). As mentioned before (Section 5.1.1) Total Suspended Particulate Matter and dustfall cannot be used to determine health risks, as the particles are normally not respirable and not presented as a concentration.

5.5.3 Variable uncertainty

There is variability in each individual's activity patterns. The specific activity patterns of the individuals potentially exposed to the $PM_{2.5}$ concentrations in this study were not known. It was conservatively assumed that individuals were exposed to the maximum concentrations for 24-hours per day.

5.5.4 Model uncertainty

Models and equations used in this Human Health Risk Assessment were from the US-EPA. The US-EPA is considered a reputable source, therefore model uncertainty was minimised.

5.5.5 Decision rule uncertainty

The compounds of concern were derived from the information on the products that will be handled at the hub, as provided to the risk assessor. The most significant exposure pathway chosen was inhalation. National and international ambient guidelines/standards were used as values that could be used to predict health risks. The South African standards used were based on human health effects. Other guidelines were from well-known reliable databases such as the WHO.

6. Conclusion

The results from the Human Health Risk Assessment of the potential risks to human health from exposure to the modelled dust emissions from the Steelmaking and proposed Logistics Hub are provided below.

The benchmarks (“safe” concentrations) used to quantify the potential for developing adverse health effects under each scenario, were the South African standards for $PM_{2.5,}$ and lead, as these standards may be enforced by law. For comparison, the World Health Organization (WHO) guidelines were used (Table 4). Where South Africa did not have standards (such as for manganese and silica), international standards or guidelines (including those of the WHO) were used (Section 5.3).

In the risk characterisation step of the assessment, risks were quantified by calculating a unitless Hazard Quotient (HQ) (Tables 5 to 7 and Section 5.4).

Scenario 1

Under Scenario 1 (Table 5), there is a potential for acute and chronic health effects from $PM_{2.5}$ at the Northern Fence Line, if the South African standards are used, and a potential for acute and chronic effects at Receptor Point 2 (main road) and the Northern Fence Line when using the WHO guidelines. However, all HQs determined for residential areas were below 1, indicating that adverse health effects would be unlikely, even in sensitive individuals.

Scenario 2

Under Scenario 2 (Table 6), the HQs calculated indicate that it would be unlikely for any individual to develop adverse health effects from exposure to the modelled $PM_{2.5,}$ manganese or lead when using South African standards, WHO guidelines, IRIS guidelines (from the US-EPA) or the Centres for Disease Control (CDC) guideline.

Concentrations for silica were not modelled. However, as a worst-case scenario it was assumed that 100% of the modelled annual $PM_{2.5}$ concentrations under Scenario 2, consisted of crystalline silica. These modelled concentrations and a guideline from the California-EPA were used to quantify the potential for adverse health effects. All HQs calculated indicated that it would be unlikely for any individual to develop chronic health effects such as silicosis.

Scenario 3

Under Scenario 3 (Table 7), the HQs calculated were similar to the situation under Scenario 1. There is a potential for acute and chronic health effects from $PM_{2.5}$ at the Northern Fence Line, when the South African standards were used, and a potential for acute and chronic effects at Receptor Point 2 (main road) and the Northern Fence Line when using the WHO guidelines. However, all HQs calculated for residential areas, using the modelled data, showed it will be unlikely for any individual, even sensitive individuals, to develop adverse effects.

Impact Signifiance

In term of the impact significance criteria used in the environmental impact assessments, with mitigation, the impact significance of Scenarios 1 and 3 are rated as Medium Negative, and the impact significance of Scenario 2 is rated as Low Negative.

The proposed Logistics Hub development (Scenario 2) should therefore be considered for development subject to the implementation of the mitigation and monitoring measures, maintenance and operation controls included in the EMPr and the Fugitive Dust Management Plan. Application of these measures will ensure that the public are not negatively impacted or at risk due to the operations at the Saldanha Logistics Hub.

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APPENDIX I Literature search on pollutants of Concern ($PM_{2.5}$, Manganese, Lead and Silica)

Background on Particulate Matter (PM)

Particulate matter (PM) concentrations are often used as a proxy for air pollution, as it is considered as the air pollutant causing the most adverse human health effects (EEA, 2024). PM is defined by the World Health Organization (WHO) (2021) as: "A mixture of solid and liquid particles in the air, that are small enough not to settle out on the earth's surface under the influence of gravity, classified by aerodynamic diameter". Therefore, $PM_{2.5,}$ is defined as "particulate matter, where the particles have an aerodynamic diameter equal to or less than 2.5 µm" (WHO, 2021).

