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Attention: James McDonough

Provision of independent air quality health advice – diesel particulate emissions

1.0 Introduction

Environmental Risk Sciences Pty Ltd (enRiskS) has been commissioned by the Department of Planning and Environment (DPE) to undertake an independent review and provide advice in relation to the potential for adverse health effects in relation to exposure to diesel combustion emissions associated with a proposed quarry in the Hunter Valley in NSW.

The project is the Deep Creek Quarry, located at 279 Deep Creek Road in Limeburners Creek, NSW 2324. The project is a State Significant Development (SSD) for which an Environmental Impact Statement (EIS) has been prepared by Kleinfelder (EIS dated 2021). The EIS has been on public exhibition and a number of submissions were received. One submission specifically relates to health concerns for one resident who has been diagnosed with hypersensitivity to diesel particulate.

The EIS included an Air Quality Impact Assessment (AQIA, Appendix H to the EIS), completed by Todoroski Air Sciences. The AQIA included the modelling of diesel emissions from the mine, and predicted impacts at the location of the resident.

2.0 Objectives and scope of works

The overall objective of this assessment is to review all available information and provide advice in relation to the potential for adverse health effects from diesel emissions from the proposed project, specific to this situation.

More specifically, this assessment has involved the following:

- review the EIS in relation to understanding how diesel emissions have been assessed in the AQIA, and how the available data characterizes exposure to diesel emissions in the offsite environment, specifically the residential property where concerns have been raised
- review the available information provided in the submission relating to the health concerns raised
- review the current literature in relation to hypersensitivity to diesel particulates and whether current methods used to assess the hazards of exposure adequately address hypersensitivity aspects
- based on the above, provide advice in relation to this issue, and the potential for diesel emissions from the proposed project to adversely impact on the health of the resident.

This assessment has only addressed the concerns raised in one submission, specific to hypersensitivity to diesel particulates. No other submissions related to the project have been considered.

3.0 Methodology

The methodology adopted for the conduct of this assessment is in accordance with the relevant National protocols/ guidelines including:

- enHealth (2012a) Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards
- enHealth (2012b) Australian Exposure Factor Guide

Where required, additional guidance has been obtained from relevant Australian and International guidance consistent with current industry best practice, such as that available from the USEPA and the World Health Organization (WHO). These guidance documents are referenced, where utilised, in this assessment.

4.0 Review of assessment of diesel particulate exposures in EIS

4.1 Proposed development

The issues of concern being evaluated relate to the emissions to air from a specific project, the proposed hard rock quarry referred to as Deep Creek Quarry located on the boundary of the localities of Limeburners Creek and Allworth, New South Wales.

The application evaluated in the EIS seeks approval to produce up to 500,000 tonnes per annum (tpa) of hard rock quarry products for a period of up to 30 years, with the primary target being rhyolite that has the unique properties required to meet high specification road building standards throughout New South Wales.

The quarry covers an area of 30 ha, with the extraction area of approximately 18 ha and approximately 12 ha for infrastructure and ancillary facilities to support the operations.

The infrastructure associated with the project will include a new sealed access road and intersection with The Bucketts Way, site office, workshop, weighbridge and stockpile area with associated water management infrastructure.

4.2 Air quality impact assessment

Appendix H of the EIS presents the Air quality impact assessment (AQIA) conducted by Todoroski Air Sciences (report dated 24 September 2021). The AQIA evaluated the project as proposed, and included an assessment of all activities proposed, including road traffic.

The AQIA evaluated impacts in the offsite community, including at a number of specific sensitive receptors surrounding the site. The concerns being evaluated in this review relate to receptor [redacted] located to the east of the site, noted to be adjacent to Bucketts Way and north of the proposed quarry access road. The location of the site and all sensitive receptors, including [redacted] are shown on **Figure 1**.

