PUBLIC HEALTH RISK ASSESSMENT

Respirable crystalline silica exposure from Montrose Quarry

Prepared for:

Boral Construction Materials

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BASIS OF REPORT

This report has been prepared by SLR Consulting Australia Pty Ltd (SLR) with all reasonable skill, care and diligence, and taking account of the timescale and resources allocated to it by agreement with Boral Construction Materials (the Client). Information reported herein is based on the interpretation of data collected, which has been accepted in good faith as being accurate and valid.

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DOCUMENT CONTROL

Reference	Date	Prepared	Checked	Authorised
640.30563-R01-v2.0 (DRAFT, UPDATED with monitoring results)	27 February 2024	Tarah Hagen, MSc, DABT, FACTRA	Giorgio De Nola, MSc, RACTRA	Tarah Hagen
640.30563-R01-v1.0 (DRAFT)	20 December 2022	Tarah Hagen, MSc, DABT, FACTRA	Giorgio De Nola, MSc, RACTRA	Tarah Hagen

EXECUTIVE SUMMARY

Boral Construction Materials (Boral) requested SLR Consulting Australia Pty Ltd (SLR) to undertake a human health risk assessment (HHRA) for potential future residential exposure to respirable crystalline silica (RCS) in proximity to the Montrose Quarry in Victoria, Australia (the Quarry). To secure long-term access to extractive resources, Boral proposes to extend the extraction limit of the Quarry to access known reserves within the existing buffer areas. This HHRA is an update of a preliminary draft HHRA undertaken in 2022 (v1.0 of this Report); the updated HHRA incorporates baseline ambient air quality monitoring conducted between July 2022 and July 2023, along with the updated air quality modelling for the proposal.

From a review of national and international agency toxicological information for RCS, silicosis and lung cancer are regarded as the health effects of most concern associated with occupational exposure to RCS dust, with silicosis considered by international agencies as the most sensitive health end point for which health risks from exposure to RCS should be assessed. These agencies consider prevention of silicosis will provide protection against other possible health effects that may be associated with exposure to high levels of airborne RCS in the workplace or ambient outside air. The health effects of potential concern associated with non-occupational exposure to airborne RCS are assumed to be similar to those that may occur as a result of occupational exposure to RCS. Development of the disease is related to the amount of crystalline silica accumulated in the lungs over time, therefore estimates for annual averages of RCS are considered to be most appropriate for use in this HHRA as these are measures for the long-term exposure circumstances potentially encountered by a resident living in close proximity to the Quarry.

The basis of three ambient air guideline values (two sourced from national/international agencies, and one derived from adapting the calculations in a recent review commissioned by Safe Work Australia) are presented in this report. The three values for RCS are remarkably similar: 2 µg/m^3 from TCEQ (2009), 3 µg/m^3 from OEHHA (2004)/EPA VIC (2021), and 3.6 µg/m^3 from adaptation of the threshold cumulative lung silica burden from SLR (2020). Since the TCEQ (2009) and OEHHA (2004) guideline values are based on the same critical study, point of departure, and application of the same uncertainty factor, it was considered appropriate to use the midpoint of the available guideline values (i.e. 3 µg/m^3, measured as an annual average in $PM_{2.5}$) in this assessment. This concentration is an airborne concentration of RCS to which an individual can be exposed for life without significant risk of harm.

Monitoring data were collected at a location in close proximity to the eastern boundary of the Quarry between July 2022 and June 2023. The monitoring location was agreed to in consultation with the Environmental Protection Authority Victoria (EPA VIC). The estimated annual average concentration of RCS was 0.2 µg/m^3, taken to represent existing RCS conditions in this assessment. Ambient air quality modelling was also undertaken by SLR (2024) for the proposed changes at the Quarry estimating emissions of RCS for the following three scenarios:

- Scenario 0: Base Case – Existing Operations.
- Scenario 1: Project Year 5 – Quarry extension stage 3 (worst case construction).
- Scenario 2: Project Year 15 onwards – Quarry operation stage 7 and 8 (maximum operating conditions).

EXECUTIVE SUMMARY

Predictions were undertaken for 33 nearby residences to the east and north-east, and the south and south-west of the Quarry. The maximum predicted annual average cumulative RCS concentrations at any receptor (i.e. residence) over the 5 years modelled occurred at R1 for all three scenarios modelled. The maximum cumulative RCS concentrations were 0.5, 0.5, or 0.4 µg/m^3 for Scenarios 0, 1, and 2, respectively (SLR 2024).

The potential risk of harm to human health of residents living in proximity to the Quarry as a result of RCS exposure in dust originating from the Quarry has been estimated by comparing the assumed current measured and potential future maximum modelled annual average RCS concentrations at the maximum modelled receptor (i.e. residential) location bordering the Quarry (0.2 or 0.5 µg/m^3) with the concentration of RCS that an individual can be exposed to for life without significant risk of harm (3 µg/m^3). Use of the maximum cumulative RCS concentrations near the Quarry is conservative since these would be higher than at other residences.

The results of the comparison show that the measured and modelled RCS emissions are markedly lower than the RCS concentration associated with no significant risk of harm, thus the risk of silicosis in the population living around Montrose Quarry due to RCS exposure from the Quarry is considered to be low.

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1 Introduction

Boral Construction Materials (Boral) requested SLR Consulting Australia Pty Ltd (SLR) to undertake a human health risk assessment (HHRA) for potential exposure of nearby local residents to respirable crystalline silica (RCS) in air that is emanating from the Montrose Quarry in Victoria, Australia (the Quarry). The Quarry is located at 56 Canterbury Road, Montrose in the eastern suburbs of Melbourne as shown below in Figure 1. This HHRA is focussed on those people living in close proximity to the Quarry and potential future exposure as a result of changes in Quarry operations. There are plans to expand the operations at the Quarry.

Figure from Google Earth.

Figure 1 Quarry Location (in Montrose, Victoria)

A previous version of this report (Draft v1.0, dated December 2022) was preliminary in nature as it had been conducted prior to undertaking air quality modelling for the proposed changes to Quarry operations. The updated version of the report herein now incorporates baseline ambient air quality monitoring conducted between July 2022 and July 2023, along with the updated air quality modelling for the proposal[1]. As a result, many of the conservative exposure assumptions that were adopted in the preliminary HHRA based on historical air quality measurements for dust were able to be refined through the use of contemporary monitoring data. This, together with results from updated air quality modelling, enables a more accurate estimation of potential future exposure to RCS by local residents who live in close proximity to the Quarry.

1.1 Objectives

The objectives of the HHRA herein are to:

- Undertake an assessment of the potential risk of harm to human health of residents living in proximity to the Quarry as a result of RCS exposure in dust originating from the Quarry taking into consideration potential increases to RCS exposure due to the proposed changes in Quarry operations.

The HHRA is limited in scope as it does not consider human health risks associated with any other chemicals associated with the Quarry (apart from RCS), nor occupational health risks at the Quarry, nor risks to ecology or the environment (on-site and off-site).

1.2 The Quarry and Surrounds

Montrose Quarry is a hard rock quarry, with an approximate boundary shown in Figure 2, that currently produces between 700,000-900,000 tonnes per annum (Boral 2021). Existing operational activities at the site include excavation of the quarry face (including drilling and blasting), face loading and hauling, crushing, screening, blending, stockpiling, washing and pre-coating, as well as transport by trucks. The pit is located in the centre of the Quarry and much of the plant used to process rock at the quarry is located in the northwest corner of the site and includes a Crushing Plant[2] and other buildings[3].

The Quarry consists of multiple parcels of land that are zoned[4] predominantly as Special Use Zones (Schedule 1 or Schedule 6) of the Yarra Ranges local government Authority. A parcel of land in the southwest of the Quarry (on site) is zoned as Green Wedge. There are varying land uses around the Quarry including the following:

- North: Industrial land use then neighbourhood residential. Note that the land parcel immediately west of the Quarry as shown in Figure 2 may include operations associated with the Quarry.
- East: Neighbourhood residential in the northern portion and a public reserve in the central and southern portions then low density residential.

[1] Reported in SLR (2024). Report entitled "Montrose Quarry Extension Air Quality Impact Assessment". SLR Project Number 640.V30655.00000, v01, dated 16 February 2024.

[2] The Crushing Plant consists of a Primary Plant, Secondary Plant, Pug mill Plant, Screen house, Dust Extraction Plant, Crusher 02 and Screen 5 & 6.

[3] Other buildings at the Quarry include the Workshop, Primary Switch room, Secondary Switch room, Tertiary Switch room, Pug mill Switch room and Amenities.

