Section 3. Community profile

3.1 General

This section provides an overview of the community potentially impacted by the project. The key focus of the assessment presented is the local community, however some aspects of the assessment require consideration of statistics that are derived from larger populations, such as those within the Northern Area Health District and the greater Sydney Area. Hence, where relevant, information related to both the local community and other areas within Sydney (and NSW) have been presented.

3.2 Surrounding area and population

The Hornsby Quarry site is located in the suburb of Hornsby. The quarry is located in an area that is surrounded by bushland (immediately surrounding the quarry pit extending to the west) that is used for recreational purposes by the local community (including bushwalking and mountain biking), low density residential homes (to the northwest, north and south), Hornsby TAFE and other commercial/retail businesses (to the east). It is noted that Mt Wilga Private Hospital (rehabilitation hospital) is located to the north of the quarry.

Sensitive receivers

Sensitive receivers are locations in the local community where more sensitive members of the population, such as infants and young children, the elderly or those with existing health conditions or illnesses, may spend a significant period of time. These locations comprise hospitals, child care facilities, schools and aged care homes/facilities. The assessment of potential impacts on the surrounding community, particularly in relation to air quality, has considered all impacts at all individual residential homes and business in the vicinity of the quarry as shown on **Figure 3-1**.

Figure 3-1 Location of sensitive receivers

3.3 Population profile

The population within the area surrounding the quarry has been assumed to be consistent with the statistics available for the population of the larger suburb of Hornsby. No data is available for smaller areas within the suburb of Hornsby.

Tables 3-1 and 3-2 present a summary of a selected range of demographic measures relevant to the suburb of Hornsby with comparison to the statistical area of Hornsby South, greater Sydney and the rest of NSW (excluding greater Sydney).

Table 3-1 Summary of population statistics

Location	Total Population		% Population by Key Age Groups				
	Male	Female	0-4	5-19	20-64	65+	30+
Hornsby	9694	10169	7.2	15.7	65.5	11.6	62
Larger Statistical Areas							
Hornsby South (Statistical Area)	43701	46404	6.2	19.4	59.6	14.7	62
Greater Sydney	2162221	2229453	6.8	18.7	61.7	12.9	60
Rest of NSW (excluding greater Sydney)	1239007	1273942	6.3	19.7	55.9	18	63

Ref: Australian Bureau of Statistics, Census Data 2011

Based on this general population data, the suburb of Hornsby is generally similar to Greater Sydney and NSW.

Table 3-2 Selected demographics of population of interest

Location	Median age	Median household income ($/week)	Median mortgage repayment ($/month)	Median rent ($/week)	Average household size	Unemployment rate (%)
Hornsby	35	1436	2167	380	2.5	5.7
Larger Statistical Areas						
Hornsby South (Statistical Area)	38	1730	2383	400	2.8	5.2
Greater Sydney	36	1447	2167	351	2.7	5.7
Rest of NSW (excluding greater Sydney)	41	961	1560	220	2.4	6.1

Ref: Australian Bureau of Statistics, Census Data 2011

The social demographics of an area have some influence on the health of the existing population. The population located in the suburb of Hornsby is generally similar to that in Greater Sydney.

3.4 Existing health of population

3.4.1 General

The assessment presented in this report has focused on key pollutants that are associated with the project and include an assessment of particulate matter (namely $PM_{2.5}$ and PM_{10}). For these pollutants there are a large number of sources in the project area including other combustion sources (other than from the project), other local construction/earthworks and personal exposures (such as smoking) and risk taking behaviours that have the potential to affect the health of any population.

When considering the health of a local community there are a large number of factors to consider. The health of the community is influenced by a complex range of interacting factors including age, socio-economic status, social capital, behaviours, beliefs and lifestyle, life experiences, country of origin, genetic predisposition and access to health and social care. Hence, while it is possible to review existing health statistics for the local areas surrounding the project, and compare them to the Greater Sydney area and NSW, it is not possible or appropriate to be able to identify a causal source, particularly individual or localised sources.

Most of the health indicators presented in this report are not available for each of the smaller suburbs/statistical areas surrounding the site. Health indicators are only available from a mix of larger areas (that incorporate the study area) - the Northern Sydney Area Health Service and/or the combined area of Northern Sydney and the Central Coast. The health statistics for these larger areas (and in some cases data for the Greater Sydney area) are assumed to be representative of the smaller population located in the vicinity of the Hornsby Quarry site, given the similarity of the demographics of these populations to Greater Sydney.

3.4.2 Health-related behaviours

Information in relation to health-related behaviours (that are linked to poorer health status and chronic disease including cardiovascular and respiratory diseases, cancer, and other conditions that account for much of the burden of morbidity and mortality in later life) are available for large health population areas in Sydney and NSW. This includes risky alcohol drinking, smoking, consumption of fruit and vegetables, overweight and obesity and adequate physical activity. The study population is grouped in the larger population area of Northern Sydney and Central Coast. The incidence of these health-related behaviours generally indicates the population in the Northern Sydney and Central Coast area:

- Has similar rates of risky alcohol drinking, recommended consumption of vegetables and overweight and obesity compared with NSW.
- Has higher rates of recommended consumption of fruit and adequate physical activity compared with NSW.