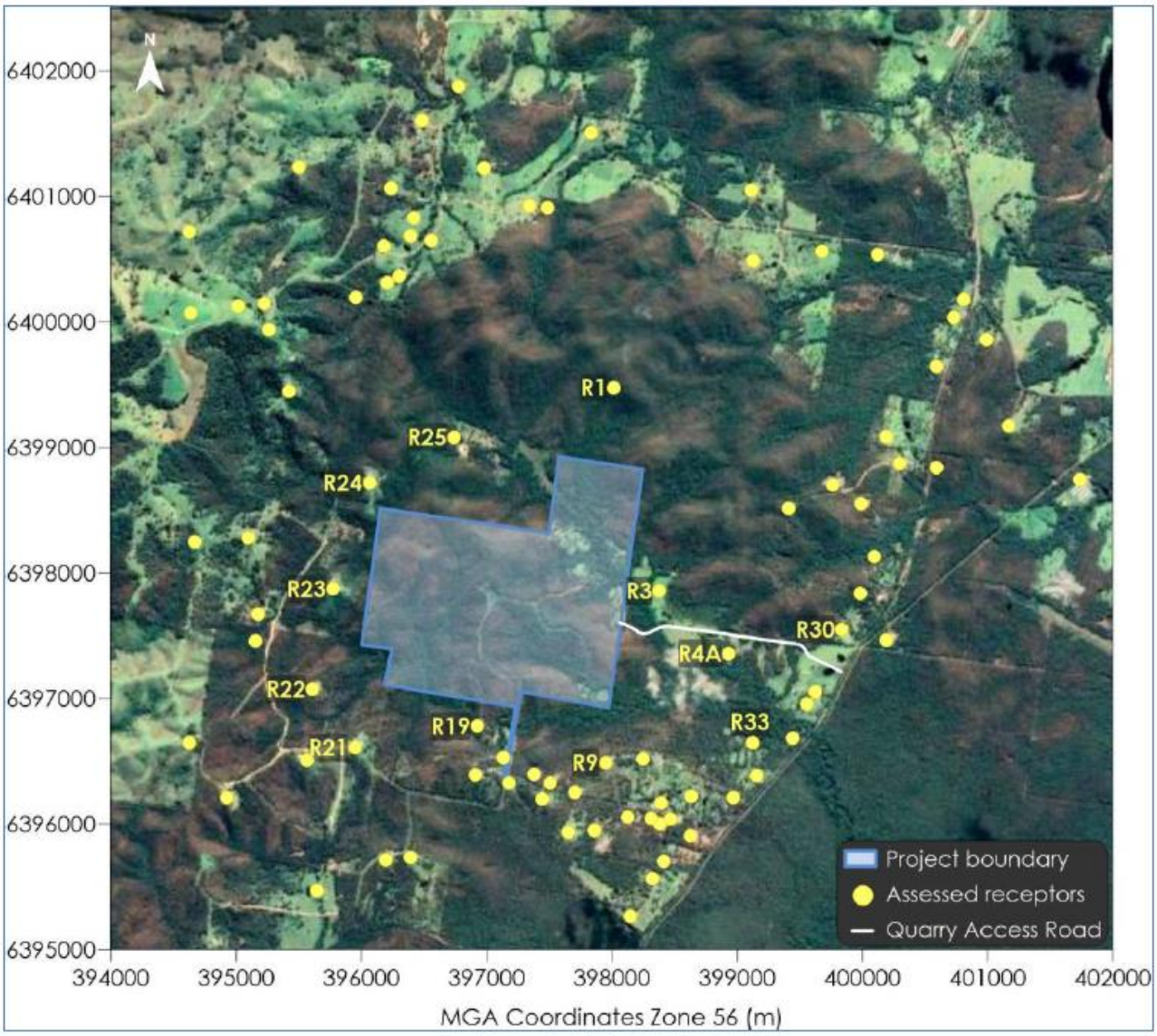

Figure 1: Site location and surrounding sensitive receptors evaluated in the AQIA (receptor of concern to this assessment is [redacted]) (source: AQIA)

The AQIA addressed impacts on the surrounding community in relation to emissions to air of particulate matter, odours and nitrogen dioxide, from project related activities.

Most of the dust emissions from the proposed project relate to quarrying activities (drilling, blasting, loading and unloading of material, vehicles travelling on and off the site, crushing and screening processes and windblown dust from exposed areas and stockpiles.

However, the assessment has included on-site and off-site vehicle and plant equipment that have the potential to generate particulate emissions from diesel exhaust. This has included vehicles travelling along The Bucketts Way and the Quarry Access Road, which includes existing vehicles using The Bucketts Way and vehicles associated with the proposed Hillview Hard Rock Quarry located approximately 6.9 km to the north of the Deep Creek Quarry project. The emissions modelled from these sources included particulate matter as PM_{10} (fine particulate fraction). It was then assumed that $PM_{2.5}$ (more fine particulate fraction, more directly relevant to health) would comprise 92% of PM_{10} following USEPA guidance, as particulate

matter from combustion sources such as diesel equipment and vehicles is very fine and mostly comprises $PM_{2.5}$.

In relation to $PM_{2.5}$ the AQIA determined the following, for receptor [redacted]

- The maximum change (incremental increase) in $PM_{2.5}$ concentration from all dust sources associated with the project (including vehicle traffic as noted above):
  - = 0.8 µg/m^3 over a 24-hour average
  - = <0.1 µg/m^3 over an annual average
- The maximum change (incremental increase) in $PM_{2.5}$ concentration from plant/equipment and all vehicle traffic associated with the project which is of most relevance for assessing diesel particulate exposures:
  - **= 0.06 µg/m^3 over a 24-hour average, which is 7.5% of $PM_{2.5}$ from all sources**
  - **= 0.02 µg/m^3 over an annual average, which is 20% of $PM_{2.5}$ from all sources**

In relation to PM_{10} the AQIA determined the following, for receptor [redacted]

- The maximum change (incremental increase) in PM_{10} concentration from all dust sources, which are dominated by emissions from the quarrying activities, not plant/equipment and vehicle emissions, associated with the project (including vehicle traffic as noted above):
  - = 5.4 µg/m^3 over a 24-hour average
  - = 0.4 µg/m^3 over an annual average
- The maximum change (incremental increase) in PM_{10} concentration from plant/equipment and all vehicle traffic associated with the project which is of most relevance for assessing diesel particulate exposures:
  - = 0.07 µg/m^3 over a 24-hour average, which is 1.3% of PM_{10} from all sources
  - = 0.022 µg/m^3 over an annual average, which is 5.5% of PM_{10} from all sources

The modelled PM_{10} concentrations from exhaust emissions are essentially the same as $PM_{2.5}$, consistent with the source being exhaust emissions from equipment and vehicles.

The concentrations of $PM_{2.5}$ derived from exhaust emission sources is of most relevance and importance for assessing potential exposures to diesel particulate matter. These concentrations are highlighted in blue above.

The AQIA also presents the cumulative impact of PM_{10} and $PM_{2.5}$ over an annual average. The cumulative impact is the incremental impact from all truck and traffic emissions plus the background (from ambient/other sources). The incremental impact from all truck and traffic emissions is a small fraction of the cumulative impact. Background concentrations of $PM_{2.5}$ relevant to the project are as follows:

- 24-hour average varies with each day and is assessed in the AQIA each day, at [redacted] the maximum background level of 25.9 µg/m^3 as $PM_{2.5}$ has been measured – the maximum incremental impact of 0.06 µg/m^3 at [redacted] is 0.2% of this background (i.e. a negligible contribution to total $PM_{2.5}$ exposures)
- Annual average background concentration of $PM_{2.5}$ is 7.3 µg/m^3 - the incremental impact of 0.02 µg/m^3 at [redacted] is 0.3% of this background (i.e. a negligible contribution to total $PM_{2.5}$ exposures).