[4] Zoning information (or general land uses) was obtained from VicPlan: https://mapshare.vic.gov.au/vicplan/

- South: Green wedge land use which includes residences (residential) and a public use zone (water basin).
- West: Industrial land use then general residential land use followed by more industrial land use.

Residents located nearest to the actual Quarry are located immediately to the east of the site (along the northern portion of the eastern boundary) or to the north beyond the industrial area. There are some residences in the Green Wedge zone to the southwest also in relative close proximity to the Quarry pit.

Background figure from Google Earth. All boundaries shown are approximations.
Overlaid land uses from VicPlan: https://mapshare.vic.gov.au/vicplan/

Figure 2 The Quarry and Surrounding Land Uses

1.3 Proposed Changes at the Quarry

To secure long-term access to extractive resources, Boral proposes to extend the extraction limit of the Quarry to access known reserves within the existing buffer areas. It is proposed to extend the open-pit quarry extraction limit towards the south (as shown in Figure 3). The development of the proposed pit expansion is envisaged to occur in eight stages as summarised in Table 1.

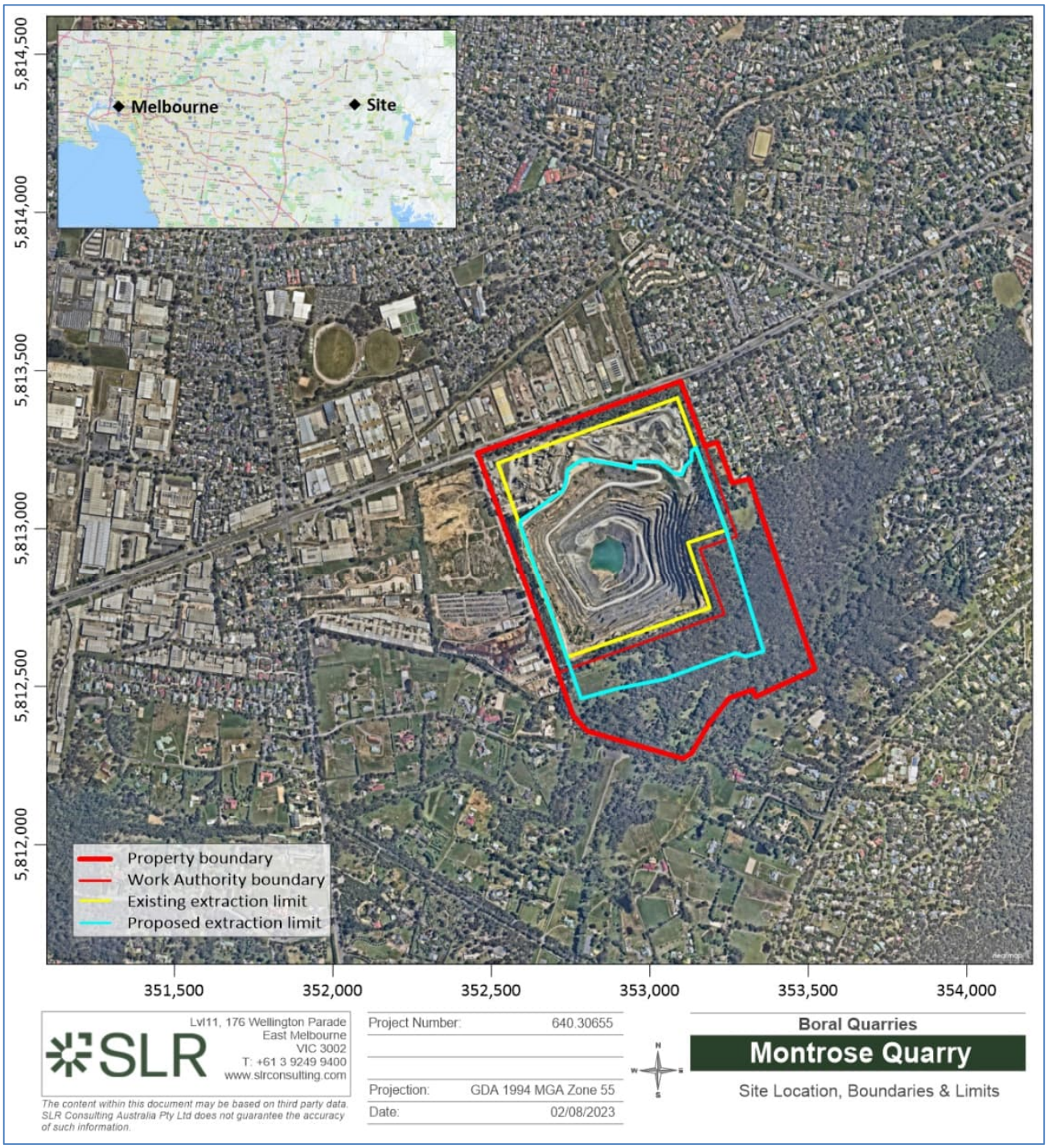

Figure 3 Existing and Proposed Boundaries of the Quarry (from SLR 2024).

There will not be any changes to the existing hot mix asphalt plant and concrete batching plant at the Quarry. These plants will remain unchanged in terms of throughput, production, location, etc.

Table 1 Project Extraction Volumes by Stage (from SLR 2024)

Stage	Years from Start	Volume (m^3)	Overburden (m^3)	Resource (m^3)	Resource (t)(1)
1	0.5	55,400	55,400	-	-
2	2.3	682,400	408,000	274,400	740,880
3	5.5	1,609,000	638,800	970,200	2,619,540
4	7.2	938,100	349,500	588,600	1,589,220
5	10.2	1,354,300	124,500	1,235,500	3,335,850
6	14.7	1,336,700	-	1,337,000	3,609,900
7	21.8	2,132,900	-	2,132,900	5,758,830
8	29.3	2,246,900	-	2,246,900	6,066,630
Final	32.2	878,500	-	878,500	2,371,950
Total	32.2	11,234,200	1,576,000	9,658,000	26,076,600
(1) 2.7 t/m^3					

2 Methodology overview

The HHRA undertaken herein broadly follows the enHealth (2012) *Environmental Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards*. Relevant toxicological information was sourced from the scientific literature with sources cited in this report. SLR undertook the ambient air quality monitoring and air quality modelling which forms the basis of the exposure considerations in this report. The assessment herein was undertaken as follows:

1. Issue identification: This step involved gathering background information and introduction to the issue under investigation (as discussed in Section 1).

2. Hazard assessment: This step involved consulting national and international toxicological reviews for the substance under consideration (RCS) to provide an overview of the potential health effects which could result from exposure to RCS. This included defining critical endpoints and sourcing relevant concentration response information. The information is presented in Section 3. The basis of three ambient air guideline values (two sourced from national/international agencies, and one derived from adapting the calculations in a recent review commissioned by Safe Work Australia) are discussed and were considered to inform the risk characterisation in this report.

3. Exposure Assessment: This step involved describing the available data to help inform exposure assumptions. This involved considering air quality monitoring to inform existing RCS concentrations in air (Section 4.1) and air quality modelling of RCS concentrations resulting from the proposed changes to the Quarry (Section 4.2).

4. Risk Characterisation and Uncertainties: This step combined the information from the hazard assessment and exposure assessment to determine the potential risk of harm from exposure to RCS by residents living in close proximity to the Quarry. An analysis of the uncertainties associated with the assessment and their potential impact on overall conclusions was also undertaken (Section 5).

3 Health Effects of Crystalline Silica

3.1 Particle size considerations

Ambient air quality monitoring and modelling of particulates is generally done for three particulate size fractions: Total Suspended Particulates (TSP), particulates less than 10 µm in diameter (PM_{10}), and particulates less than 2.5 µm in diameter ($PM_{2.5}$). Particle size is physiologically relevant as it is an important determinant for where particles will deposit in the respiratory tract.