3.4.3 Health indicators

In relation to some specific health indicators relevant for this assessment **Table 3-3** presents the available data for the slightly smaller population areas defined under the Northern Sydney Area Health and for the Hornsby, Ku-ring-gai and the Hills local government areas (or GP health areas). These have been compared with available data for Sydney and NSW. The health indicators include those that are specifically relevant to the quantification of exposure to particulate matter presented in **Section 4.4.**

Table 3-3 Summary of key health indicators

Health Indicator	Data available for Population (rate per 100,000 population)					
	Hornsby Shire	Ku-ring-gai Shire	The Hills Shire	Northern Sydney Area Health	Greater Sydney	NSW
Mortality						
All causes – all ages*	--	--	--	496.6[1]	586.9[1]	670#[2]
All causes ≥30 years*	--	--	--	--	--	1087#[2]
Cardiopulmonary ≥30 years*	--	--	--	--	--	490#[2]
Cardiovascular – all ages*	--	--	--	--	--	164#[2]
Respiratory – all ages*	--	--	--	--	--	57#[2]
Hospital admissions						
Coronary heart disease	539.5[3]	462.7[3]	597.5[3]	442.3[4]	391.6[4]	608.7[4]
COPD >65 years	647.9[3]	558.1[3]	735.6[3]	745.2[4]	1194.2[4]	1470.4[4]
Cardiovascular disease						
All ages	--	--	--	1642.3[5]	1582.6[5]	1949.9[5]
>65 years*	--	--	--			23352#[3]
Respiratory Disease						
All ages	--	--	--	1520.1[5]	1530.3[5]	1770.2[5]
>65 years*	--	--	--	--	--	8807#[3]
Asthma						
Asthma hospitalisations (ages 5-34 years)	--	--	--	85.7[4]	105.1[4]	133.6[4]
Current asthma for ages 16 and over	--	--	--	12.1%[4]	7.8%[4]	11.3%[4]

* Health indicators directly relevant to the characterisation of potential impacts associated with exposure to particulate matter as presented in **Section 4**
# Data provided by NSW Health (upon written request) for the purpose of this assessment.
All other data has been obtained from Health Statistics New South Wales
1 - Data from 2006-2007
2 – Data for 2005-2007
3 - Data for 2009-2011
4 – Data for 2010-2011
5 – Data for 2011-2012
-- No data available

In relation to asthma, **Figure 3-2** shows the general indicators reported for the larger population area of Northern Sydney and Central Coast compared with the data available for NSW.

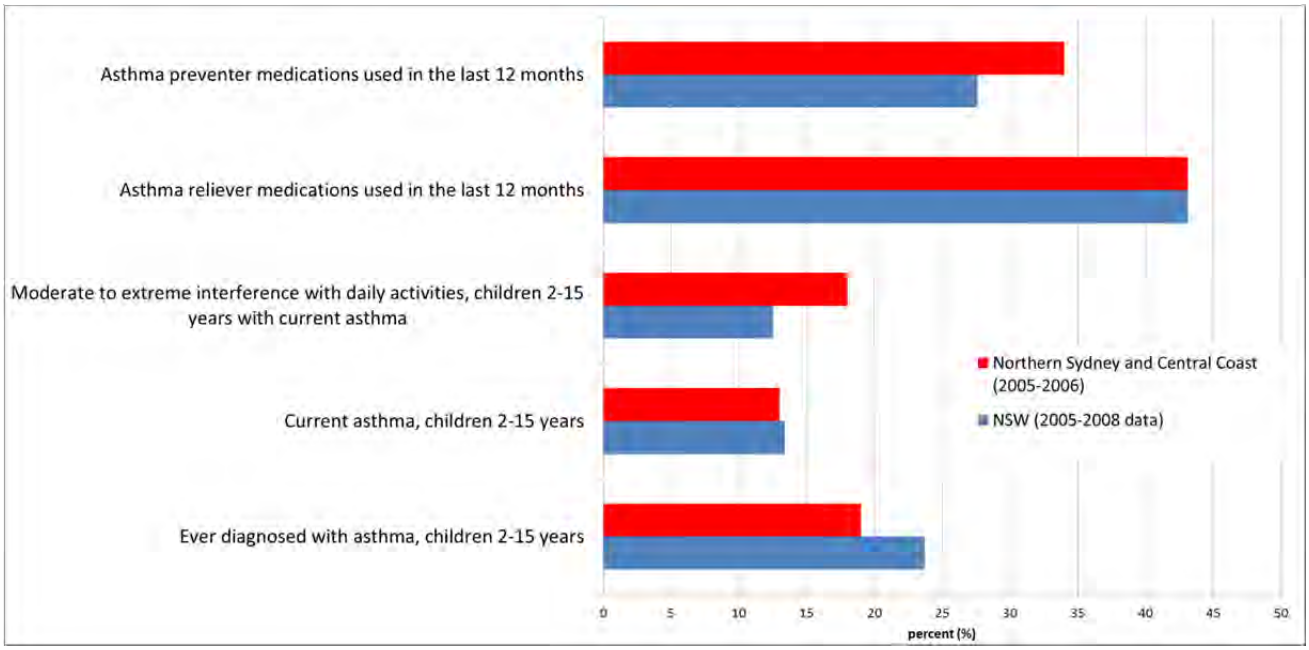

Figure 3-2 Summary of asthma prevalence and management (NSW and Northern Sydney/Central Coast)

Review of the available data generally indicates that for the population in the Northern Sydney area (including the Northern Sydney and Central Coast combined areas where relevant) the health statistics (including mortality rates and hospitalisation rates for most of these categories) are generally lower than compared with a number of other health areas and the whole of NSW.

For the assessment of potential health impacts from the project, where specific health statistics for the smaller population adjacent to the quarry is not available (and not reliable due to the small size of the population), adopting health statistics from the whole of NSW is considered to provide a representative, if not cautious (i.e. over estimating existing health issues), summary of the existing health of the population of interest.