Particulate matter is emitted from natural as well as anthropogenic (man-made) sources. Examples of natural sources include wind-blown dust and pollen, veld fires and volcanos, whereas anthropogenic sources include emissions from combustion processes (motor vehicles, industries, coal-fired power stations, domestic fuel use), construction, mining and others. Particulate matter may be emitted directly from a source (primary PM), or may form in the atmosphere through fate and transport (secondary PM). For example, sulphate particulates may form from sulphur dioxide gas. Particulate matter from combustion processes and secondary particulates are normally fine (in the $PM_{2.5}$ range) while larger particulates may form from mechanical processes and contain material from the earth's crust as well as wind-blown dust and fugitive dust from roads and industries (WHO, 2000). Particles between 0.2 and 2.5 µm in diameter mostly comprise secondary sulphate ion, secondary nitrate ion, secondary ammonia ion, carbonaceous and crustal material (IARC, 2016

According to the WHO, "air pollution is the single biggest environmental threat to human health, based on its notable contribution to disease burden, and it is particularly true for particulate matter" (WHO, 2021). In general, the biggest sources of air pollution are of anthropogenic origin. Of these, combustion processes and especially fossil fuel burning, are the most important (WHO, 2021). In South Africa, there are not only outdoor sources of combustion such as in transport and power generation, but also indoor sources where many households use fuel other than electricity, for cooking and heating, especially during winter months. Indoor air pollution, including PM may sometimes be higher indoors than outdoors, due to sources being indoors. These sources may be domestic fuel use, tobacco smoking and use of cleaning and other consumer products (WHO, 2021).

Although it is known that the chemical composition of PM may have an influence on the health effects caused, and that PM from different sources may consist of different components, PM is still classified based on the aerodynamic diameter of the particles (WHO, 2021). The main reason is because the WHO is of the opinion that there are insufficient data available to base guidelines for different

components of PM on (WHO, 2021). It is believed that the smaller a particle is, the deeper it may enter the respiratory system, and therefore may potentially be more of a risk to human health. Aerodynamic diameter is thus used to classify PM.

Health effects of $PM_{2.5}$

Studies on air pollution and health have been conducted in most of the WHO regions, although evidence-based recommendations mostly come from studies in high income countries such as North America and Europe. However, more and more studies now emerge from Asia and Oceania, especially long-term studies from China. Although these studies also found associations between air pollutants and diseases, their concentration-response graphs sometimes differ, in that a linear relationship is only observed at lower concentrations (WHO, 2021).

In 2005, the annual guideline for $PM_{2.5}$ was based on only two studies (the Harvard Six Cities study and the American Cancer Society Cancer Prevention Study II), where exposure was assessed from a few monitoring sites per city (WHO, 2021). Lately satellite observations, chemical transport models and land-use regression models are being used in addition to monitoring, to obtain fine temporal and spatial scales (WHO, 2021).

In general, evidence of an association between exposure to $PM_{2.5}$ and adverse health effects, exists for all-cause mortality, acute lower respiratory infections, chronic obstructive pulmonary disease, ischemic heart disease, lung cancer and stroke (WHO, 2021). Studies also suggest an association with type 2 diabetes, impacts on unborn babies, Alzheimer's disease and other neurological diseases (WHO, 2021). Lately health conditions that were not previously considered, now also seem to be associated with PM, for example asthma, diabetes, reproductive and neurocognitive outcomes (WHO, 2021).

$PM_{2.5}$ and cancer

In 2016, the International Agency for Research on Cancer (IARC, 2016), declared particulate matter in outdoor air pollution as a confirmed human carcinogen.

Forastiere et al (2024) considered 75 systematic review and meta-analyses articles on $PM_{2.5}$, to find a concentration-response-function for lung cancer in adults above 30 years of age. They established an increase in Relative Risk of 1.16 for every 10 $\mu g/m^3$ increase in long-term $PM_{2.5}$. The uncertainty in this concentration response function was considered to be low.

Pritchett et. al. (2022), reviewed epidemiological (cohort or case-control) studies published between 1980 and 2021, to determine the status of knowledge on the association between PM and primary gastro-intestinal (GI) cancers. They demonstrated an increased Relative Risk (RR) from exposure to $PM_{2.5}$ for colorectal cancer and liver cancer (Pritchett et. al., 2022).

$PM_{2.5}$ and poor cognitive function and dementia

Cheng et. al. (2022), wrote a review paper on the association between long-term exposure to $PM_{2.5}$ and dementia. They did a literature search to find original cohort studies published between 1900 and 2022, involving adults exposed to $PM_{2.5}$ for longer than a year. Their meta-analysis showed a significant association between long-term exposure to $PM_{2.5}$ and dementia.

Wang et al. (2020), identified a gap in the knowledge of the association between $PM_{2.5}$ and cognitive function in developing countries with relatively high concentrations of $PM_{2.5}$. They followed 13,324 older Chinese adults with normal cognitive function, from 2002 to 2014. At baseline their ages ranged from 65 to >100 years, but they all had normal cognitive function. $PM_{2.5,}$ concentrations were determined via satellite for the areas these individuals resided at baseline. The authors found $PM_{2.5,}$ to be a risk factor for poor cognitive function, although only in the age groups 65 to 79 and >100, but not in the age groups 80 to 89, and 90 to 99 (Wang et. al., 2020). Confounding factors such as age, sex, place of residence (urban or rural), life style and underlying diseases such as high blood pressure and diabetes were taken into account (Wang et al. 2020).