5.0 Health concerns raised in submissions

The specific health concern raised in submissions to DPE relating to the project EIS indicated the following:

- the individual was diagnosed with hypersensitivity to diesel particulate in August 2018
- it is understood that the individual has a lung injury registered with the coal mines dust disease board
- during day-to-day activities the individual indicates that limited trips to town are undertaken as they feel unwell after, with exposure to an idling diesel making them feel immediately unwell
- they were informed that they have a high risk of developing cancer
- includes general information on diesel particulate matter, including that it is classed as a group 1 carcinogen (consistent with the information included in **Section 6**)
- the proposed haul road is located along the fence line with their property and the prevailing wind direction would result in emissions being directed onto the property. **Figure 2** was provided with the submission illustrating these aspects.

No additional information on the health issues experienced by the individual has been provided.

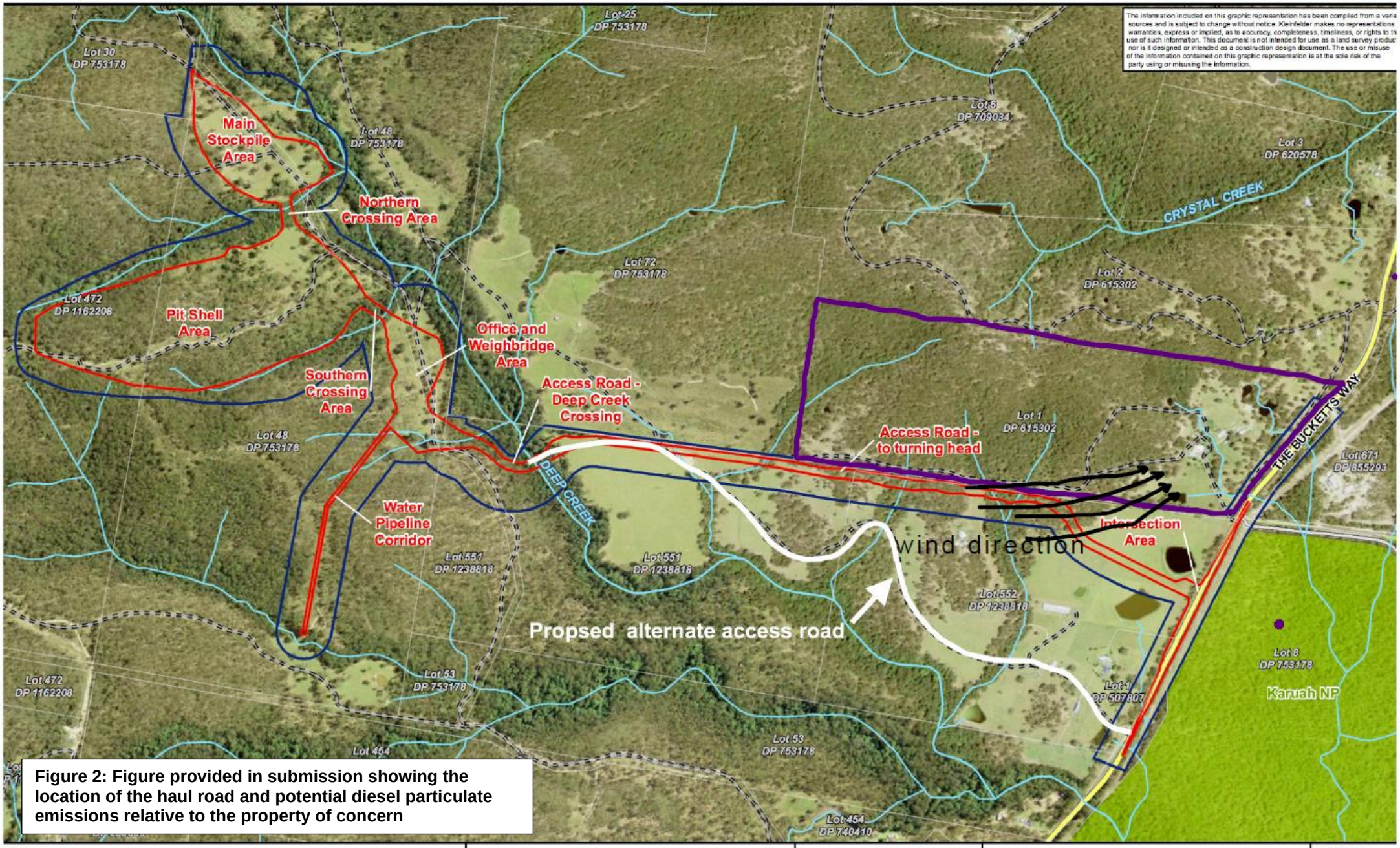

Figure 2: Figure provided in submission showing the location of the haul road and potential diesel particulate emissions relative to the property of concern

gend

- Development Footprint
- Study Area
- Lot Boundaries
- National Park
- Named Watercourse
- Unnamed Watercourse
- Arterial Road
- Local Road
- Track

Metres

0 50 100 200 300 400 500

N

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www.kleinfelder.com

PROJECT REFERENCE: 20203112
DATE DRAWN: 29/10/2020 13:15 Version 1
DRAWN BY: BDeane
DATA SOURCE: NSW DFSI - 2019

Study Area and Development Footprint

Ironstone Developments Pty Ltd
EPBC Referral - Deep Creek Quarry
Bucketts Way, Limeburners Creek NSW 2324

FIGURE: **2**

6.0 Diesel particulate matter

6.1 General

Diesel exhaust is emitted from plant and equipment and 'on-road' diesel engines (vehicle engines) and can be formed from the gaseous compounds emitted by diesel engines (secondary particulate matter). After emission from the exhaust pipe, diesel exhaust undergoes dilution and chemical and physical transformations in the atmosphere, as well as dispersion and transport in the atmosphere. The atmospheric lifetime for some compounds that are present in diesel exhaust ranges from hours to days.