Uncertainties

There are limitations in the use of this data for the quantification of impact and risk. This data is derived from statistics recorded by hospitals and doctors, reported by postcode of residence, and are dependent on the correct categorisation of health problems upon presentation at the hospital. There may be some individuals who may not seek medical assistance particularly with less serious conditions and hence there is expected to be some level of under-reporting of effects commonly considered in relation to morbidity. Quantitatively, the baseline data considered in this assessment is only a general indicator (not a precise measure) of the incidence of these health endpoints.

3.5 Existing environment

3.5.1 Existing air quality

The existing air quality in the study area is described in the technical working paper: air quality (AQIA) (AECOM, 2015). For this project the AQIA has used background air quality data collected as part of the NorthConnex Project for the year 2014 from the closest ambient air monitoring station at James Park.

It is noted that air quality in the greater Sydney area is most significantly affected by bushfires (including hazard reduction burns) and dust storms with transport-related emissions identified as the largest source of human-related pollution. In general, NSW is considered to have good air quality in relation to international standards. Review of $PM_{2.5}$ and PM_{10} in many countries by the WHO[1] identified that concentrations reported in Australia were low (amongst the lowest of all countries evaluated) compared with international levels.

Exceedances of the NEPC guidelines and advisory goals for particulate matter (PM) do occur in Sydney (as presented in the AQIA), primarily due to occasional bushfires, dust storms and hazard reduction burns rather than more every day conditions. In relation to $PM_{2.5}$, review of the sources (emissions) that contribute to the measured $PM_{2.5}$ reported in the Sydney area by the NSW EPA (based on emissions inventory data – for the year 2008, published 2012[2]), indicates that the most significant sources are household activities (including residential wood heaters – with peak emissions in the winter months from wood-smoke).

3.5.2 Existing noise environment

The existing noise environment in the area surrounding the Hornsby Quarry site is characterised by the local road network (including the Pacific Highway) and other transport infrastructure such as Hornsby railway station and the Northern rail line. Hence the main contributors to the existing noise environment are road traffic (including heavy vehicles using the existing road network) and passenger and freight rail movements. Commercial and light industrial areas along and around the Pacific Highway road corridor would also contribute to the local noise environment. Notwithstanding, the quarry site is located within a dense bushland area to the west of the Hornsby town centre and is surrounded by residential receivers to the south and north. It is expected that residential receivers in proximity to the quarry would experience a noise amenity consistent with a suburban setting (AECOM 2015).

Background noise monitoring has been undertaken at 6 locations in the study area that includes:

- 4 locations to establish background levels in areas considered representative of the local suburban area (with one of these locations also used to evaluate road noise); and

[1] WHO, Ambient (outdoors) air pollution in cities database 2014, available from http://www.who.int/phe/health_topics/outdoorair/databases/cities/en/

[2] http://www.epa.nsw.gov.au/woodsmoke/index.htm

- an additional 2 locations to evaluate road noise.

The background noise data is used to define appropriate construction noise management limits consistent with the NSW EPA Interim Construction Noise Guideline and NSW Road Noise Policy.

Background noise levels in the suburban area (4 locations sampled) were as follows:

- Day (7am to 6pm): rating background levels ranged from 34 to 39 dB(A) as $LA_{90,15}$
- Evening (6pm to 10pm): rating background levels range from 33 to 37 dB(A) as $LA_{90,15}$
- Night (10pm to 7am): rating background levels ranged from 31 to 33 dB(A) as $LA_{90,15}$

Section 4. Review of air impacts

4.1 Air impact assessment

4.1.1 Summary

Emissions to air associated with the project have been evaluated in detail within the technical working paper: air quality (AECOM 2015) (AQIA). The AQIA has considered emissions to air that may occur during all activities associated with the project.

The AQIA has considered the following emissions:

- The generation of dust during spoil stockpiling and emplacement activities. Dust-generating activities include the handling of material from haul trucks dumping spoil, transfer of spoil from temporary stockpiles to the conveyor and the distribution of spoil within the quarry void. Haulage routes within the site would be paved therefore wheel generated dust has not been considered as part of this assessment.
- Diesel exhaust emissions from truck haulage activities, mobile equipment and onsite generators would also result in the emission of combustion products including oxides of nitrogen, carbon monoxide, particular matter (PM), polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs).

Two scenarios have been modelled in the AQIA for the carrying out of the project as outlined in **Table 4-1**. As the timing of stockpiling and void filling is likely to be staggered Scenario 1 accounts for only stockpiling activities while Scenario 2 takes into account both stockpiling and void filling consecutively.

Table 4-1: AQIA Modelling Scenarios

Scenario	Scenario Description
Scenario 1	- Movement of up to 4,620 m^3/day of spoil from NorthConnex project construction site to the Hornsby Quarry site. - Storage of 170,000 m^3 of spoil in temporary stockpiles located on the flats within the eastern part of the Hornsby Quarry site.
Scenario 2	- Movement of up to 4,620 m^3/day of spoil from NorthConnex project construction site to the Hornsby Quarry site. - Storage of 170,000 m^3 of spoil to temporary stockpiles located on the flats within the eastern part of the Hornsby Quarry site. - Transport of up to 7,920 t/day of spoil from the temporary stockpiles via conveyor to the quarry floor. A volume of 1,000,000 m^3 of spoil would be used to fill the void.

In addition to the above scenarios, emissions derived from trucks using haulage route roads to and from the site have been assessed at sensitive receptors located close to the haulage route roads.

Scenario 2 results in the more significant levels of emissions and potential for air quality and health impacts. Hence this scenario has been the focus of further evaluation in this assessment.