- Particles with a 50% sampling efficiency of 100 µm in diameter are termed 'inhalable', i.e. they can be inhaled into the nose or mouth. However, as the air passes down into the lungs larger particles are progressively deposited in the upper airways, taken up the mucociliary ladder and expectorated or swallowed. This makes the 'inhalable' fraction less of a health concern than the 'respirable' fraction of dust (see below).
- The 'thoracic' fraction is the fraction of 'inhalable' particles that can penetrate the bronchial region with a 50% sampling efficiency of approximately 10 µm. Many of these particles are large enough to be efficiently taken up the mucociliary ladder and do not penetrate deep into the lungs.
- Only the 'respirable' fraction of dust is able to reach the lower bronchioles and alveolar (i.e. gas-exchange) regions of the lung, where persistent substances can accumulate as they are not readily removed from the lung.
    - In Australian workplaces, respirable dust is measured by a size selective device according to Australian Standard AS 2985-2009: *Workplace atmospheres – Method for sampling and gravimetric determination of respirable dust*. This Australian Standard has adopted the ISO 7708 definition of respirable dust and defines it as the percentage of 'inhalable' particulate matter collected by a device conforming to a sampling efficiency curve which passes through specific points. For example, PM_2 is 97% respirable, PM_4 is 56% respirable and PM_{10} is 2% respirable (SWA 2013).
    - In ambient air both PM_{10} and $PM_{2.5}$ are often termed 'respirable', although in strict terms only a small percentage of PM_{10} is actually respirable. $PM_{2.5}$, by definition, includes particles <2.5 µm in size which includes ultrafine PM (i.e. <0.1 µm).

The sampling convention definitions for the different fractions of particulate matter are visually shown in Figure 4. For RCS, it is the 'respirable' fraction which is physiologically important. It is also noteworthy that available guideline values for RCS are applied to the 'respirable' fraction, i.e. ambient $PM_{2.5}$ (see Section 3.3).

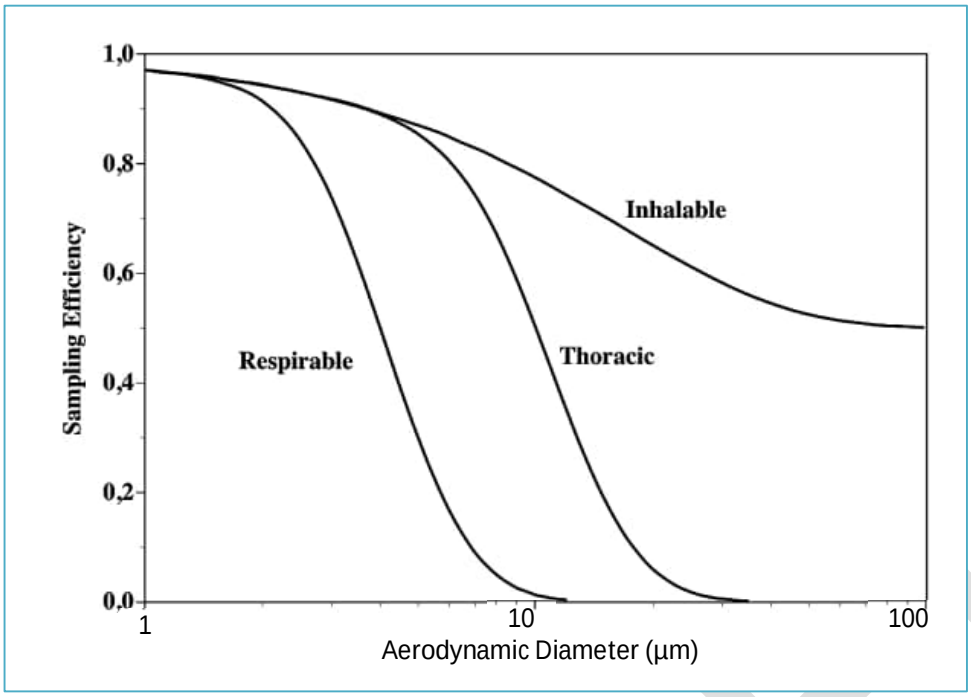

Figure 4 ISO sampling conventions for the different fractions of dust (from Lidén and Harper 2006)

3.2 Overview of health effects from crystalline silica exposure

Silica may either be crystalline or non-crystalline; it is occupational exposure to respirable crystalline silica that is associated with adverse health effects (de Klerk et al. 2002, IARC 1997, WHO 2000). It is stated in CICAD (2000) that "*to date, there are no known adverse health effects associated with non-occupational exposure to quartz dust*[5]*".* However, chronic (and sometimes very high acute) exposure to crystalline silica in the occupational setting is associated with increased incidences of tuberculosis, bronchitis, emphysema, chronic obstructive pulmonary disease, renal diseases, silicosis and lung cancer. Of these potential health effects, silicosis and lung cancer are the effects of most concern associated with occupational exposure to respirable quartz dust (US EPA 1996, CICAD 2000, de Klerk et al. 2002, SLR 2020).

In an occupational setting, inhalation exposure[6] to respirable crystalline silica can result in a number of adverse pulmonary responses, including the following (AIOH 2018, ATSDR 2019, Castranova and Vallyathan 2000, de Klerk et al. 2002, IARC 2012).

[5] The most common form of crystalline silica is quartz. Quartz and crystalline silica are terms that are often used interchangeably in the scientific and medical literature.

[6] Other routes of exposure to silica are insignificant and/or not associated with adverse health effects. Very little silica is absorbed into the body after ingestion, it is also expected that negligible amounts of silica would be absorbed through the skin, and silica compounds are not metabolised (ATSDR 2019, 2020).

- Acute silicosis[7] (also termed alveolar proteinosis or silicoproteinosis); this results from exposure to 'extremely high' levels of RCS which are typically not well defined[8], over a short period of time, often within a few weeks to less than 5 years' of exposure (ATSDR 2019, NICNAS 2018, RACP 2020, SLR 2020, TCEQ 2009). It is a rare and fatal condition in humans. Like accelerated or chronic silicosis, acute silicosis progresses in the absence of further exposure (ATSDR 2019, UK HSE 2011). Acute silicosis has been reported in occupations such as sandblasting, surface drilling, tunnelling, silica flour milling, ceramic making, and more recently, in stone benchtop manufacturing (AIOH 2018, Castranova and Vallyathan 2000, SWA 2019, UK HSE 2011). A common attribute of these professions is that they involve tasks that produce small particles of airborne dust with high silica content.
- Accelerated silicosis; this may occur after exposure to 'high' concentrations of RCS for 1–10 years (RACP 2020, UK HSE 2001). Both acute and accelerated silicosis cases may be diagnosed in the same studies. Again, the exposure concentrations which may result in accelerated silicosis are not well defined. UK HSE (2001) indicates the disease may occur when exposed to daily average RCS concentrations of approximately 1–5 mg/m^3 but does not provide references for this. The Australian Government (2004) states that extremely high exposures (e.g. exposures of 90 mg/m^3 over 8 hours per day for 6 months) are associated with much shorter latency and more rapid disease progression, but also provide no references for this information. The symptoms of accelerated silicosis are similar to those of chronic silicosis[9], but clinical and radiographic progression is much more rapid (AIOH 2018, ATSDR 2019) and fibrosis may be irregular and more diffuse or not apparent on the chest radiograph (NIOSH 2002).
- Chronic silicosis; this form of silicosis, also termed classic silicosis, is the most common form of silicosis and is the critical health effect (along with lung cancer) that forms the basis of current ambient air guidelines (Section 3.3). Chronic silicosis may result from inhalation of RCS over prolonged periods, which promotes the formation of classic fibrotic nodules. Symptoms may or may not be obvious (CCOHS 2017). However, as chronic silicosis progresses, pulmonary function deficits become more obvious resulting in symptoms such as fatigue, extreme shortness of breath, chest pain or respiratory failure (Castranova and Vallyathan 2000, CCOHS 2017).
- Progressive massive fibrosis (also called conglomerate silicosis or complicated silicosis); this is a form of progression in severity of chronic silicosis, where smaller nodules in the lung agglomerate (ATSDR 2019). Compromised pulmonary function can lead to right ventricular failure, congestive heart failure and increased risk of pneumothorax. General health significantly declines and severe pulmonary damage can result in death (ATSDR 2019). Like for acute and accelerated silicosis, the disease may progress even after exposure ceases.

[7] Silicosis is one of the more destructive forms of pneumoconiosis (characterised by scarring of the lungs), which is contracted by prolonged exposure to high levels of fine crystalline silica dust. Pneumoconiosis is a condition characterised by permanent deposition of substantial amounts of particulate matter in the lungs and by the tissue reaction to its presence; depending on the chemical nature of the particulate it may range from relatively harmless forms of tissue hardening to the destructive fibrosis of silicosis. Silicosis is an irreversible and progressive condition in which healthy lung tissue becomes replaced with areas of fibrosis.

[8] Most reviews consulted do not provide information regarding what is meant by 'extremely high' or 'very high' exposures. UK HSE (2009) indicates acute silicosis may occur after RCS exposures in the order of 1.5 mg/m^3 on a daily basis for a year or two, however do not provide references for this statement.

[9] In both the accelerated and chronic forms of the disease, lung inflammation leads to the formation of excess connective tissue, or fibrosis, in the lungs (OSHA 2016).