Diesel engine exhaust is a complex mixture comprised of both gaseous and particulate components. The particulate phase contains organic compounds including PAHs and nitro-PAHs, as well as a range of trace metals (IARC 2013).

Data from the USEPA (USEPA 2002) indicates that diesel exhaust as measured as diesel particulate matter made up about six per cent of the total ambient/urban air $PM_{2.5}$. In this project, emissions to air from the project specifically relates to exhaust emissions from diesel plant/equipment and vehicles, with the assessment focusing on total fine particulates as $PM_{2.5}$. For the purpose of this review, it is assumed that 100% of $PM_{2.5}$ derived from these sources is diesel particulate matter.

The available evidence indicates that there are human health hazards associated with exposure to diesel particulate matter. The hazards include acute exposure-related symptoms, chronic exposure related non-cancer respiratory effects, and lung cancer.

In relation to non-carcinogenic effects, acute or short-term (e.g. episodic) exposure to diesel particulate matter can cause acute irritation (e.g. eye, throat, bronchial), neurophysiological symptoms (e.g. light-headedness, nausea), and respiratory symptoms (cough, phlegm). There also is evidence for an immunologic effect–exacerbation of allergenic responses to known allergens and asthma-like symptoms (USEPA 2002).

Chronic effects include respiratory effects. Review of these effects by the USEPA (USEPA 2002) identified a threshold concentration for the assessment of chronic non-carcinogenic effects. The review conducted by the USEPA also concluded that exposures to diesel particulate matter also consider $PM_{2.5}$ goals (as these also address the presence of diesel particulate matter in urban air environments). The review found that the diesel particulate matter chronic guideline would also be met if the $PM_{2.5}$ guideline was met.

The USEPA review (USEPA 2002) also identified that such exposures are '*likely to be carcinogenic to humans by inhalation*'. A more recent review by IARC (Attfield et al. 2012; IARC 2013; Silverman et al. 2012) classified diesel engine exhaust as carcinogenic to humans (Group 1) based on sufficient evidence that exposure is associated with an increased risk for lung cancer. In addition, outdoor air pollution and particulate matter (that includes diesel particulate matter) have been classified by IARC as carcinogenic to humans based on sufficient evidence of lung cancer. In relation to the mechanism by which cancer occurs following inhalation, the IARC evaluation (IARC 2013) considered the available data and determined that in animals diesel engine exhaust particles exhibit a variety of genotoxic effects *in vitro* and *in vivo*. Inhalation exposures in animals resulted in oxidative damage, adducts and strand breaks in lung DNA as well as heritable germ-cell mutations. In humans the data suggested a genotoxic mechanism. The USEPA review concluded the development of cancer is suggestive of both a mutagenic and non-mutagenic modes of action.

Based on the above review, and where methods outlined by enHealth (enHealth 2012) are followed, an assessment of the potential for adverse health impacts for all members of the community can be undertaken using:

- threshold guidelines to assess health effects other than cancer from acute and chronic exposures to diesel particulate matter – this includes effects such as irritation, respiratory effects, neurophysiological and immune effects
- non-threshold approach to assess carcinogenicity, as diesel particulate matter can be considered to be genotoxic.

This is further discussed in the following sections.

6.2 Further review in relation to hypersensitivity

6.2.1 General

Establishing guidelines that need to be protective of all adverse health effects (as discussed above) follows established methodologies that incorporate factors to ensure that susceptible populations are addressed. The USEPA defined a chronic inhalation guideline (termed reference concentration of RfC) as

> *"An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious noncancer effects during a lifetime."*

The same considerations are incorporated into the development of acute inhalation guidelines, where derived to be protective of the public.

When reviewing the potential for individual susceptibility to have been addressed, the following needs to be considered:

- the critical effect and mechanisms of action
- information available on susceptible populations and whether studies included in the derivation of the guidelines either included data from susceptible populations or accounted for these groups through selection of uncertainty factors

6.2.2 Critical effect

The critical effect, i.e. the most sensitive health effect that has been identified from all available studies which results in the most health protective guideline (i.e. other effects would occur at higher levels of exposure). For diesel particulate matter respiratory effects are the most sensitive and identified as the critical effect (USEPA 2002). Guidelines established to be protective of respiratory effects are considered to be protective of immunological effects, as the immunological effects are a response to the changes to the respiratory system.

The way in which adverse effects occur (i.e. mode of action) following inhalation of diesel particulate matter is also relevant (USEPA 2002):

- diesel particulate matter deposited in the upper airway would be cleared from the respiratory tract or translocated to other areas within the respiratory system
- smaller particulates would reach deeper into the lungs
- diesel particulate matter interacts with airway epithelial cells and phagocytosis by alveola macrophages (AMs)[1], which release chemotactic factors that attract neutrophils and additional AMs

[1] Alveolar macrophages (AMs) guard the alveolar space of the lung. Phagocytosis by AMs plays a critical role in the defense against invading pathogens, the removal of dead cells or foreign particles, and in the resolution of inflammatory responses and tissue remodeling, processes that are mediated by various surface receptors of the AMs.