Emissions related to the project have been assessed using CALPUFF (for the modelling of emissions from stockpiling and filling activities at the quarry) and CAL3QHCR (for the modelling of emissions from vehicles on surface roads around the project). Meteorological data collected from

NorthConnex monitoring stations and from the BOM and OEH monitoring stations used in the NorthConnex EIS assessment and terrain information relevant for the area was used to predict pollutant concentrations at 420 sensitive receiver locations surrounding the site.

Emissions associated with the spoil management activities have been based on Australian Government Department of Environment's, National Pollution Inventory (NPI) Emission Estimation Technique Manuals.

Vehicle emission rates have been based on internationally-recognised emission factors coupled with emission source types (diesel trucks and other equipment) and projected traffic volumes.

The AQIA has evaluated the key pollutants that are relevant to emissions to air during the project, which include:

- Particulate matter (PM) including size fractions PM_{10} and $PM_{2.5}$ which are of importance for the assessment of potential health impacts from the movement and handling of soil/rock and emissions from trucks.
- Oxides of nitrogen (in particular NO_2).
- Carbon monoxide (CO).
- Volatile organic compounds (VOCs) as total VOCs.
- Polycyclic aromatic hydrocarbons (PAHs, as total PAHs) which are particularly associated with diesel emissions.

Background levels of key pollutants (particulate matter, carbon monoxide and nitrogen dioxide) levels have been determined from available data on from the James Park NorthConnex monitoring station. The background air quality data is relevant for the assessment of cumulative impacts from the project.

Predicted impacts at all sensitive receiver locations have been provided for consideration in this assessment. The impacts have been presented as incremental impacts (i.e. the project only) and cumulative impacts (i.e. the project plus background air quality).

4.1.2 Mitigation measures

Given the proximity of the materials handling activities to the surrounding sensitive receptors and the quantity of material being transported onto the site, source based mitigation designed to reduce dust emissions through the application of specific measures, have been applied. The best practice measures that were assumed to be undertaken were:

- All site haul roads will be sealed.
- Water sprays will be used on the spoil stockpiles and conveyor transfer points.
- Water carts will wet down the material being worked by the roller and bulldozers.
- Wind barriers, such as shade cloth on boundary fencing, will be erected around the stockpile site perimeter.
- Management of speed limits on internal site haul roads.

4.1.3 Vehicle emissions

Diesel vehicles/equipment (considered in this project) emit a range of air pollutants that are known to be associated with adverse health impacts. Common air pollutants emitted from these vehicles include (DEH 2003):

- nitrogen oxides, in particular nitrogen dioxide;
- carbon monoxide;
- fine particulates;
- volatile organic compounds (in particular benzene, toluene and total xylenes [BTX] and 1,3-butadiene) and aldehydes (formaldehyde and acetaldehyde); and
- polycyclic aromatic hydrocarbons where the toxicity will vary depending on the presence of individual polycyclic aromatic hydrocarbons.

The assessment of emissions from vehicles requires consideration of key urban air pollutants (nitrogen oxides, carbon monoxide), the individual compounds likely to be present in the more general measures of volatile organic compounds (which include BTX, 1,3-butadiene and the aldehydes) and polycyclic aromatic hydrocarbons, and particulates. These are further discussed in the following sections.

4.2 Review of key air pollutants

4.2.1 Oxides of nitrogen

Nitrogen oxides (NOx) refer to a collection of highly reactive gases containing nitrogen and oxygen, most of which are colourless and odourless. Nitrogen oxide gases form when fuel is burnt. Motor vehicles, along with industrial, commercial and residential combustion sources, are primary producers of nitrogen oxides.

In Sydney, the OEH (2012) estimated that on-road vehicles account for about 62 per cent of emissions of nitrogen oxides, industrial facilities account for 12 per cent, other mobile sources account for about 22 per cent with the remainder from domestic/commercial sources.

In terms of health effects, nitrogen dioxide is the only oxide of nitrogen of concern (WHO 2000c). Nitrogen dioxide is a colourless and tasteless gas with a sharp odour. Nitrogen dioxide can cause inflammation of the respiratory system and increase susceptibility to respiratory infection. Exposure to elevated levels of nitrogen dioxide has also been associated with increased mortality, particularly related to respiratory disease, and with increased hospital admissions for asthma and heart disease patients (Morgan et al. 1998). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of nitrogen dioxide (NEPC, 2010). The health effects associated with exposure to nitrogen dioxide depend on the duration or exposure as well as the concentration; hence guidelines have been developed in Australia (and internationally) that reflect both acute and chronic exposures.

Guidelines are available from the NSW EPA and NEPC (NEPC 2003) that are based on protection from adverse health effects following short-term (acute) and longer-term (chronic) exposure. Review of these guidelines by NEPC (2010) identified additional supporting studies for the evaluation of potential adverse health effects and indicated that these should be considered in the current review of the National Ambient Air Quality NEPM (no interim or finalisation date available).

The air guidelines currently available from NEPC are consistent with health based guidelines currently available from the WHO (2005) and the USEPA (2010[3], specifically listed to be protective of exposures to sensitive populations including asthmatics, children and the elderly). On this basis the current NEPC guidelines are considered appropriate for the assessment of potential health impacts associated with the project.