$PM_{2.5}$ and mortality

Many studies have found associations between $PM_{2.5}$ and mortality. What is less known, is whether associations are greater for certain types of mortality than for others. Liu et al (2023a) conducted a time-stratified case-crossover study in six provinces in China during 2013–2018, involving nearly 5.45 million mortality cases. Short-term exposure to $PM_{2.5}$ was determined for these cases, using monitored and modelled data. Due to the fact that such a large dataset was available, concentration-response functions could be determined for all the different causes of death that had more than 1000 cases. They identified 33 causes of death that were significantly associated with $PM_{2.5}$. Ninety four percent of deaths were due to one of three causes, namely circulatory diseases (51.0 %), neoplasms (abnormal growth of tissue) (26.5 %), and respiratory diseases (16.4 %) (Liu et al 2023a).

Studies on the association of mortality and PM were mostly done in countries with relatively low PM concentrations. One such study with high statistical power due to the large study population, was the

study by Di et. al. (2017) that was also highlighted in the 2021 WHO Air Quality Guidelines. This study was based on a cohort of nearly 61 million people from nearly 40 000 different areas in the US (including small cities and rural areas), followed for up to 13 years (median 7 years). During this time there were 22.6 million deaths, of which nearly 12 million were while the $PM_{2.5}$ concentration was below 12 μg/m^3 (Di et. al., 2017). The annual average during the whole study, ranged between 6.2 and 15.6 μg/m^3 only (Di et. al., 2017).

Di et. al. (2017) found the risk of mortality in the study, due to exposure to $PM_{2.5,}$ to be 7.3%, but when they restricted the analyses to $PM_{2.5}$ concentrations below 12 μg/m^3, the risk of death was 13.6%. This showed that the slope of the exposure-response graph is steeper at concentrations below 12 μg/m^3 (Di et. al., 2017). They also did not find a threshold limit value, not even at 5 μg/m^3 (Di et. al., 2017).

Another study, a meta-analysis by Chen and Hoek (2020), was also used by the WHO in their revision of the Air Pollution Guidelines (WHO, 2021). In this study, about 3000 abstracts of articles were screened and eventually 107 studies chosen, to be included in a meta-analyses. Outcomes included, were death from: ischaemic heart disease, stroke, chronic obstructive pulmonary disease (COPD), acute lower respiratory infection (ALRI), lung cancer and death from a natural cause (Chen and Hoek, 2020). It was found that the association between $PM_{2.5}$ and death from these causes, was stronger than the association with PM_{10} (Chen and Hoek, 2020).

In general, the meta-analysis of the short-term exposure studies demonstrated linear concentration-response graphs for PM_{10} and $PM_{2.5}$ (Orellano et al, 2020). The authors also considered the relative risk for every 10 μg/m^3 increase in short-term $PM_{2.5}$ exposure. A relative risk of 1.0065 (CI:1.0044 – 1.0086) was shown for all-cause mortality, 1.0072 (CI: 1.0012 – 1.0132) and 1.0073 (CI 1.0029 – 1.0116) for cerebrovascular mortality (stroke) and respiratory mortality respectively, while the relative risk for cardiovascular mortality was 1.0092 (CI: 1.0061 – 1.0123) (Orellano et al, 2020). The associations between the RRs found for $PM_{2.5}$ and all-cause and cause-specific mortality, were linear (Orellano et al, 2020).

A gap was identified in the knowledge on mortality in areas with relatively high PM concentrations (Brown et. al., 2022). In India, for example, the average $PM_{2.5}$ concentration in 2014 was 47 μg/m³ compared to the 10 μg/m³ recommended by the WHO (in 2000) as a guideline at the time (Brown et. al., 2022). A study by Brown et. al., (2022) conducted on Indian mortality data (212,573 deaths among people 15–69 years of age) and satellite-based PM concentration data, showed a Relative Risk (RR) of

1.09 for deaths due to stroke, for every 10 μg/m^3 increase in the concentration of $PM_{2.5.}$ The same study found no significant association between $PM_{2.5}$ and total mortality when stroke is excluded. Also, no significant association with all-cause mortality or mortality from chronic respiratory disease or ischemic heart disease (Brown et. al., 2022).

Mortality and components of $PM_{2.5}$

It is important to note that PM may consist of many different components, depending on their original source, and that these different components may have different health effects (Li et al, 2019; Chen et. al., 2021). A study by Thurston et al. (2016) involving more than 4 million adults in more than 100 metropolitan areas of the United States, indicated that the risk of ischaemic heart disease mortality associated with $PM_{2.5}$ differ according to components and source. For example, the risk was five times higher for $PM_{2.5}$ from coal combustion, than for the same mass in general ambient air. Similarly, diesel traffic-related elemental carbon soot produced a risk of ischemic heart disease, but $PM_{2.5}$ from wind-blown soil and biomass combustion was not associated with a risk of ischemic heart disease mortality (Thurston et al., 2016).