- as the lung burden of diesel particulate matter increases there is a aggregation of the particle laden AMs in the alveoli adjacent to bronchioles resulting in the presence of particles in alveolar and peribronchial interstitial tissues and associated lymph nodes
- the macrophages may release cytokines, growth factors and protease that cause cell inflammation, cell injury, cell proliferation, hyperplasia and fibrosis, which is more important with the rate of deposition in the lung exceeds the rate of alveolar clearance. This can result in the generation of oxygen radicals (i.e. oxidative stress) and lung injury
- diesel particulate matter is poorly soluble so clearance via dissolution is insignificant
- the organic compounds absorbed on the surface can be desorbed and may result in metabolic reactions, and appears to be associated with the immunological effects observed
- the available evidence indicates that diesel particulate matter has the potential to produce pathological and immunological changes in the respiratory tract. Moreover, the magnitude of these responses is determined by the dose delivered to the respiratory tract and is attributable to both the carbon core and the adsorbed organic materials

The available information indicates the following in relation to acute exposures to diesel particulate matter (de Homdedeu et al. 2020; Ristovski et al. 2012; USEPA 2002):

- odour is not a good indicator of exposure due to individual variability in the ability to detect odours and the level at which it may be detected or be considered objectionable
- various biochemical and pathophysiological alterations, such as serum immunoglobulin E (IgE) changes, altered levels of cytokines/chemokines, and goblet-cell hyperplasia, with nearly all these responses being key changes and markers of allergic inflammatory disorders of the airways such as asthma and nasal allergies
- a major point of significance about these findings is that they indicate that diesel particulate matter could be viewed as having the potential to elicit inflammatory and immunological responses and responses typical of asthma, and that diesel particulate matter may be a likely factor in the increasing incidence of allergic hypersensitivity (by increasing epithelial permeability and protein allergenicity, acting as a carrier and/or adjuvant)
- these studies have also shown that effects are due primarily to the organic fraction and that DPM enhances the allergic response to known allergens
- results from these studies, including those with laboratory animals, indicate that DPM could be viewed as having the potential to influence the development of airway inflammation and disease through its adjuvant properties and by causing the release of proinflammatory mediators.

Less data is available on the chronic effects of exposure to diesel particulate matter, with animal studies indicating pulmonary histopathology (principally fibrosis), DNA damage and chronic inflammation and remodelling of the airways.

6.2.3 Susceptibility

Key reviews of the health effects of diesel particulate matter have identified the following key factors in relation to the potential susceptibility of the population (IARC 2013; USEPA 2002):

- genetic polymorphisms – with no consistent results that identify any specific genotypes that are more sensitive
- vulnerable populations – children are considered more vulnerable as they spend more time outdoors where exposure to diesel particulate matter would be higher, have higher respiratory rates than adults and have under-developed lungs
- underlying lung and airway disease – in particular existing disease that impairs the clearance of diesel particulate matter (poorly soluble particles) from the lungs and increases the lung burden. In

addition, smokers or workers in specific industries that result in high particulate lung burdens may be more susceptible to the effects of diesel particulate exposures. There are limited studies relating to the susceptibility of individuals with pre-existing lung disease, with a rat study showing no increased susceptibility

- status of the respiratory tract microbiome – the respiratory tract is lined with microflora that expresses enzymes which may increase the metabolic activation of some components of diesel particulate matter and use of antibiotics may affect composition and potential susceptibility.

In relation to many of the studies used in the assessment of the effects of inhalation exposures to diesel particulate matter, rats and mice are considered more sensitive than humans in relation to pulmonary responses but are similarly sensitive to inflammation. In relation to long-term effects rats and mice have sustained inflammation responses making them more sensitive to chronic effects including lung cancer. Hence guidelines established on the basis of the lowest effect observed (or no effects observed) in rats and mice would be sufficiently representative (and likely conservative) of the sensitivity/susceptibility of the human population.

Inhalation exposures to diesel particulate matter have been found to result in increased allergic hypersensitivity. Without any additional information or detail, this allergic hypersensitivity reaction may be the diesel particulate matter hypersensitivity diagnosed in the submission.

The database on studies evaluated in relation to the assessment of effects of diesel particulate exposures included a number of human studies (controlled studies, occupational and epidemiological studies). These included controlled studies on healthy and mildly asthmatic individuals, and individuals first exposed or challenged by ragweed (known allergen) or similar (Landwehr et al. 2021; Long & Carlsten 2022; USEPA 2002). One study (Diaz-Sanchez et al. 1999) specifically assessed the potential for diesel particulate matter to be a primary sensitizer in humans, determining that exposure may increase allergic sensitization.

There are limitations on what can be undertaken with human studies in terms of sensitive individuals (with individuals with significant medical conditions excluded due to ethics) (Long & Carlsten 2022), hence it is also relevant to consider the animal studies considered in these reviews. In relation to assessing the potential for hypersensitive allergen type responses the following studies are relevant (USEPA 2002):

- a mouse study (Miyabara et al. 1998) where exposure to allergens and diesel exhaust was assessed, with hyperresponsiveness of the airway and airway inflammation caused by sensitization identified as effects
- a mouse study (Takano et al. 1998) where the animals were sensitized prior to exposure to the particulates with hyperresponsiveness of the airway, and enhanced allergen-related respiratory diseases identified as effects
- another mouse study (Fujimaki et al. 1997) involved sensitization of the mice prior to exposure to diesel exhaust, with effects on antigen-specific IgE antibody production identified.