Assessment of acute exposures:

The NEPC ambient air quality guideline for the assessment of acute (short-term) exposures to nitrogen dioxide relates to the maximum predicted total (cumulative) 1-hour average concentration in air. The guideline of 246 µg/m^3 (or 120 ppbv) is based on a lowest observed adverse effect level (LOAEL) of 409 to 613 µg/m^3 derived from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in children and aggravation of asthma. An uncertainty factor of two to protect susceptible people (i.e. asthmatic children) was applied to the LOAEL (NEPC 1998). On this basis the NEPC (and Environment Protection Authority) acute guideline is protective of adverse health effects in all individuals, including sensitive individuals.

The maximum 1-hour average concentration of nitrogen dioxide (cumulative concentration) predicted at the surrounding receptors is 139.8 µg/m^3, below the health based guideline of 246 µg/m^3. Hence there are no adverse health effects expected in relation to acute exposures to nitrogen dioxide in the local area surrounding the project. Hence no further detailed assessment of these exposures is warranted.

Assessment of chronic exposures:

The NEPC ambient air quality guideline for the assessment of chronic (long-term or lifetime) exposures to nitrogen dioxide relates to the maximum predicted total (cumulative) annual average concentration in air. The guideline of 62 µg/m^3 (or 30 ppbv) is based on a lowest observed adverse effect level (LOAEL) of the order of 40 – 80 ppbv (around 75-150 µg/m^3) during early and middle childhood years which can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent 'colds', a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school. An uncertainty factor of two was applied to the LOAEL to account for susceptible people within the population resulting in a guideline of 20-40 ppbv (38-75 µg/m^3) (NEPC 1998). On this basis the NEPC (and OEH) chronic guideline is protective of adverse health effects in all individuals, including sensitive individuals.

The maximum annual average concentration of nitrogen dioxide (cumulative concentration) predicted at the surrounding receptors is 22.7 µg/m^3, below the health based guideline of 62 µg/m^3. Hence there are no adverse health effects expected in relation to long-term/chronic exposures to nitrogen dioxide in the local area surrounding the project. Hence no further detailed assessment of these exposures is warranted.

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[3] Most recent review of the Primary National Ambient Air Quality Standards for Nitrogen Dioxide published by the USEPA in the Federal Register Volume 75, No. 26, 2010, available from: http://www.gpo.gov/fdsys/pkg/FR-2010-02-09/html/2010-1990.htm

4.2.2 Carbon monoxide

Motor vehicles are the dominant source of carbon monoxide in air (DECCW 2009). Adverse health effects of exposure to carbon monoxide are linked with carboxyhaemoglobin (COHb) in blood. In addition, association between exposure to carbon monoxide and cardiovascular hospital admissions and mortality, especially in the elderly for cardiac failure, myocardial infarction and ischemic heart disease; and some birth outcomes (such as low birth weights) have been identified (NEPC 2010).

Guidelines are available in Australia from NEPC (NEPC 2003) and NSW EPA (OEH) that are based on the protection of adverse health effects associated with carbon monoxide. Review of these guidelines by NEPC (2010) identified additional supporting studies[4] for the evaluation of potential adverse health effects and indicated that these should be considered in the current review of the National Ambient Air Quality NEPM (no interim or finalisation date available). The air guidelines currently available from NEPC are consistent with health based guidelines currently available from the WHO (2005) and the USEPA (2011[5], specifically listed to be protective of exposures by sensitive populations including asthmatics, children and the elderly). On this basis the current NEPC guidelines are considered appropriate for the assessment of potential health impacts associated with the project.

The NEPC ambient air quality guideline for the assessment of exposures to carbon monoxide has considered LOAEL (lowest observed adverse effect level) and NOAELs (no observed adverse effect level) associated with a range of health effects in healthy adults, people with ischemic heart disease and foetal effects. In relation to these data, a guideline level of carbon monoxide of nine ppmv (or 10 mg/m^3 or 10 000 µg/m^3) over an 8-hour period was considered to provide protection (for both acute and chronic health effects) for most members of the population. An additional 1.5 fold uncertainty factor to protect more susceptible groups in the population was included. On this basis the NEPC (and the Environment Protection Authority) guideline is protective of adverse health effects in all individuals, including sensitive individuals.

The Environment Protection Authority has also established a guideline for 15-minute average (100 mg/m^3) and 1-hour average (30 mg/m^3) concentrations of carbon monoxide in ambient air. These guidelines are based on criteria established by the WHO (WHO 2000b) using the same data used by the NEPC to establish the guideline (above) with extrapolation to different periods of exposure on the basis of known physiological variables that affect carbon monoxide uptake.

The maximum 1-hour average concentration of carbon monoxide (cumulative concentration) predicted at the surrounding receptors is 1983 µg/m^3, below the health based guideline of 30,000 µg/m^3. The maximum 8-hour average concentration of carbon monoxide (cumulative concentration)

[4] Many of the more current studies are epidemiology studies that relate to a mix of urban air pollutants (including particulate matter) where it is more complex to determine the effects that can be attributed to carbon monoxide exposure only.

[5] Most recent review of the Primary National Ambient Air Quality Standards for Carbon Monoxide published by the USEPA in the Federal Register Volume 76, No. 169, 2011, available from: http://www.gpo.gov/fdsys/pkg/FR-2011-08-31/html/2011-21359.htm

predicted at the surrounding receptors is 1086 µg/m^3, below the health based guideline of 10,000 µg/m^3. Hence there are no adverse health effects expected in relation to long-term/chronic exposures to carbon monoxide in the local area surrounding the project. Hence no further detailed assessment of these exposures is warranted.