A few other studies have also demonstrated an association between $PM_{2.5}$ and effects on gut microbiota, although one study could not find an association, possibly as a result of a small study population (101 participants) (Li et. al., 2023).

$PM_{2.5}$ and life expectancy

It is believed that if the global $PM_{2.5}$ concentrations could be reduced to the World Health Organization guidelines, the average human being would live on average 2.3 years longer (Greenstone & Hasenkopf, 2023).

Asia and Africa contribute 92.7% to years of life lost as a result of pollution, yet only 35.6% of countries in Asia and 4.9% of countries in Africa have air quality standards (Greenstone & Hasenkopf, 2023). Globally $PM_{2.5}$ is believed to be a bigger risk to years of life lost than alcohol abuse, tobacco use, transport injuries, sexually transmitted illnesses including AIDS, or neglected tropical diseases, including malaria (Greenstone & Hasenkopf, 2023). This is despite the fact that China has reduced its air pollution levels by 42.3% between 2013 and 2021 (Greenstone & Hasenkopf, 2023). Air pollution in South Asia, especially in India, Pakistan and Bangladesh, has however increased and it is believed that these countries are, as a result, close to reducing their life expectancy by 5.1 years (Greenstone & Hasenkopf, 2023).

$PM_{2.5}$ and respiratory disease

Hu et al (2023) investigated the lower respiratory infection burden attributable to $PM_{2.5,}$ using data from the 2019 Global Burden of Disease Study. The authors showed an overall decrease in lower respiratory infections attributable to $PM_{2.5,}$ yet $PM_{2.5}$ still contributed about 0,7 million deaths (from lower respiratory infection) in 2019. They also found developed countries had a higher burden from $PM_{2.5}$ in ambient air and developing countries a higher burden from $PM_{2.5}$ in household air pollution (Hu et. al., 2023).

When only days with $PM_{2.5}$ concentrations below 15 µg/m^3 (the WHO guideline at the time) were considered, association between $PM_{2.5}$ and respiratory disease remain, but there was no significance association with cardiovascular disease. The same happened when associations were adjusted for NO_2, indicating that the observed association between $PM_{2.5}$ and cardiovascular admissions may be as a result of simultaneous exposure to other pollutants (Hasegawa et. al. 2023).

Forastiere et al (2024) considered 75 systematic review and meta-analyses articles on long-term $PM_{2.5}$, and established an increase in Relative Risk of 1.34 for every 10 µg/m^3 increase in $PM_{2.5}$ for asthma in children. The uncertainty in this concentration response function was low.

$PM_{2.5}$ and cardio-vascular disease (CVD)

Liu et al. (2023) identified a gap in the knowledge of the global burden of CVD attributable to $PM_{2.5}$ in ambient air. They then conducted a study where they estimated the spatial and temporal trends in disability adjusted life years (DALYs) as well as mortality due to CVD from exposure to ambient $PM_{2.5}$ (Liu et.al., 2023). One DALY can be explained as the equivalent of one year of life lost due to pre-mature death or living with a disability or disease.

Available data for CVD, consisted of data on ischemic heart disease (IHD) (chronic stable angina, chronic IHD, acute myocardial infarction, and IHD associated heart failure) and stroke. This was done at global, regional and national levels for the period 1990 to 2019, using data from the Global Burden of Disease Study of 2019. They found the global number of CVD deaths due to ambient $PM_{2.5}$ exposure, to be about 2.48 million, which represents 13.4% of the global total CVD-related deaths. As far as the DALYs were concerned, results showed 60.9 million (15.5%) DALYs of CVD were attributable to ambient $PM_{2.5}$ in 2019 (Liu et.al., 2023).

Forastiere et al (2024) considered 75 systematic review and meta-analyses articles on $PM_{2.5}$, to find concentration-response-functions for ischemic heart disease, stroke and hypertension in adults above 30 years of age. They established an increase in Relative Risk for every 10 $\mu g/m^3$ increase in long-term $PM_{2.5}$ of 1.13, 1.16 and 1.17 for ischemic heart disease, stroke and hypertension respectively. The uncertainty in these concentration response functions was considered to be low.

$PM_{2.5}$ and complications during pregnancy and adverse birth outcomes

The fetus is in a stage of physiological development and is therefore very susceptible to exposure of any substance that may cause adverse health effects, such as death, injury or disease.

Li et. al. (2019), assessed many case-control studies, cohort studies and meta-analyses studies conducted in countries such as Canada, the USA and China, to determine the impact that maternal exposure to $PM_{2.5}$ may have on birth outcomes. Results from these different studies indicate that exposure to $PM_{2.5}$ during any stage of pregnancy may lead to adverse birth outcomes.