The above studies, along with all other published studies relevant to evaluating adverse effects of inhalation exposures to diesel particulate matter, were considered when identifying the critical effects and the critical study – i.e. the study where effects were identified at the lowest level of exposure. Such evaluations are presented in detail by the WHO (WHO 1996), OEHHA (OEHHA 1998a, 1998b), USEPA (USEPA 2002) and more recently by Health Canada (Health Canada 2016)

6.3 Assessing health impacts of exposure

6.3.1 Underlying methodology

Health risk assessment for chemically induced hypersensitivity has two components (WHO 2012):

- the likelihood that a chemical will induce sensitisation in a previously non-sensitised individual
- the likelihood that a chemical will provoke an allergic reaction in those who are already sensitised.

However, in most cases, the risk assessment focuses on the first step, resulting in a safe situation of not becoming sensitised. Effectively and obviously, this would also protect subjects from allergic reactions unless already sensitised. Hence, in most cases, the lower level of elicitation is hardly taken into account in risk assessment.

In relation to diesel particulate matter, the approaches adopted in establishing guidelines have included a range of studies that address the first step. There is the potential that some of the epidemiological data, from occupational environments, included some individuals already sensitise, however this is not known in the populations evaluated.

When deriving a guideline that is protective of all adverse health effects (non-cancer effects in this case), this involves identifying a point of departure (POD), from no effect to some (the most sensitive) effect from the available studies and then diving by an uncertainty or assessment factor (UF or AF), as summarised below:

$$\text{Guideline (threshold)} = \frac{\text{POD (most sensitive effect)}}{\text{UF or AF}}$$

The UF or AF is used to ensure that the guideline is applicable to all humans, including sensitive individuals. It is made up of the following (Dourson, Felter & Robinson 1996):

- Interhuman/interindividual (or intra-species) – used to account for the variation in human sensitivity from exposure, and typically ranges from 1 to 10
- Experimental animal to human – used when extrapolating from a valid experimental animal study to the assessment of humans, and ranges from 1 to 10
- Subchronic to chronic – used when deriving a chronic guideline from a study that has evaluated potential effects from a less than chronic exposure (subchronic) and is typically a factor of 10. This factor is not used where an acute or subchronic guideline is required
- LOAEL to NOAEL – this factor is used where the POD is a lowest observed adverse effect level (LOAEL) rather than a no observed adverse effect level (NOAEL) which is the preferred POD when establishing a guideline protective of all effects, and is typically a factor of 10
- Database strength – this factor is used where the database of studies is considered to be incomplete for the assessment of all adverse effects (as no single study or limited number of studies will adequately address all adverse outcomes), and is typically a factor of 10, however some organisations like Health Canada and WHO may adopt a factor up to 100
- Modifying factor – this is an additional factor based on professional judgement to address scientific uncertainties not adequately accounted for with the above factors, and is typically in the range of 1 to 10.

As in any form of toxic reaction, “dose” is important, in that initial sensitization requires at least a certain minimum exposure (concentration of allergen, its local availability at the site of administration, and the duration of contact or exposure) (WHO 2012).

In someone already sensitized, the likelihood of producing a clinical disorder and its severity are also related to dose, although, by definition, the quantity of allergen required to produce an effect [may be] very much smaller than that associated with a conventional toxic action (WHO 2012).

Hence, when assessing a chemical or substance where hypersensitivity is relevant, and an individual may have already been sensitized, the above factors may or may not be protective.

Guidance on assessing such responses, which are immunological responses, is available from key organisations such as the WHO/IPCC (WHO 2012) and RIVM (ter Burg, Wijnhoven & Schuur 2010). This guidance recommends the use of a sensitization assessment factor (SAF) that essentially replaces the interindividual UF or AF described above. The SAF is intended to more robustly ensure that the variability of response in the population includes those sensitized such that hypersensitive outcomes are protected.

All available guidelines indicates that in the absence of information on the potential variability in susceptibility in the population the maximum factor of 10 for interindividual variability should be used.

Currently, there are no universally accepted models applicable to humans that permit the determination of the dose–response relationship or relative potency of enzymes or low molecular weight chemicals for causing production of allergen-specific antibodies or symptoms of allergy via the inhalation route. However, studies that evaluate the generation of IgE antibodies is a conservative endpoint-that can considered. Hence where such studies are included in those evaluated to determine the critical study, hypersensitive responses may be addressed (WHO 2012). Where insufficient data/studies are available that address immunological responses relevant to respiratory sensitisation, the database strength factor may be used to address this limitation.

6.3.2 Guidelines for assessing diesel particulate exposures

As discussed above guidelines for assessment potential health risks of exposure to diesel particulate matter include threshold guidelines that address non-carcinogen effects, and non-threshold values that enable an assessment of cancer risk.

Threshold non-cancer effects

These are the effects most relevant to the hypersensitive reactions, namely respiratory and immune responses. All available evaluations on diesel particulates have calculated a chronic inhalation guideline of 5 $\mu g/m^3$. Review of the available studies indicates that the database of information includes the generation of IgE antibodies as well as increases mRNA and studies that include allergen challenged studies. These are summarised in **Table 1**.

Only Health Canada has derived a short-term guideline for exposure to diesel particulate matter. This guideline is also included in **Table 1**.