4.3 Review of volatile organic compounds and polycyclic aromatic hydrocarbons

4.3.1 General

The AQIA has considered emissions of volatile organic compounds and polycyclic aromatic hydrocarbons to air from the project. Both volatile organic compounds and polycyclic aromatic hydrocarbons refer to a group of compounds with a mix of different proportions and toxicities. It is the individual compounds within the group that are of importance for evaluating adverse health effects. The composition of individual compounds in the volatile organic compounds and polycyclic aromatic hydrocarbons evaluated will vary depending on the source of the emissions. Hence it is important that the key individual compounds present in emissions considered for this project are speciated (i.e. identified and quantified as a percentage of the total volatile organic compounds or total polycyclic aromatic hydrocarbons) to ensure that potential impacts associated with exposure to these compounds can be adequately assessed.

Volatile organic compounds in air in Sydney (OEH 2012) are primarily derived from domestic/commercial sources (54 per cent) with on-road vehicles contributing around 24 per cent, industrial emissions eight per cent with the remainder from off-road mobile sources and other commercial sources.

Volatile organic compounds and polycyclic aromatic hydrocarbons from the project are associated with emissions from trucks and equipment used in the project. The makeup of the volatile organic compounds and polycyclic aromatic hydrocarbons emissions would depend on the mix of vehicles considered as these pollutants will be emitted in different proportions from petrol and diesel powered vehicles. In addition, the age and the fuel used by the vehicle fleet would affect these emissions. For the purpose of this assessment emissions are all assumed to be derived from heavy duty diesel vehicles.

4.3.2 Volatile organic compounds

Volatile organic compounds have been modelled in the AQIA based on emissions from all vehicles considered. The proportion of each of the individual volatile organic compounds that may be present in the air is then estimated based on the assumed composition of the vehicle fleet and the type of fuel used. Most of the VOC emissions comprise a range of hydrocarbons that are of low toxicity (such as methane, ethylene, ethane, butenes, butanes, pentenes, pentanes, heptanes etc) (EPA 2012). From a toxicity perspective the key volatile organic compounds that have been considered for the vehicle emissions are BTX, 1,3-butadiene, acetaldehyde and formaldehyde (consistent with those identified and targeted in studies conducted in Australia on vehicle emissions) (DEH 2003; EPA 2012).

The proportion of each of the key volatile organic compounds considered are derived from the 2008 Calendar Year Air Emissions Inventory for the Greater Metropolitan Region in NSW (EPA 2012), for heavy duty diesel vehicles, where the following mass fractions have been adopted (as % of total) VOCs):

- 1,3-Butadiene = 0.4%
- Acetaldehyde = 3.81%
- Benzene = 1.07%
- Formaldehyde = 9.86%
- Xylenes = 0.38%
- Toluene = 0.47%

4.3.3 Polycyclic aromatic hydrocarbons

Polycyclic aromatic hydrocarbons have been considered in the AQIA as key pollutants that may be derived from diesel powered heavy goods vehicles. The presence of polycyclic aromatic hydrocarbons in diesel exhaust has been found to be more a function of the polycyclic aromatic hydrocarbon content of the fuel than of engine technology. For a given refinery and crude oil, diesel fuel polycyclic aromatic hydrocarbon levels correlate with total aromatic content and T90 (distillation temperature where 90 per cent of the fuel is evaporated). Representative data on aromatic content for diesel fuels in Australia are limited, however, emissions tests have been conducted on a range of light and heavy vehicles under different traffic congestion conditions (DEH 2003). The data presented from these emissions tests is assumed to include fuels commonly used in Australia and are considered to provide an indication of the likely proportions of individual polycyclic aromatic hydrocarbons in diesel exhaust.

The polycyclic aromatic hydrocarbons reported in diesel exhaust by DEH (DEH 2003) comprise the 16 most commonly reported (and highest proportion) polycyclic aromatic hydrocarbons present in exhaust. The data available from this study is quite dated (from vehicles manufactured from 1990 to 1996) and use of this data is likely to provide an overestimation of polycyclic aromatic hydrocarbon emissions from current (and future) diesel vehicles. The evaluation of potential health impacts associated with exposure to polycyclic aromatic hydrocarbons from the project requires consideration of the 16 individual polycyclic aromatic hydrocarbons, present at the highest levels in exhaust and which have the most information on chronic health effects.

The toxicity of individual polycyclic aromatic hydrocarbons varies significantly, with some considered to be carcinogenic while others are not carcinogenic. For the carcinogenic polycyclic aromatic hydrocarbons, these are commonly assessed as a group with the total carcinogenic polycyclic aromatic hydrocarbon concentration calculated using weighting factors that relate the toxicity of individual carcinogenic polycyclic aromatic hydrocarbons to the most well studied polycyclic aromatic hydrocarbon, benzo(a)pyrene. For the carcinogenic polycyclic aromatic hydrocarbons the weighting factors presented by CCME (CCME 2010) have been adopted. Other polycyclic aromatic hydrocarbons that are not carcinogenic have been considered separately.