Low Birth Weight

A birth weight of 2499 g or less, is generally considered as low birth weight (Li et. al., 2019). In a meta-analysis of many studies in different countries, Li et. al. (2019) showed an association between exposure to $PM_{2.5}$ during the entire pregnancy, and having a baby with low birth weight.

Still birth

The birth of a baby who died in the womb at 28 weeks or later, is considered a still birth (UNICEF, 2023). In 2015 the global still birth rate was 18.4 per 1000 total births (Li et. al., 2019). This rate came down to 13.9 stillbirths per 1,000 births globally in 2021 (UNICEF, 2023). The still birth rate differs between countries, with low- and middle-income countries having higher rates. South Africa, for example, had a rate of 32.8 per 1,000 total births in 2021, while the rate in the United Kingdom was 4.2 during the same assessment (UNICEF, 2023). There are many possible causes for this phenomenon, including exposure to $PM_{2.5}$ during the third trimester, as was found in at least three studies (Li et al, 2019).

MANGANESE (CDC 2012)

Manganese is naturally occurring in the earth's crust and is a trace element necessary for good health. Manganese is used in steel production to improve hardness and is also a fuel additive (MMT) and a fertiliser and even as a pigment in cosmetics. Manganese is released to air through human activities such as mining and also from industries and vehicle exhaust fumes.

Manganese is essential in the human body as a cofactor for a number of enzymes. Manganese may however, accumulate in lower organisms of the food chain. The highest concentrations of manganese are in grains, nuts, legumes and fruit.

The absorption of manganese through inhalation depends on the size of the particles. Some particles may be absorbed into the blood from the lungs, while nanoparticles may be small enough to be directly transported to the brain via the olfactory nerves. Only about 3-5% of ingested manganese is absorbed. Once absorbed, the manganese is distributed throughout the body with most of the manganese found in liver, kidneys and pancreas.

The inhalation and ingestion of a large quantity of dust or fumes, as in an occupational environment, may cause inflammation in the lungs and reduce lung function, and may affect the nervous system. Manganese in the brain may cause manganism, a neurologic disease that cannot be reversed, the symptoms of which includes tremours, a mask-like face and spasms of the facial muscles, speech disturbance and a difficulty in walking.

Children experience the same health effects from manganese than adults including the neurodevelopmental effects (when exposed to very high concentrations of manganese) which may cause changes in their behaviour, memory and ability to learn.

Manganese is not considered as a confirmed human carcinogen by the International Agency for Research on Cancer (IARC) (2021), and therefore not treated as a carcinogen in this assessment.

LEAD (CDC, 2020)

Although not a very abundant element, lead is occurring naturally in the earth's crust and is widely spread throughout the world. The properties of lead such as being resistant to corrosion, makes it useful in many products such as pipes, from where it may leach into water. Lead is also used as a pigment in paint. Some traditional medicines, hair dyes and imported cosmetics may also contain lead.

Inhalation of lead in air mostly happens in an occupational environment, whereas the general public may be exposed to lead through ingestion of contaminated food and water and inhalation when using lead in hobbies such as soldering or making stained glass etc. Children may especially be exposed to lead through ingestion from hand-mouth-contact with contaminated soil, or if they ingest flacking lead-based paint.

Following inhalation of organic lead, approximately 60–80% of what is deposited in the airways is absorbed, while about 40 to 50% of ingested lead is absorbed in children and only about 3 to 10% in adults. Less lead is absorbed from soil. When an individual's stomach is empty, more lead will be absorbed. Individuals with low levels of calcium and iron will absorb more lead.

In adults 94% of the lead in their bodies is stored in the bones and teeth while in children this is about 73%. Lead may remain in blood for months, but in bone for decades. The lead in blood thus reflects recent exposure and the lead in bone cumulative exposure.

Lead has several adverse health effects including effects on the kidneys, on the blood, it may increase blood pressure, cause colic in children, neurological effects (neuropsychological in adults and affecting the intellect of children). Lead may also reduce fertility.

Children are especially susceptible to the effects of lead as they absorb more lead than adults and the main concern is impairment of neurological development on children.

Lead is not considered as a confirmed human carcinogen by the International Agency for Research on Cancer (IARC) (2021), and therefore not treated as a carcinogen in this assessment

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SILICA

Silica is part of the crust of the earth and is therefore present everywhere in the environment, and in different forms, but mainly in the form of quartz (crystalline silica) (CDC, 2019). Exposure to silica can therefore not be avoided, as it may be present in air, soil, food and water as well as consumer products. Silica may be present in food, such as rice and sugar cane, because plants use silica to strengthen leaves and stems and build protective spikes. (CDC, 2019).

From the studies investigated, it is evident that adverse effects were associated with particulates in the respirable size range and at relatively high concentrations where workers were exposed for long periods of time. Adverse health effects were not reported from inhalation of large particles or at low levels, or from incidental exposure in the ambient environment (CDC, 2019). The main health effect from inhalation, is silicosis, a progressive, irreversible, fibrotic lung disease (lung fibrosis). Connective tissue forms as part of normal healing processes in the body, but in lung fibrosis, excess connective tissue deposition occurs.