- Lung cancer; this disease has been associated with long-term occupational exposure to RCS (AIOH 2018, IARC 2012). According to AIOH (2018), most studies indicate that in the absence of silicosis, any increased risk of lung cancer above background rates should be negligible. In their review of the literature, Borm et al. (2011) and IARC (2012) concluded that the mechanism of RCS genotoxicity (which may lead to lung cancer) is secondary due to persistent inflammation. The role of inflammation driven by the surface of quartz in genotoxic and carcinogenic effects after inhalation has also been confirmed in an updated review of the genotoxicity of RCS, and the findings support a practical threshold for this endpoint (Borm et al. 2018).

As summarised in SLR (2020), silicosis is caused by crystalline silica's cytotoxicity to alveolar macrophages and consequently almost negligible clearance from the human lungs. This allows RCS to trigger progressive pulmonary inflammation at levels well below the high mass burdens required to 'overload' the lungs and impair pulmonary clearance that is typical of 'poorly soluble particles of low toxicity' (PSPLTs). Experimental animal studies show that exposures that lead to a critical lung crystalline silica burden can result in inflammation that becomes progressively severe, even after exposure has stopped. In contrast to PSPLTs, there is sufficient evidence of an association between RCS exposure and lung cancer, chronic lung inflammation and severe pulmonary fibrosis in humans (SLR 2020).

Type and severity of silicosis is influenced by the intensity, frequency and duration of exposure (ATSDR 2019). Time from first exposure to onset of disease (i.e. the latency period) varies inversely with intensity of exposure and may be as short as a few weeks for acute silicosis, to as long as 20 or more years for chronic silicosis and progressive massive fibrosis. Disease severity may continue to slowly increase over decades even after exposure has been discontinued, possibly due to RCS dust that is retained in the lungs (ATSDR 2019). Therefore, ending exposure does not necessarily prevent development or progression of silicosis. Tuberculosis has been a common complication of silicosis and is often seen in severe grades of the disease (Castranova and Vallyathan 2000).

RCS exposure has also been associated in numerous epidemiological studies with increased risk of autoimmune diseases [e.g. rheumatoid arthritis, systemic lupus erythematosus (SLE), scleroderma, anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis, sarcoidosis and end-stage renal disease) (SLR 2020)[10]. Although higher risks are generally seen in silicosis patients, there are now several studies that have reported associations between silica exposure, in the absence of silicosis, and risk of rheumatoid arthritis, scleroderma and other systemic autoimmune diseases (WHO 2006). However, the incidence of autoimmune disorders is low when compared to pulmonary diseases associated with RCS exposure, and associations have not been observed in all studies. In addition, IIAC (2020) noted in several studies there appears to have been a liberal interpretation of silica exposure with over 20% of control subjects assessed as having been exposed.

With respect to renal effects, ATSDR (2019) indicated that comparison of exposure-response data for renal effects and silicosis showed that renal toxicity typically occurs at higher cumulative exposure levels than silicosis. A meta-analysis by Mohner et al. (2017) found that while the studies of cohorts exposed to RCS found elevated standardised mortality ratios (SMRs) for renal disease, no clear evidence of a concentration-response relationship emerged.

[10] There is no adequate exposure-response data available for RCS exposure and systemic sclerosis nor many of the other non-pulmonary conditions. Overall, the results of these studies indicate that occupational exposure to RCS in some workers who may also have other risk factors for autoimmune diseases (genetic predisposition, exposure to other chemicals) may lead to an increased risk of developing an autoimmune disease (ANSES 2019, Pollard 2016). However, according to ANSES (2019) the data currently available for each disease considered individually are inadequate for determining quantitative concentration-response relationships.

AIOH (2018) indicated there is an increasing weight of evidence regarding chronic exposure to RCS (and also other non-siliceous mineral dusts) causing chronic obstructive pulmonary disease (COPD), even in the absence of silicosis (e.g. Hnizdo and Vallyathan 2003). Destruction of alveolar walls in silica dust exposed subjects can lead to emphysema which is the main contributor of COPD.

Of the potential health effects silicosis and lung cancer are regarded as the effects of most concern associated with occupational exposure to RCS dust (AIOH 2018, de Klerk et al. 2002, US EPA 1996, WHO 2000), with silicosis considered by international agencies (ACGIH 2010, OEHHA 2004, OSHA 2016, SCOEL 2003, TCEQ 2009) as the most sensitive health end point for which health risks from exposure to RCS should be assessed. These agencies consider prevention of silicosis will provide protection against other possible health effects that may be associated with exposure to high levels of airborne RCS in the workplace or ambient outside air.

The health effects of potential concern associated with non-occupational exposure to airborne RCS are assumed to be similar to those that may occur as a result of occupational exposure to RCS. Due to the fact that development of the disease is related to the amount of crystalline silica accumulated in the lungs over time, day to day variations in ambient air particulate content (where exposures are not as high as in an occupational setting) are likely to have little bearing on potential health risks from RCS exposure. Consequently, estimates for annual averages of airborne respirable particulates would be most appropriate for use in this risk assessment as these are measures for the long-term exposure circumstances potentially encountered by a resident living in close proximity to the Quarry.

3.3 Concentration response

Ambient air quality guidelines for respirable RCS have been derived by two American state agencies: i) the California Office of Environmental Health and Hazard Assessment (OEHHA 2004) and the ii) Texas Commission on Environmental Quality (2009). EPA Victoria (2021) has adopted the OEHHA (2004) guideline value as a guideline value for assessing air pollution in Victoria.

In addition, iii) Safe Work Australia recently commissioned a research report written by SLR (2020) for derivation of an occupational Short Term Exposure Limit (STEL); the report contains one of the most recent reviews of the experimental toxicological information for RCS exposures and defines a critical threshold cumulative burden for crystalline silica in the lungs. For this reason, it was considered worthwhile to include in the discussion presented in this section. The calculations presented in SLR (2020) for derivation of a STEL using the critical threshold cumulative crystalline silica lung burden have been adapted in this report to cater for exposures by the general public in order to derive a tentative RCS guideline value based on this information.

The basis of all three guideline values (those from OEHHA 2004/EPA VIC 2021, TCEQ 2009, and the guideline derived from adapting the calculations in SLR 2020) are described in subsequent sections and have been considered to inform the risk characterisation in this report.

3.3.1 OEHHA (2004)/EPA VIC (2021)

OEHHA (2004) has derived a chronic reference exposure level (REL)[11] for the non-carcinogenic (i.e. silicosis) effects of crystalline silica of 3 µg/m^3. For their point of departure (POD), the agency used a study which investigated silicosis risk retrospectively in 2,235 white South African gold miners (Hnizdo and Sluis-Cremer 1993). Exposure estimates were made for nine separate occupational categories based on a special study of dust levels in these mines. Workers in this study had a minimum of 10 years and an average of 24 years' service from 1940 until early 1970s. Miners had an annual chest radiograph while mining; they were followed until 1991 for radiographic signs of the onset of silicosis with an ILO category 1/1 (definite silicosis) or greater selected to designate silicosis. Latency period for development of silicosis was largely independent of the cumulative dust exposure (CDE). OEHHA (2004) estimated the mg/m^3-yr RCS exposure based on Hnizdo and Sluis-Cremer's estimate of 30% silica in the dust. The study was powerful enough to detect a 1.9% incidence of silicosis (9 cases out of 474 exposed) at 0.9 mg/m^3-yr silica. Because this incidence represents approximately the sensitivity limit of the data, and silicosis is a severe irreversible endpoint, OEHHA (2004) selected the $BMCL_{01}$ (i.e. the lower bound estimate of the concentration at which 1% of the population develops silicosis) of 2.12 mg/m^3-yr CDE or 0.636 mg/m^3-yr RCS as the POD for the chronic REL.

OEHHA (2004) then adjusted the occupational exposure concentration to an equivalent exposure concentration for the general public as follows:

[0.636 mg/m^3-yr ÷ 24 years] x 10 m^3/20 m^3 x 270 days/365 days = 0.0098 mg/m^3 (i.e. 9.8 µg/m^3)

The agency applied an uncertainty factor of 3x for human variability[12] to this adjusted concentration to derive a chronic REL (rounded) of 3 µg/m^3.