On the basis of this approach the speciation of individual polycyclic aromatic hydrocarbons (as per cent of total polycyclic aromatic hydrocarbons) has been calculated based on the data from DEH (2003) assuming most of the diesel vehicles will be operating in the local area under stop/start

conditions (relevant for diesel equipment on the site and stop/start activity of trucks driving on-site, unloading soil/rock and leaving the site. For the PAHs considered in this assessment the following mass fractions for each individual PAH have been adopted (as % total PAHs):

- Carcinogenic PAHs as benzo(a)pyrtene equivalents = 4.6%
- Naphthalene = 70%
- Acenaphthalene = 4.9%
- Acenaphthene = 2%
- Fluorene = 5%
- Phenanthrene = 3.4%
- Anthracene = 0.49%
- Fluoranthene = 0.45%
- Pyrene = 0.71%

4.3.4 Review of health impacts

The predicted (incremental) concentrations of individual volatile organic compounds and polycyclic aromatic hydrocarbons associated with the project (based on the speciation as outlined above) have been reviewed against published peer-reviewed health based guidelines that are relevant to acute and chronic exposures (where relevant). The health based guidelines adopted (identified on the basis of guidance from enHealth 2012) are relevant to exposures that may occur to all members of the general public (including sensitive individuals) with no adverse health effects. The guidelines available relate to the duration of exposure and the nature of the health effects considered where:

- Acute guidelines are based on exposures that may occur for a short period of time (typically between an hour and up to 14 days). These guidelines are available to assess peak exposures (based on the modelled 1-hour maximum concentration) that may be associated with volatile organic compounds in the air;
- Chronic guidelines are based on exposures that may occur all day, every day for a lifetime. These guidelines are available to assess long-term exposures (based on the modelled annual average concentration) that may be associated with volatile organic compounds and polycyclic aromatic hydrocarbons in the air.

Table 4-2 and **Table 4-3** present a summary of the maximum predicted 1-hour or annual average concentration with comparison against acute (**Table 4-2**) and chronic (**Table 4-3)** health based guidelines. The table also presents a Hazard Index (HI) which is the ratio of the maximum predicted concentration to the guideline. Each individual HI is added up to obtain a total HI for all the volatile organic compounds and polycyclic aromatic hydrocarbons considered. The total HI is a sum of the potential hazards associated with all the volatile organic compounds and polycyclic aromatic hydrocarbons together assuming the health effects are additive, and is evaluated as follows:

- A total HI ≤ 1 means that all the maximum predicted concentrations are below the health based guidelines and there are no additive health impacts of concern.

- A total HI > 1 means that the predicted concentrations (for at least one individual compound) are above the health based guidelines, or that there are at least a few individual volatile organic compounds or polycyclic aromatic hydrocarbons where the maximum predicted concentrations are close to the health based guidelines such that there is the potential for the presence of all these together (as a sum) to result in adverse health effects.

The following evaluation is based on the maximum predicted (incremental) concentration in air at any of the modelled receptor locations for Scenario 2 (which results in the highest maximum impact from any of the scenarios evaluated, including emissions from haulage roads). Concentrations at other receptors are lower and hence the tables present a worst-case evaluation only.

Table 4-2 Evaluation of potential acute impacts in local area (Scenario 2)

Key VOC	Proportion of total VOCs*	Health based acute guideline and basis (µg/m^3)	Maximum predicted 1-hour average concentration from Project** and Calculated HI	
			Maximum Concentration (µg/m^3)	HI
		Total VOCs	**64.3**	
Benzene	1.1%	**29^{A1} to 170^{T1}** (lower value adopted) A1: Acute guideline (1hr to 14 day exposure), based on immunological effects in mice. T1: Acute 1 hour health based guideline, based on depressed peripheral lymphocytes and depressed mitogen-induced blastogenesis (mice study)	0.69	0.024
Toluene	0.5%	**4500^{T2}** Acute 1 hour health based guideline, based on eye and nose irritation, increased occurrence of headache and intoxication in human male volunteers	0.30	0.000067
Xylenes	0.4%	**2200^{T3}** Acute 1 hour health based guideline, based on mild respiratory effects and subjective symptoms of neurotoxicity in human volunteers	0.24	0.00011
1,3-Butadiene	0.4%	**660^{O1}** Acute 1 hour health based guideline, based on developmental effects	0.26	0.00039
Formaldehyde	9.9%	**15^{T4}** Acute 1 hour health based guideline, based on eye and nose irritation in human volunteers	6.3	0.42
Acetaldehyde	3.8%	**470^{O2}** Acute 1 hour health based guideline, based on effects on sensory irritation, bronchoconstriction, eye redness and swelling	2.4	0.0052
			Total HI	0.45

Notes:

* Percentage of each individual volatile organic compound is based on a heavy diesel vehicles (refer to discussion above table)

** Concentrations presented for the maximum 1 hour average (as provided from the AQIA)

A1: Acute inhalation guideline (for exposures from 1 hour to 14 days) from review by ATSDR 2008 for benzene

T1: TCEQ 2007, Benzene, Development Support Document. Texas Commission of Environmental Quality, 1 hour average guideline value (include additional 3.3 fold safety factor). This acute guideline is lower than that derived by the OEHHA (based on older studies)

T2: TCEQ 2008, Toluene, Development Support Document. Texas Commission of Environmental Quality, 1 hour average guideline value (include additional 3.3 fold safety factor)

T3: TCEQ 2009, Xylenes, Development Support Document. Texas Commission of Environmental Quality, 1 hour average guideline value (include additional 3.3 fold safety factor)

T4: TCEQ 2008, Formaldehyde, Development Support Document. Texas Commission of Environmental Quality, 1 hour average guideline value (include additional 3.3 fold safety factor). This guideline is noted to be lower than the acute guideline available from the WHO (2000a, 2010) of 100 µg/m^3 for formaldehyde

O1: OEHHA 2013, Acute (1 hour average) guideline derived by the California Office of Environmental Health Hazard Assessment. The guideline developed is lower than developed by TCEQ (2008) based on the same critical study

O2: OEHHA 2008, Acute (1 hour average) guideline derived by the California Office of Environmental Health Hazard Assessment

Table 4-3 Evaluation of potential chronic impacts in local area (Scenario 2)