Many studies on silica exposure have been evaluated to determine if an association between silica exposure and lung cancer does exist (CDC, 2019). As the prevalence of lung cancer amongst workers exposed to silica was relatively low (much lower than for example the association with asbestos), meta-analyses of pooled data from occupational studies were necessary to get large enough study populations to demonstrate an association (CDC, 2019). The association found with lung cancer, showed a dependence on cumulative exposure (CDC, 2019). The International Agency for Research on Cancer (IARC) classified crystalline silica inhaled as quartz from occupational sources, as a known human carcinogen (IARC, 2021).

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APPENDIX II Qualitative Risk Assessment of Transport of Commodities

Background

Commodities will be transported to the Logistics Hub via road and rail. The manganese will be transported via rail from the Northern Cape and the rest will be transported from other areas via road. The trucks will be covered during transport, and it is thus assumed that fugitive dust from these trucks will be minimal. AMSA has however, no control over the way in which the manganese ore will be handled during loading and transport by rail. The areas under consideration are as in Figures 2 and 3 below.

In total, five million tons of commodities will be transported to the Logistics Hub per year. It is expected that there will be 11 trucks per day carrying 34 pay-load tons of commodities. At inception, 2 Mtpa of manganese will be transported to site which is to increase as maintenance on the rail way line should be complete by 2026. Therefore, approximately 5 479 tons of manganese will be transported to site daily. It is uncertain how many train wagons carrying manganese will be used daily as this may vary.

Fugitive dust

Information on fugitive emissions from trains carrying manganese ore could not be found in the literature searched. It is therefore not known how much dust will be emitted by these trains, how the dust may be dispersed, how far it will travel from the rail lines and what concentrations the public may be exposed to. The lack of information limits the ability of this assessment to quantify the potential risk to the public along the rail line from inhaling manganese dust. The risk is therefore qualitatively assessed.

How may dust be emitted from train wagons

Fugitive dust may be emitted from the train wagons through doors that do not close properly, or from the wind blowing over the open loaded wagons.

Factors influencing emissions of fugitive dust.

Factors that may influence the amount of fugitive dust emitted from train wagons carrying manganese ore, will include i) physical properties of the ore, such as the size ii) the moisture content iii) how fast the train is travelling iv) the distance the train is travelling v) the weather conditions, such as wind speed and precipitation.

Economic impact

To lose part of the load through fugitive dust, will have an economic impact. It is thus in the interest of AMSA that mitigation measures are put in place. In the case of coal, literature suggests up to 3% of the total transported load may be lost through fugitive dust.

Literature search

The focus in the available literature on emissions from transport of commodities by train, is on coal transport. In a publication by Multnomah County Health Department (MCHD) (2013) in the United States, it was estimated that the amount of "coal dust shed by trains during shipping, vary from less than one percent to up to three percent of the load". It was further mentioned that coal dust emitted from loaded train wagons may travel approximately 500 m to 2 km from the train tracks, depending on weather conditions and train speed. Being a heavy metal, these figures may not be the same for manganese dust, as it may not travel that far.

Ostro et al (2023) reported evidence of significant increases in $PM_{2.5}$ from passing coal trains in California in the US. During a six-month observation period, they measured increases in ambient $PM_{2.5}$ during the passage of four different types of trains, namely full coal trains, empty coal trains, freight trains and passenger trains. The monitoring station was approximately 21.5 meters east (generally downwind) of the rail line.

"The average (5-minute) change from passing coal trains added approximately 8.3 $\mu g/m^3$ (95% CI = 6.4, 10.3) to the ambient $PM_{2.5}$, with midpoint estimates ranging from 5 to 12 $\mu g/m^3$. Full coal cars contributed approximately 2 to 3 $\mu g/m^3$ of $PM_{2.5}$ more than freight trains. With calm winds, the nearby concentrations from coal trains were about 12 $\mu g/m^3$ versus 5 $\mu g/m^3$ for freight trains. Considering only winds from the west resulted in an increase of 25 $\mu g/m^3$ from coal trains. The peak concentration (10 second average) from coal trains was 17.4 $\mu g/m^3$, about 3.5 $\mu g/m^3$ more than freight trains. Calm wind conditions resulted in an increase of 20 $\mu g/m^3$" Ostro et al (2023).

"The results indicated an increment in maximum $PM_{2.5}$ over the control period of 22.9 $\mu g/m^3$ (95% CI = 8.1, 37.5); $p < .01$) for full coal trains. For the model calibrated and corrected for humidity, the increment from coal cars was 17 $\mu g/m^3$ (95% CI = 6.2, 28.5; $p < 0.01$), while the corresponding change in $PM_{2.5}$ was 14.1 $\mu g/m^3$ (95% CI = 7.9, 20.2; $p < 0.01$) for freight trains and 9.3 $\mu g/m^3$ (95% CI = -3.0, 21.5, NS) for empty coal cars". Under calm wind conditions, the impact from coal trains increased to almost 20 $\mu g/m^3$ (95% CI = 3.4, 36.6; $p < 0.05$), while the freight increment did not change from the previous case" Ostro et al (2023).