Table 1: Summary of available air guidelines for diesel particulate matter

Guideline/aspect	Chronic (lifetime) guidelines			Acute or short-term guidelines
	USEPA (USEPA IRIS), also adopted by OEHHA (OEHHA 1998a, 1998b)	**WHO (WHO 1996)**	**Health Canada (Health Canada 2016)**	**Health Canada (Health Canada 2016)**
Critical effect	Pulmonary inflammation and histopathology	Pulmonary inflammation and histopathology	Pulmonary inflammation and histopathology	Increased airway resistance in mildly asthmatic individuals
Key study	Chronic inhalation study in rats (Ishinishi et al. 1998)	Chronic inhalation study in rats (Ishinishi et al. 1986; Ishinishi et al. 1998)	Chronic inhalation study in rats (Ishinishi et al. 1986; Ishinishi et al. 1998)	Controlled human studies, 2 hr exposures (Mudway et al. 2004; Riedl et al. 2012; Stenfors et al. 2004)
POD	NOAEL = 0.46 mg/m^3 $NOAEL_{HEC}$ = 0.144 mg DPM/m^3	NOAEL = 0.41 mg/m^3 $NOAEL_{HEC}$ = 0.139 mg DPM/m^3	NOAEL = 0.46 mg/m^3 $NOAEL_{HEC}$ = 0.12 mg DPM/m^3	LOAEL = 0.1 mg/m^3
Assessment factors – total, comprising the following:	30	25	25	10
Interindividual	10	10	10	3
Interspecies	3	2.5	2.5	1
LOAEL to NOAEL	1	1	1	3
Database	1	1	1	1
Modifying factor	1	1	1	1
Guideline	**0.005 mg/m^3 or 5 µg/m^3**	**0.005 mg/m^3 or 5 µg/m^3 (see Note 1)**	**0.005 mg/m^3 or 5 µg/m^3**	**0.01 mg/m^3 or 10 µg/m^3**

HEC = human equivalent dose

Note1: the WHO (WHO 1996) evaluation also considered two other approaches to establishing a non-carcinogenic guideline, with the guidelines established in the range of 2 to 14 µg/m^3

The available reviews suggest that the data/studies evaluated, and the assessment factors adopted adequately address susceptibility in the population. However, some uncertainty remains in relation to the inclusions of immunotoxicology outcomes as these studies were limited at the time of the evaluations. In addition, the short-term guideline has not considered individuals already sensitized to effects. These issues have been further considered in **Section 7**.

Based on this approach the following guidelines would be protective of all such effects:

- **Acute guideline (which typically relates to exposures of an hour) = 10 µg/m^3**
- **Chronic guideline (which relates to long-term exposures – or annual average) = 5 µg/m^3**

Carcinogenic effects

Many of the organic compounds present in diesel exhaust are known to have mutagenic and carcinogenic properties and so it is appropriate that a non-threshold approach is considered for the quantification of lung-cancer endpoints.

In relation to quantifying carcinogenic risks associated with exposure to diesel exhaust, the USEPA (USEPA 2002) has not established a non-threshold value (due to uncertainties identified in the available data).

WHO has used data from studies in rats to estimate unit risk values for cancer (WHO 1996). Using four different studies where lung cancer was the cancer endpoint, WHO calculated a range of 1.6×10^{-5} to 7.1×10^{-5} per µg/m^3 (mean value of 3.4×10^{-5} per µg/m^3). This would suggest that an increase in lifetime exposure to diesel particulate matter between 0.14 and 0.625 µg/m^3 could result in a one in one hundred thousand excess risk of cancer. Hence an incremental chronic guideline of 0.5 µg/m^3 would be protective of increased lifetime cancer risks. This guideline only relates to incremental lifetime exposures, as an annual average, from a specific source (not cumulative exposures).

The California Environmental Protection Agency has proposed a unit lifetime cancer risk of 3.0×10^{-4} per µg/m^3 diesel particulate matter (OEHHA 1998a). This was derived from data on exposed workers and based on evidence that suggested unit risks between 1.5×10^{-4} and 15×10^{-4} per µg/m^3. This would suggest that an increase in lifetime exposure to diesel particulate matter of 0.033 µg/m^3 could result in a 1 in 100,000 excess risk of cancer. This estimate has been widely criticised as overestimating the risk and is not considered robust. Hence, while this is the most conservative value, it has not been used in this assessment.

7.0 Assessment of impacts from the project

7.1 General

As presented in **Section 4**, the air modelling has specifically evaluated exposure concentrations at [redacted] as $PM_{2.5}$, derived from diesel equipment and vehicles. This review has assumed that 100% of the $PM_{2.5}$ assessed from all vehicle emissions comprises diesel particulate matter.

This assessment has considered both incremental impacts from the project and cumulative impacts, relevant to the assessment of exposure to diesel particulate matter.

The assessment of incremental impacts has focused on diesel emissions from all vehicles associated with the project, including trucks using the haul road and Bucketts Way. For the purpose of this assessment, the incremental impacts of $PM_{2.5}$ modelled at [redacted] have been assumed to comprise 100% diesel particulate matter, as diesel emissions are the key sources evaluated.

The assessment of cumulative impacts has included consideration of background. Background concentrations of $PM_{2.5}$ have been used in the AQIA based on measured concentrations relevant to the area. Background $PM_{2.5}$ would be sourced from a large number of regional sources, with diesel emissions only part of the total $PM_{2.5}$. Limited information is available on the proportion of background or ambient $PM_{2.5}$ that comprises diesel particulate matter, however California[2] indicates that diesel particulate matter comprises about 8% of the outdoor $PM_{2.5}$. A study specific to air quality in the Sydney metropolitan area (Broome et al. 2020) indicates that exhaust from diesel vehicles comprises 7.6% of $PM_{2.5}$. These proportions of background that comprises diesel particulate matter is expected to be conservative for rural areas. In addition, the incremental assessment considered in this assessment has included all existing truck

[2] https://ww2.arb.ca.gov/resources/summary-diesel-particulate-matter-health-impacts

movements on Bucketts Way that would already contribute to background. For this assessment diesel particulate matter has been conservatively assumed to comprise 8% of background $PM_{2.5}$.