The REL is meant to be applied only to particles of crystalline silica (quartz, cristobalite, tridymite) of respirable size, as defined by the occupational hygiene methods described by ACGIH (2004)/ISO (1995) which has a 50% cut-point at 4 µm, i.e. essentially PM_4. In Australia ambient air monitoring of particulates is limited to particulates of 2.5 and 10 µm. The 2.5µm particulate size is 97% respirable and the 10µm size only 2% respirable, as measured by Australian Standard AS 2985-2004. Thus, the OEHHA (2004) ambient air standard if used in Australia would be 3 µg/m^3 RCS (measured as $PM_{2.5}$). As discussed previously, EPA Victoria (2021) has adopted the OEHHA (2004) guideline value as a guideline value for assessing air pollution in Victoria and recommends it be applied to $PM_{2.5}$.

[11] A chronic reference exposure level (REL) is defined as an airborne level that would pose no significant health risk to individuals exposed to that level for an indefinite period of time. RELs are standards based solely on health considerations and are developed from the best available data in the scientific literature. Thus, a chronic REL is an airborne concentration to which an individual can be exposed for life without significant risk of harm. Although OEHHA (2004) do not attach an averaging time to their RELs, it is usual to use annual average modelled concentrations for comparison with a chronic REL. This is consistent with the Agency for Toxic Substances and Disease Registry (ATSDR) definition of chronic exposure being greater than 365 days.

[12] The selection of 3x by OEHHA (2004) as an uncertainty factor for human variability was based on several considerations: i) the workers who developed silicosis at low RCS concentrations are by definition the most sensitive workers to RCS-induced silicosis and since more than 14,000 workers were examined in the principal and supporting studies, the sensitive individuals represent at least part of the range of sensitivity to be expected in the general population; ii) use of a default human uncertainty factor of 10 with the data from the key study would result in a chronic REL of 0.9 µg/m^3, a level in the range of ambient background levels in California and there is no evidence that these background levels are causing silicosis, iii) the concentration response curve for silicosis due to inhalation of RCS is steep therefore an uncertainty factor is still considered warranted.

3.3.2 TCEQ (2009)

The Texas Commission on Environmental Quality (TCEQ 2009) have derived both an acute (1-hour average) and chronic (annual average) health-based reference exposure level (ReV) for RCS.

Acute ReV

For derivation of the acute ReV, TCEQ (2009) used short-term toxicity data from experimental animal studies as they concluded there was insufficient human data for development of a guideline. They chose a study by Warheit et al. (1991)[13] as the key study since it included a well-conducted evaluation of acute exposure (6 hours), whereas supporting studies examined subacute exposures (6 hours per day for either 3 or 5 days). TCEQ (2009) considered the lowest concentration (10 mg/m^3) from the Warheit et al. (1991) study to be a Lowest Observed Adverse Effect Concentration (LOAEC) for RCS (pulmonary inflammatory response).

TCEQ adjusted the LOAEC (i.e. POD) from the 6 hour exposure duration (C_1) in the Warheit et al. (1991) study to an adjusted POD (i.e. POD_{ADJ}) of 18.2 mg/m^3 for a 1 hour exposure concentration (C_2) using Haber's Rule as modified by ten Berge et al. (1986) ($C_1^n \times T_1 = C_2^n \times T_2$) with n=3 (noting 3 is the default in an absence of data), where both concentration and duration play a role in toxicity[14].

TCEQ (2009) then used the multiple path particle dosimetry model (MPPD) v2.0 to calculate the deposition fraction of silica in the target respiratory region (considered to be the tracheobronchial and pulmonary regions) for both rats and humans. The regional dose deposition ratio (RDDR) was then calculated to be 0.775 using the following formula[15]:

[13] In this study, male Crl:CD BR rats were exposed at 10, 50 or 100 mg/m^3 Min-U-Sil® quartz (mass median aerodynamic diameter, MMAD = 3.7 µm) or carbonyl iron particles (MMAD = 3.6 µm) for 6 hours or for 6 hours per day for 3 days. Three animals exposed at 10 mg/m^3 silica, 3 animals exposed at 50 mg/m^3, 6 animals exposed at 100 mg/m^3 silica, and 14 sham animals were evaluated at 0, 24, and 48 hours post-exposure as well as 1, 2, and 3 months post-exposure. The study assessed various endpoints including inflammation, cytotoxicity, and histopathology. The results can be summarised as follows:

- At 50 mg/m^3 for 6 hours, rats exhibited a sustained pulmonary inflammatory response.
- At 10 mg/m^3 for 6 hours, rats did not exhibit an initial inflammatory response. Although authors noted increased neutrophils in these animals at 1 and 3 months post-exposure. Similarly, alkaline phosphatase (ALP) activity (a marker of tissue damage and type II pneumocyte differentiation) did not initially differ significantly from controls in animals exposed at 10 mg/m^3. However, ALP activity was increased at 1 month post-exposure. Lactate dehydrogenase (LDH; a marker of cytotoxicity) increased in a concentration-dependent manner within 24 hours after exposure and remained elevated up to 3 months post-exposure. Protein concentrations in bronchoalveolar lavage fluid (BALF) did not differ from controls in animals exposed at 10 mg/m^3. Interestingly, *in vitro* phagocytosis by macrophages was increased in animals exposed at 10 mg/m^3 and decreased in animals exposed at the two higher concentrations compared to controls, suggesting increasing cytotoxicity with exposure concentration.
- Animals exposed at the varying concentrations of silica for 6 hours developed pulmonary lesions. However, there is no discussion of concentration-specific histopathological effects in the publication.

[14] $C_2 = [(C_1)^3 \times (T_1/T_2)]^{1/3} = [(10\ mg/m^3)^3 \times (6\ hours/1\ hour)]^{1/3} = 18.2\ mg/m^3 = POD_{ADJ}$

[15] TCEQ do not describe how the surface areas they used in the MPPD modelling were chosen. Their documentation suggests both tracheobronchial and pulmonary regions are relevant but human and experimental rat data indicate it is the alveolar region where RCS has its action on the lungs (SLR 2020).

$$RDDR = \left[\frac{(V_E)A}{(V_E)H}\right] x \left[\frac{DF_A}{DF_H}\right] x \left[\frac{NF_H}{NF_A}\right]$$

Where:
V_E = Minute volume. This was 137.3 mL/min for rats and 13,800 mL/min for humans.
DF = Deposition fraction in the target region of the respiratory tract. This was 0.111 for rats and 0.226 for humans.
NF = Normalising factor (to normalise the surface areas of the relevant region in the lungs). This was 543,200 cm^2 for humans and 3,422.5 cm^2 for rats.
A = Animal
H = Human

The calculated RDDR of 0.775 was then used by TCEQ (2009) to dosimetrically adjust from an animal POD to a human equivalent concentration POD (POD_{HEC})[16] of 14.1 mg/m^3. TCEQ (2009) then applied a composite uncertainty factor of 270 (3x for use of a LOAEC rather than a NOAEC, 3x for interspecies extrapolation, 10x for human variability, and 3x for database uncertainty[17] and rounded up to 300) to the POD_{HEC} to derive a 1 hour air guideline value[18] of 47 µg/m^3.

Although not explicitly specified, the study on which the acute ReV is based involved exposure to silica particles with a mass median aerodynamic diameter (MMAD) of 3.7 µm, which suggests the acute ReV should be applied to the respirable fraction of RCS in ambient air. The closest fraction to respirable which is monitored for in air is the $PM_{2.5}$ fraction.

Chronic ReV

For derivation of the chronic ReV, TCEQ (2009) selected the same study also used by OEHHA (2004)/EPA VIC (2021) in South African gold miners (Hnizdo and Sluis-Cremer 1993) to derive a POD. TCEQ (2009) derived practically the same $BMCL_{01}$ as OEHHA (2004) of 0.635 mg/m^3-yr RCS as the POD for the chronic ReV. TCEQ (2009) noted that this value of 0.635 mg/m^3-yr RCS was based on the estimates of cumulative (respirable) dust exposure for the cohort of gold miners assuming the dust contained an average quartz content of approximately 30%. However, according to Gibbs and Du Toitt (2002), the actual quartz content would have been approximately 54%, rather than 30%. Therefore, TCEQ (2009) considered it more appropriate to multiply the $BMCL_{01}$ by a ratio of 54/30 to derive the adjusted POD of 1.143 mg/m^3-yr RCS.

TCEQ (2009) the adjusted the POD to exposures for the general population as follows.

$$Cumulative\ POD_{HEC} = Cumulative\ POD_{Occup} x\ {}^{VE_{ho}}/_{VE_h}\ x(shifts\ per\ year_{occup}/days\ per\ year_{res})$$

[16] POD_{HEC} = POD_{ADJ} x RDDR = 18.2 mg/m^3 x 0.775 = 14.1 mg/m^3 = 14,100 µg/m^3.