Ferreira and Vas (2004) conducted a study in a wind tunnel on fugitive dust emissions from train wagons. They found the loss in coal dust ranged between 80 g and 1.2 kg of coal per km from uncovered wagons, depending on type of coal, the moisture content of the coal, the use of surfactant coatings, the wind speed and the speed at which the train was travelling. Most fugitive coal dust losses were due to spillage and wind erosion off the top surface in the wagons. Covered wagons lost less than 20% of the quantity that was lost from the uncovered wagons (Ferreira and Vaz 2004).

Human Health Effects

Since the trucks will be covered, fugitive dust from road transport should be minimal. However, fugitive dust may be emitted from trains transporting manganese ore to the Logistics Hub. The human health impacts of manganese (discussed in Appendix I) are based on relatively high concentrations as found in occupational environments. For manganese, this concentration is above 30 $\mu g/m^3$ as no health effects could be found at an exposure of 30 $\mu g/m^3$. The lowest concentration of manganese at which health effects were found, was 50 $\mu g/m^3$. No information could be found on exposure of the public to fugitive manganese dust from passing trains.

Recommendations

It is recommended that once the Logistics Hub is operational, manganese be monitored for a period of time (preferably one year to cover all seasons) in communities at risk.

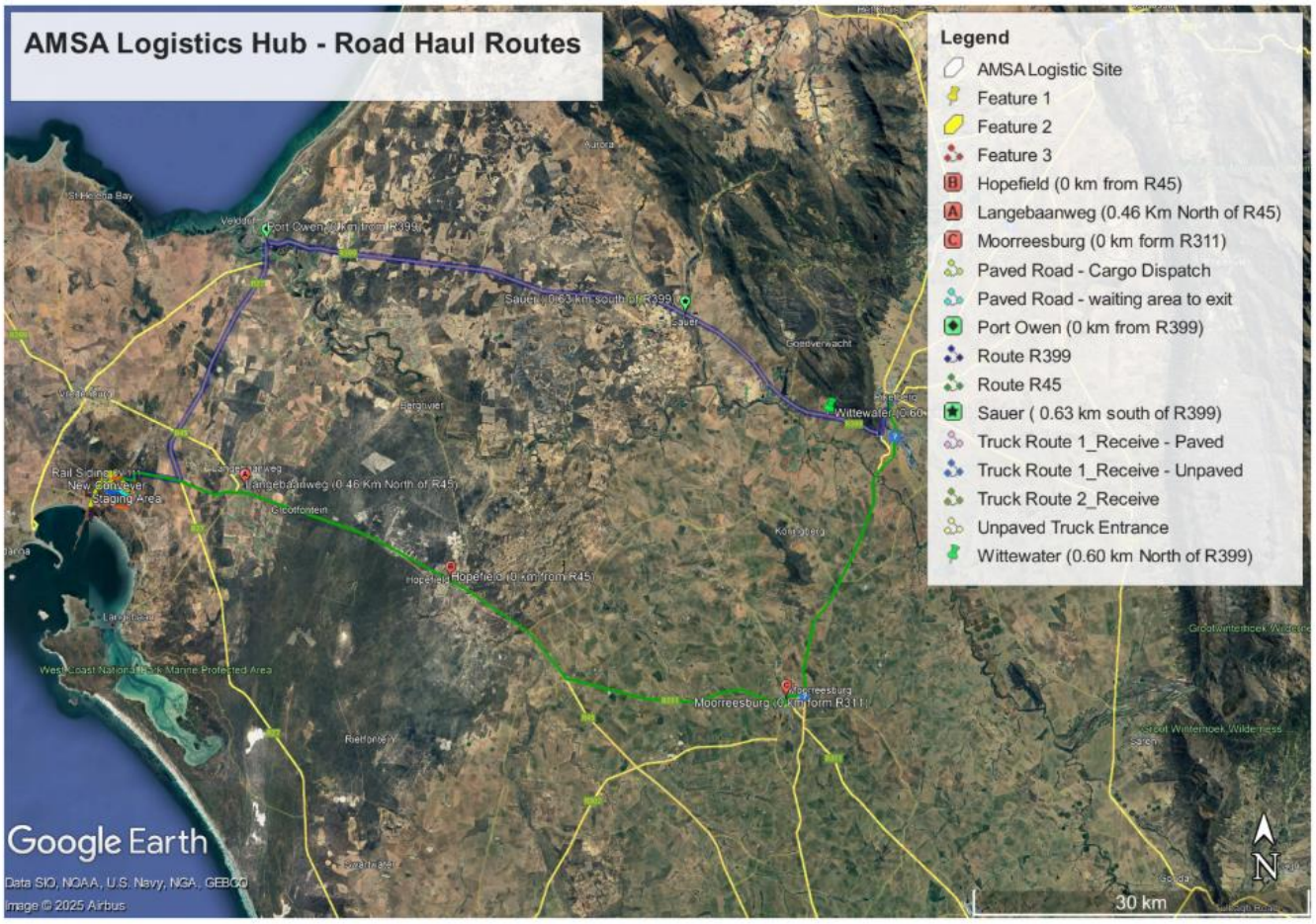

Figure B2. Areas affected by the Road Haul Roads to and from the Logistics Hub.