Using this data, the maximum predicted concentration in air at [redacted] can be directly compared with the guidelines considered to be protective of acute and chronic effects (including hypersensitive effects) as detailed in **Section 6.3.2**.

7.2 Short-term effects at [redacted]

Incremental assessment

The maximum concentration of $PM_{2.5}$, as diesel particulate matter as 24-hour average is 0.07 µg/m^3. To address an exposure more in line with the short-term guideline, the maximum 24-hour average concentration has been converted to a maximum 1 hour average by multiplying by 2.5 (Ontario 2012).

The maximum 1 hour average concentration at [redacted] = 0.07 µg/m^3 x 2.5 = 0.175 µg/m^3.

This is below the short-term guideline of 10 µg/m^3.

The margin of safety relevant these exposures is 60 – this means that the maximum exposure is 60 times less than the guideline protective of all adverse effects.

Cumulative assessment

The background concentration of $PM_{2.5}$ relevant to the area, is variable over each day. Where the maximum incremental exposure from the project is predicted, the 24-hour average $PM_{2.5}$ background concentration is 8.6 (as reported in Table F-13 of the AQIA). Assuming 8% of the background $PM_{2.5}$ comprises diesel particulate matter results in a 24-hour average background concentration of 0.7 µg/m^3.

To address an exposure more in line with the short-term guideline, the background 24-hour average concentration has been converted to a maximum 1 hour average by multiplying by 2.5 (Ontario 2012).

The background 1 hour average concentration = 0.7 µg/m^3 x 2.5 = 1.75 µg/m^3.

Adding the incremental impact from the project (as above) of 0.175 µg/m^3, results in a cumulative diesel particulate matter concentration of 1.9 µg/m^3. The contribution of the project to the total diesel particulate exposure (short-term) is low (9%).

This is below the short-term guideline of 10 µg/m^3.

The margin of safety relevant these exposures is 5.

Overall

The MOS relevant to the incremental and cumulative exposures is more than sufficient to address any uncertainties associated with the studies considered in the derivation of the short-term guideline in relation to hypersensitive individuals.

It is noted that at times, the background 24-hour average $PM_{2.5}$ concentration is higher, likely as a result of other variable sources such as controlled burns/bushfires that are unrelated to diesel emissions, which would be expected to be more consistent. Hence this assessment has not considered this data as the proportion of $PM_{2.5}$ that is diesel particulate matter during such conditions is unknown.

7.3 Chronic effects at [redacted]

Incremental assessment

The maximum concentration of $PM_{2.5}$, as diesel particulate matter at [redacted] as a result of all vehicle emissions from the project as an annual average is 0.02 µg/m^3.

This is below the chronic guideline of 5 µg/m^3. This guideline is protective of all members of the community including sensitive individuals and increased cancer risks.

The margin of safety relevant these exposures is 250 – this means that the maximum exposure is 250 times less than the guideline protective of all adverse effects.

The incremental annual average concentration relevant to diesel particulate emissions from the project have also been compared against the guideline of 0.5 µg/m^3 that is protective of increased lifetime cancer risks (including lung cancer). The incremental annual average concentration of 0.02 µg/m^3 is below the guideline of 0.5 µg/m^3, with a margin of safety of 25. Note that this calculation only relates to incremental lifetime (annual average) exposures from the project.

Cumulative assessment

The background concentration of $PM_{2.5}$ relevant to the area is 7.3 µg/m^3. Assuming 8% of the background $PM_{2.5}$ comprises diesel particulate matter results in a 24-hour average background concentration of 0.58 µg/m^3.

Adding the incremental impact from the project (as above) of 0.02 µg/m^3, results in a cumulative diesel particulate matter concentration of 0.6 µg/m^3. The contribution of the project to the total diesel particulate exposure is low (3%).

This is below the chronic guideline of 5 µg/m^3 and the margin of safety relevant these exposures is 8.

Overall

The MOS relevant to the incremental and cumulative exposures is more than sufficient to address any uncertainties associated with the studies considered in the derivation of the long-term/chronic guideline in relation to hypersensitive individuals.

7.4 Outcome of assessment

Based on the available information, exposure to diesel particulate matter at [redacted] derived from the project would be low and are below guidelines protective of adverse health effects of diesel particulate matter, including consideration of hypersensitive individuals.

Cumulative exposures to diesel particulate matter (from the project and other background sources) are also low and remain below the guidelines protective of adverse health effects for all members of the community. It is noted that the project contribution to the total diesel particulate matter exposure at [redacted] is low. Hence current exposures to diesel particulate matter in ambient air would essentially remain unchanged with the project.

8.0 Limitations and closure

Environmental Risk Sciences has prepared this report for the use of NSW Department of Planning and Environment in accordance with the usual care and thoroughness of the consulting profession. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this report.

It is prepared in accordance with the scope of work and for the purpose outlined in the **Section 1** of this report.

The methodology adopted and sources of information used are outlined in this report. Environmental Risk Sciences has made no independent verification of this information beyond the agreed scope of works and assumes no responsibility for any inaccuracies or omissions. No indications were found that information contained in the reports provided for use in this assessment was false.

This report was prepared in June 2023 and is based on the information provided and reviewed at that time. Environmental Risk Sciences disclaims responsibility for any changes that may have occurred after this time.

This report should be read in full. No responsibility is accepted for use of any part of this report in any other context or for any other purpose or by third parties. This report does not purport to give legal advice. Legal advice can only be given by qualified legal practitioners.

If you require any additional information, please do not hesitate to contact Jackie on 0425 206 295.

Yours sincerely,

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