[17] TCEQ (2009) used an uncertainty factor (UF) of 3x for extrapolation from a LOAEC to a NOAEC, because they reasoned that the link between the inflammation and cytotoxicity noted in the key study and clinical outcomes is unknown and the default exposure duration adjustment using Haber's Rule with an exponent of 3 tends to be conservative. An UF of 3x for extrapolation from animals to humans was used instead of the default 10x factor, because dosimetric adjustments using the RDDR were conducted to account for toxicokinetic differences but not toxicodynamic differences. Because the variability of the acute response in humans is unknown, the full default UF of 10x was used to account for potential sensitive human subpopulations such as those with existing pulmonary inflammation due to other causes. An additional UF of 3x was applied to account for the lack of acute studies in other species and that exposure in the Warheit et al. (1991) study was to very few animals, i.e. 3, 3, and 6 animals were exposed, respectively, at 10, 50, and 100 mg/m^3.

[18] 14,100 µg/m^3 ÷ 300 = 47 µg/m^3.

Where:
Cumulative POD_{HEC} = Cumulative residential human-equivalent POD for RCS
Cumulative POD_{Occup} = Cumulative occupational POD for RCS (i.e. 1.143 mg/m^3-yr RCS).
VE_{ho} = Occupational ventilation rate for an 8-hour day (10 m^3/day)
VE_h = Non-occupational ventilation rate for a 24-hour day (20 m^3/day)
Shift per $year_{occup}$ = yearly occupational exposure frequency (270 days per year)
Shifts per $year_{res}$ = yearly residential exposure frequency (365 days per year).

The resulting Cumulative POD_{HEC} is 0.423 mg/m^3-yr RCS. The Cumulative POD_{HEC} was then converted to an equivalent annual average exposure concentration over a lifetime by dividing the value by 70 years to derive a POD_{HEC} of 0.00604 mg/m^3 (i.e. 6.04 µg/m^3). TCEQ (2009) then applied an uncertainty factor of 3 to account for human variability within the general population to derive a chronic ReV of 2 µg/m^3.

The derivation of the chronic ReV by TCEQ (2009) is therefore very similar to the OEHHA (2004) REL derivation, the only difference being how the POD was adjusted to exposure for the general public. The critical study, actual POD and the uncertainty factor used were all the same.

Although not explicitly specified, TCEQ (2009) indicate the chronic toxicity values were developed based on occupational epidemiology studies to particles ≤ 4 µm, which suggests the chronic ReV can be applied to the $PM_{2.5}$ fraction of RCS in ambient air.

3.3.3 Adaptation of critical threshold cumulative crystalline silica lung burden from SLR (2020)

SLR (2020) recently summarised in detail the available toxicological information on crystalline silica in order to derive a proposed occupational short-term exposure limit (STEL) for RCS. The methodology for derivation of a STEL was adapted herein to derive an ambient air guideline value which would be protective of the critical threshold cumulative crystalline silica lung burden in humans of the general population, which means it is also protective of long-term exposures that results in the same critical silica lung burden.

The steps followed for derivation of a STEL in SLR (2020) were adapted herein as follows.

- SLR (2020) consolidated 13 rat toxicology studies to identify a POD which represents a cumulative RCS exposure by rats (mg/m^3.hr) that does not exceed the critical pulmonary target load required for progressive inflammation in rat lungs to occur (TL_{Rat}). This TL_{Rat} is 0.2 mg/lungs, and the cumulative RCS exposure in rats that does not give rise to 'run away' inflammation and steeply increasing accumulation of silica is 500 mg/m^3.hr. A cumulative exposure metric of 500 mg/m^3.hr means the same load would be delivered by 1,000 mg/m^3 in 0.5 hours, or 250 mg/m^3 in 2 hours, etc. This cumulative exposure in rats is designated as CE_{Rat}. These values remain unchanged from SLR (2020).
- SLR (2020) converted the critical silica lung load in rats (TL_{Rat}) identified from the literature to a human equivalent lung load (TL_H) assuming similar sensitivity for rat and human lungs. The resulting TL_H was 51 mg/human lungs.

- In the review, this was adjusted for contributions from the health-based proposed time-weighted average (TWA) for RCS (L_{TWA}) taking into consideration pulmonary clearance and time to reach steady state critical load for humans (TL_{STEL}) (SLR 2020). In this report, if the comparison was to modelled incremental RCS data from the facility, the appropriate adjustment would instead be for general public exposure by adjusting for the total lung load of silica resulting from exposure to background concentrations of RCS in ambient air (TL_{Bckngd}); the left-over silica lung load allowable from other sources is termed $TL_{other\ sources}$. However, since the comparison in this preliminary HHRA is to total RCS measured in monitoring programs at Montrose, the above adjustment was not made. Instead the total critical human lung load (TL_H) was carried forward in the calculations.
- In line with the methodology in SLR (2020), the cumulative critical lung load for humans (TL_H) was matched with the POD cumulative rat exposure (CE_{Rat}) taking into account the physiological respiratory differences between rats and humans. The cumulative exposure in humans required to deliver the equivalent deposited amount of silica in human lungs (CE_H) as in rats is 60,690 mg/m^3.h as per the below equation:

$$CE_H = \left(\frac{TL_H x CE_{Rat}}{TL_{Rat}}\right) x\ RDDR$$

Where:
CE_H = Cumulative exposure in humans required to deliver equivalent deposited silica in human lung.
TL_H = Human equivalent critical lung load of silica. 51 mg/human lungs as per SLR (2020).
CE_{Rat} = Cumulative exposure in rats required to deliver critical amount of deposited silica in rat lung. 500 mg/m^3.h as per SLR (2020).
TL_{Rat} = Critical pulmonary target load required for progressive inflammation in rat lungs to occur. 0.2 mg/rat lungs as per SLR (2020).
RDDR = Regional Deposited Dose Ratio. 0.476 as per SLR (2020).

- Time to steady state is 2,310 calendar days as described in SLR (2020). Since exposure to the general public can occur 24 hours per day, this equates to 55,440 total 1-hour periods across which the cumulative exposure needs to be spread associated with the critical silica load in humans (CE_H).
- The human cumulative exposure (CE_H in mg/m^3.hr) was then corrected for the number of 1-hour periods in the number of days required to reach the critical steady state lung load in humans (i.e. 60,690 mg/m^3.h ÷ 55,440 hrs = 1.09 mg/m^3. This is the unfinished human equivalent POD prior to the application of uncertainty factors.
- The calculated unfinished POD was adjusted by the following uncertainty factors (UF): i) 10x as the standard default UF for use of a LOAEC rather than a NOAEC; ii) 3x for extrapolation from animals to humans, consistent with SLR (2020), and iii) 10x for potential differences in response sensitivity between people in the general population. The latter was modified from the factor of 3x in SLR (2020), as the STEL was for workers rather than the general population which could include very young, old, and infirmed persons that are potentially more vulnerable to exposure to chemicals. The composite UF of 300x was applied which results in an ambient air guideline equivalent concentration of 3.6 µg/m^3 (1.09 mg/m^3 ÷ 300 = 0.0036 mg/m^3 or 3.6 µg/m^3).

The resulting ambient air guideline value of 3.6 µg/m^3 derived via adaptation of the critical threshold cumulative lung silica burden from SLR (2020) should be applied to the 'respirable' (i.e. $PM_{2.5}$) fraction of crystalline silica in air as an annual average concentration.

3.3.4 Summary of available guidelines

The various guideline values for RCS summarised in previous sections are remarkably similar: 2 µg/m^3 from TCEQ (2009), 3 µg/m^3 from OEHHA (2004)/EPA VIC (2021), and 3.6 µg/m^3 from adaptation of the threshold cumulative lung silica burden from SLR (2020). Since the TCEQ (2009) and OEHHA (2004) guideline values are based on the same critical study, POD, and application of the same UF, it is considered appropriate to use the midpoint of the available guideline values (i.e. 3 µg/m^3, measured as an annual average in $PM_{2.5}$) in this assessment. This concentration is considered to be an airborne concentration of RCS to which an individual can be exposed for life without significant risk of harm.

4 Potential Exposure

4.1 Measured existing RCS concentrations

To inform this HHRA and the Air Quality Impact Assessment (SLR 2024), SLR undertook a baseline ambient air quality monitoring program for the Quarry. The monitoring was conducted between July 2022 and July 2023 and, in consultation with the Environmental Protection Authority Victoria (EPA VIC)[19], included the following at one location (shown in Figure 5):

- Continuous monitoring of PM_{10} and $PM_{2.5}$; and
- Batch monitoring of RCS (as $PM_{2.5}$).