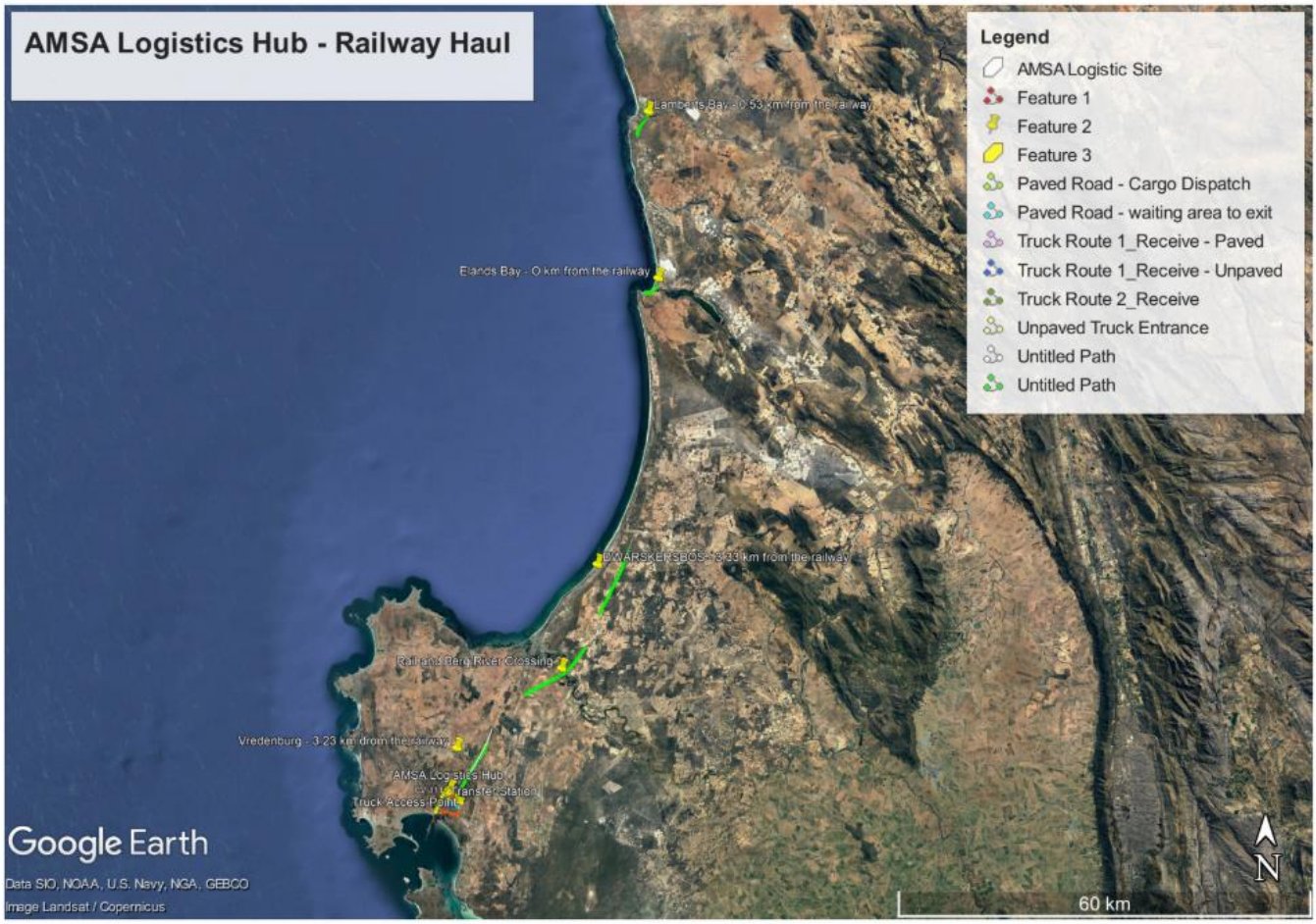

Figure B2. Areas affected by the rail route to and from the Logistics Hub.

References

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Ostro B, Spada N, Kuiper H. 2023. Assessment of Coal and Petroleum Coke Pollution. Air Quality, Health, and Environmental Justice. Environmental Justice. California Air Resources Board. [Online] Available at: https://ucdavis.app.box.com/s/sh55sgeix0r39k07zfsai1tcamux8qpw. (accessed 29 March 2025)