SLR (2023) provides the details of the monitoring campaign and full results. The concentrations monitored at this location are considered to be generally representative of regional background plus influences from the existing operations at the Quarry (including quarrying, asphalt production and concrete batching) and from nearby industries to the north and west of the Quarry. Only the results for RCS are discussed in this report as they pertain to the risk assessment conducted herein.

Monthly 7-day (approximate) average RCS concentrations monitored between July 2022 and June 2023 are summarised in Figure 6. The estimated annual average concentration of RCS from the monitoring data was 0.2 µg/m^3. This concentration was used to represent existing RCS conditions in this assessment.

[19] Although not stated in SLR (2023), this location was chosen since it is generally downwind from the operations and the sensitive receptors (i.e. residences) near this location are the closest sensitive receptors to the Quarry. In addition, power could be made available for the monitor.

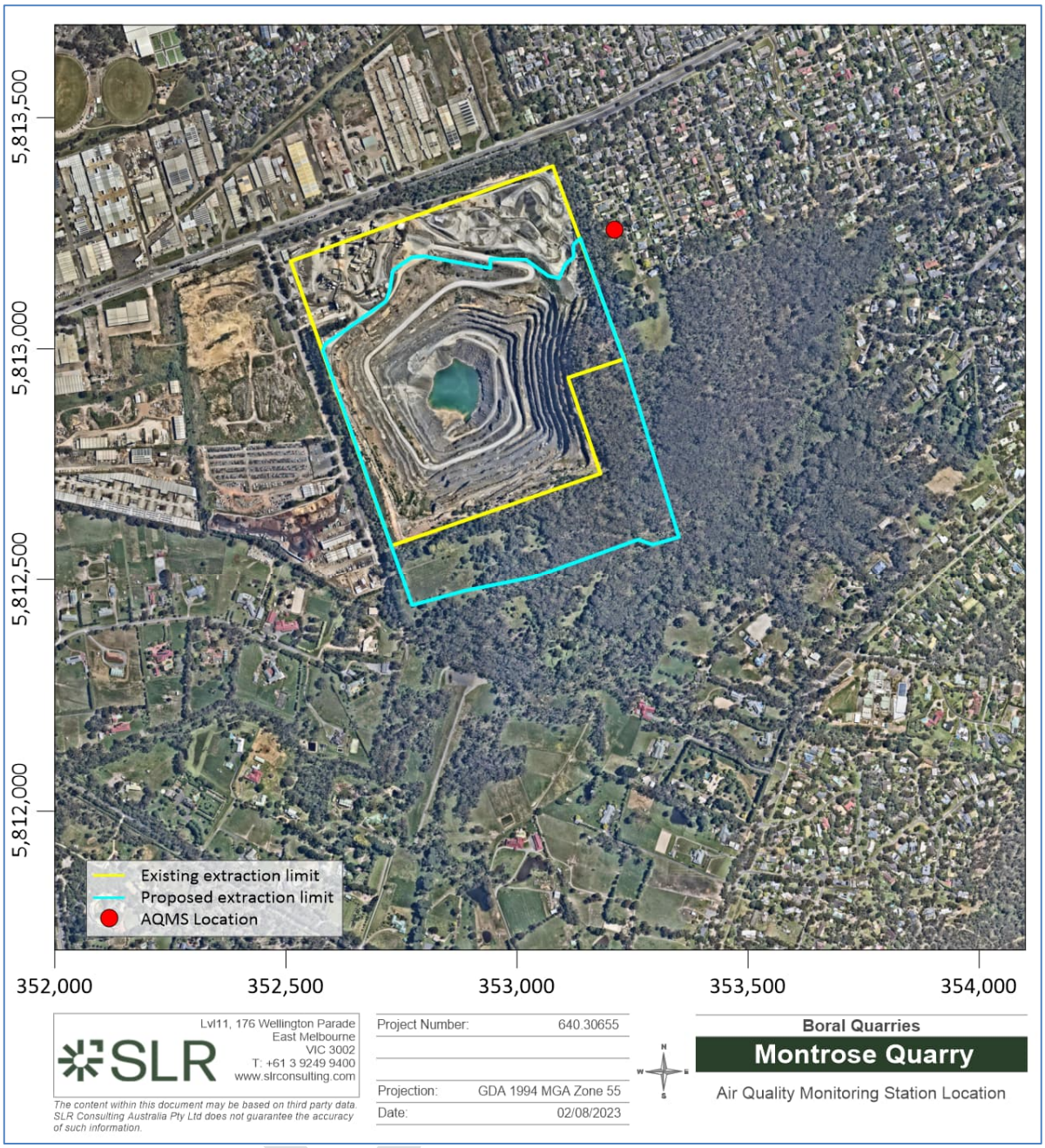

Figure 5 Location of Ambient Air Quality Monitoring in Relation to the Quarry (from SLR 2024).

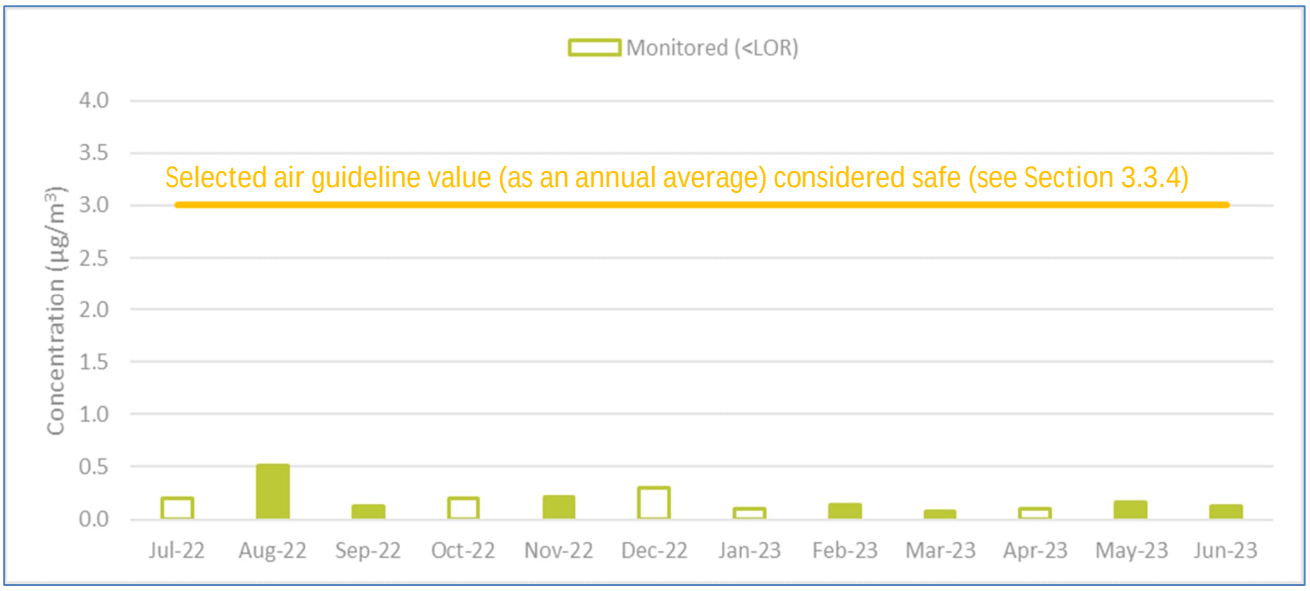

Figure 6 Monthly 7-day Average RCS Concentrations (July 2022 to June 2023) (adapted from SLR 2024).

4.2 Results of air quality modelling

SLR (2024) undertook air quality modelling for the proposed changes at the Quarry estimating emissions of dust as particles (PM_{10} and $PM_{2.5}$) and RCS. The estimates were undertaken for the following three scenarios:

- Scenario 0: Base Case – Existing Operations.
- Scenario 1: Project Year 5 – Quarry extension stage 3 (worst case construction).
- Scenario 2: Project Year 15 onwards – Quarry operation stage 7 and 8 (maximum operating conditions).

Predictions were undertaken for 33 nearby residences to the east and north-east, and the south and south-west of the Quarry (see Figure 7). The closest residence to the Quary (R12) is approximately 55m from the existing (and proposed) quarry extraction limit.

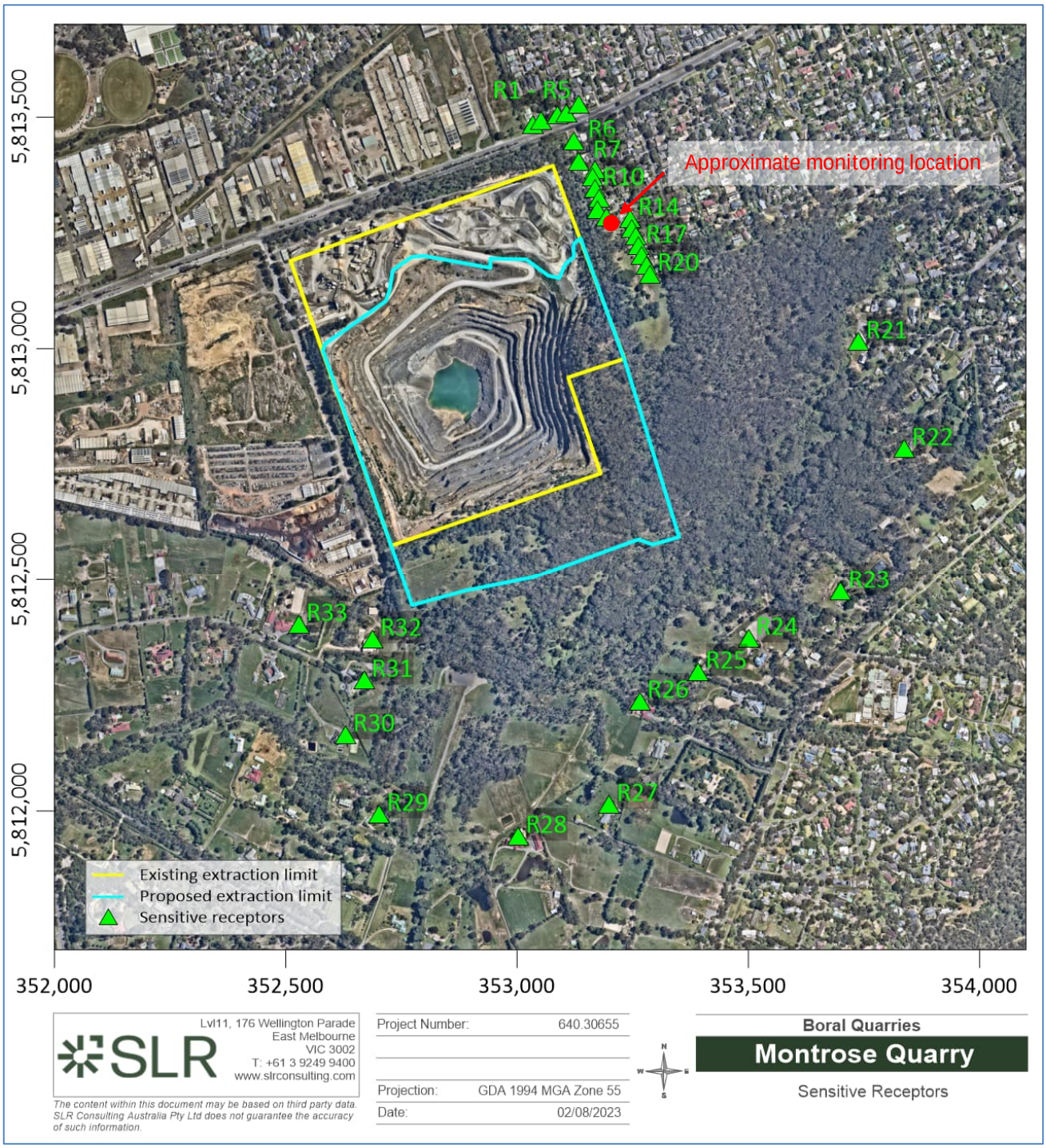

Figure 7 Identified Nearest Sensitive Receptors, i.e. Residences, to the Quarry Evaluated in Air Quality Impact Assessment (from SLR 2024).

The AQIA (SLR 2024) used the measured existing concentrations in conjunction with the predicted concentrations of RCS to estimate maximum cumulative RCS concentrations (for the existing situation plus the proposed changes). SLR (2024) notes the predictions may already include contributions from existing operations (Scenario 0), and therefore the cumulative predicted concentrations are likely over-predictions, i.e. they are conservative. For example, the modelled cumulative RCS concentration at R13 adjacent to the ambient air quality monitoring location was 0.3 µg/m^3 for Scenario 0, consisting of 0.1 µg/m^3 attributable to the Quarry and 0.2 µg/m^3 assumed background (i.e. the latter based on existing RCS concentrations measured at the monitoring location). In reality, the measured concentration at this location already incorporates the contribution from the existing Quarry operations.

The maximum predicted annual average cumulative RCS concentrations at any receptor (i.e. residence) over the 5 years modelled occurred at R1 for all three scenarios modelled. The maximum cumulative RCS concentrations were 0.5, 0.5, or 0.4 µg/m^3 for Scenarios 0, 1, and 2, respectively (SLR 2024). The maximum modelled cumulative concentration of 0.5 µg/m^3 has been used in this HHRA.

From an exposure point of view, it is important to use an overall average RCS concentration in the assessment since, at environmental exposure conditions, it is the long-term average exposure concentration that will most influence overall lung burden of RCS.

5 Risk Characterisation and Uncertainties

The potential risk of harm to human health of residents living in proximity to the Quarry as a result of RCS exposure in dust originating from the Quarry has been estimated by comparing the assumed current measured annual average RCS concentration and the potential future maximum modelled annual average RCS concentration at the maximum modelled receptor (i.e. residential) location bordering the Quarry (Section 4) with the concentration of RCS that an individual can be exposed to for life without significant risk of harm (Section 3.3.4). Also included for comparison is the maximum modelled cumulative annual average RCS concentration for the residence adjacent to the monitoring location (i.e. R13). Use of the maximum modelled cumulative RCS concentrations near the Quarry is conservative since these would be higher than at other residences. The results of the comparison shown in Table 2 show that the measured and modelled RCS emissions are markedly lower than the RCS concentration associated with no significant risk of harm, thus the risk of silicosis in the population living around Montrose Quarry due to RCS exposure from the Quarry is considered to be low.

Table 2 Comparison of current measured and potential future modelled RCS concentrations with RCS concentration which is without significant risk of harm (i.e. 'safe' RCS concentration)

Exposure Scenario	Exposure concentration (maximum annual average RCS in $PM_{2.5}$, µg/m^3)	'Safe' annual average concentration of RCS in $PM_{2.5}$, µg/m^3
Current conditions (i.e. measured)	0.2 (1)	3.0 (Section 3.3.4)
Maximum modelled future cumulative conditions (at R1)	0.5 (2)	
Maximum modelled future cumulative conditions (at R13)	0.4 (3)	

(1) From Section 4.1.
(2) From Section 4.2.
(3) From Appendix F, Table F20, in SLR (2024). R13 is adjacent to the monitoring location.

The following assumptions were made in this HHRA which potentially influence the overall certainty of the conclusions. Nevertheless, the assumptions made tend to err on the side of safety or conservatism, indicating that the conclusions likely over- rather than under-estimate the risk of harm to residents living in close proximity to the Quarry. The principal assumptions and uncertainties made in this HHRA include the following:

- There is currently no evidence that non-occupational exposure to RCS is associated with silicosis or lung cancer (the two endpoints of most concern with respect to RCS exposure). Nevertheless, adaptation of occupational exposures and experimental animal toxicology data has enabled derivation of guideline values for RCS in ambient air that are protective of health effects from chronic RCS exposure. These guidelines incorporate a number of uncertainty/safety factors which extrapolate the data, so it is protective of the general population. It is unlikely the guidelines used will fail to be protective of all or nearly all individuals. This is the very essence of the philosophy of creating public health guidelines. In addition, it instils confidence that all three guidelines discussed in this report are remarkably similar.
- The assessment has relied on the results of air monitoring and air modelling undertaken by SLR (2023, 2024). There is a high level of confidence that the information is unlikely to under-predict the outcomes, since:
    - The air monitoring location was selected and agreed upon after consultation with EPA VIC and corresponds with the location of the closest residences to the Quarry.
    - The air monitoring data was for a location coinciding approximately with the maximum modelled location in the AQIA.
    - The AQIA made a number of conservative assumptions which are more likely to over- rather than underestimate the modelled cumulative RCS concentrations (SLR 2024).
    - Data for the maximum modelled location out of any of the 33 modelled locations was used in the assessment.

6 Overall Conclusion

The risk of silicosis, and hence other health effects, due to RCS arising from the assumed current and proposed operations at Montrose Quarry is considered to be low. This is consistent with the overall consensus in the scientific literature that to date, there are no known adverse health effects associated with non-occupational exposure to RCS.

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