Key VOC	Proportion of total VOCs/ PAHs*	Health based chronic guideline and basis (µg/m^3)	Maximum predicted annual average concentration from Project** and Calculated HI	
			Max Conc. (µg/m^3)	HI
Total VOCs			**0.84**	
Benzene	1.1%	**1.7**[W1] Benzene is classified as a known human carcinogen by IARC. Chronic guideline based on excess risk of leukaemia	0.0090	0.0053
Toluene	0.5%	**5000**[U1] Chronic guideline based on neurological effects in an occupational study (converted to public health value using safety factors)	0.0039	7.9E-07
Xylenes	0.4%	**220**[A1] Chronic guideline based on mild subjective respiratory and neurological symptoms in an occupational study (converted to public health value using safety factors)	0.0032	0.000015
1,3-Butadiene	0.4%	**0.3**[U2] 1,3-Butadiene is classified by IARC as a probable human carcinogen. Chronic air guideline based on an excess risk of leukaemia	0.00336	0.011
Formaldehyde	9.9%	**3.3**[T1] Formaldehyde is classified by IARC as carcinogenic to humans. The guideline developed is based on the protection of all adverse effects including cancer and non-cancer (including short term effects)	0.0828	0.025
Acetaldehyde	3.8%	**9**[U3] Chronic guideline based on nasal effects (in a rat study) (converted to a public health value using safety factors)	0.0320	0.0036
Total PAHs			**2.1x10^{-4}**	
Naphthalene	70.0%	**3**[U4] Chronic guideline based on nasal effects (in a mouse study) (converted to a public health value using safety factors)	1.5 x10^{-4}	5.0x10^{-5}
Acenaphthylene	4.9%	200[U5S] / Refer to notes for ref U5	1.0 x10^{-5}	5.2E-08
Acenaphthene	2.0%	200[U5] / Refer to notes for ref U5	4.3 x10^{-6}	2.1 x10^{-8}
Fluorene	5.0%	140[U5] / Refer to notes for ref U5	1.1 x10^{-5}	7.6 x10^{-8}
Phenanthrene	3.4%	140[U5S] / Refer to notes for ref U5	7.2 x10^{-6}	5.2 x10^{-8}
Anthracene	0.5%	100[U5] / Refer to notes for ref U5	1.0 x10^{-6}	1.0 x10^{-8}
Fluoranthene	0.5%	140[U5] / Refer to notes for ref U5	9.6 x10^{-7}	6.8 x10^{-9}
Pyrene	0.7%	100[U5] / Refer to notes for ref U5	1.5 x10^{-6}	1.5 x10^{-8}
Benzo(a)pyrene TEQ	4.6%	**0.00012**[W2] BaP is classified by IARC as a known human carcinogen, which relates to BaP as well as all the other carcinogenic PAHs assessed as a BaP toxicity equivalent value. The chronic guideline is based on protection from lung cancer for an occupational study	9.8 x10^{-6}	0.082
			Total HI for VOCs and PAHs	**0.13**

Notes for Table 4-3:

*	Percentage of each individual volatile organic compounds and polycyclic aromatic hydrocarbons is based on a weighted average of emissions from the range of vehicle types proposed to be used on the project in 2019 and 2029, and for normal traffic flow or congested traffic flow (refer to discussion above table)
**	Concentrations presented for the annual average are as provided from the AQIA
A	Polycyclic aromatic hydrocarbon speciation data for normal traffic conditions – utilised in the assessment of scenarios 2a and 2b
W1:	WHO 2000 Air Quality Guidelines, value for benzene is based on non-threshold carcinogenic effects (excess lifetime risk of leukaemia). Guideline value based on incremental cancer risk of $1x10^{-5}$, consistent with guidance provided by NEPM (1999 amended 2013) and enHealth (2012)
W2:	WHO 2010 Guidelines for Indoor Air Quality, value for BaP is based on non-threshold carcinogenic effects from occupational study of coke workers (lung cancer is critical effect). Guideline value based on incremental cancer risk of $1x10^{-5}$, consistent with guidance provided by NEPM (1999 amended 2013) and enHealth (2012)
T1:	TCEQ 2008, Formaldehyde, Development Support Document. Texas Commission of Environmental Quality. The air guideline is derived on the basis of irritation of the eyes and airway discomfort in humans, with review of carcinogenic and other non-carcinogenic effects found to be adequately protected by this guideline. The guideline is more conservative than derived by the WHO (2010)
A1:	ATSDR 2007, Toxicological Profile for Xylene, chronic inhalation guideline derived is the most current robust evaluation
U1:	USEPA evaluation for toluene (most recently reviewed in 2005). This is the most current evaluation of effects associated with chronic inhalation exposure to toluene and is consistent with the value used to derive the NEPM (1999 amended 2013) health based guidelines
U2:	USEPA evaluation of 1,3-butadiene (most recently updated in 2002) with the chronic guideline adopted as the lower from the evaluation of non-threshold carcinogenic effects and non-cancer effects. This is the most conservative evaluation of this compound. A more recent review by TCEQ (2013) on the basis of the same critical studies as well as more current studies resulted in a higher chronic air guideline value.
U3:	USEPA evaluation of acetaldehyde (most recently updated in 1991). The guideline established is lower than more recent reviews undertaken by the WHO (2000) and the Californian OEHHA where less conservative evaluations are presented.
U4:	USEPA evaluation of naphthalene (most recently updated in 1998). The guideline established is and is consistent with the value used to derive the NEPM (1999 amended 2013) health based guidelines
U5:	Guideline available from the USEPA. Chronic guidelines for non-carcinogenic polycyclic aromatic hydrocarbons are based on criteria derived from oral studies (for critical effects on the liver, kidney and haematology) which are then converted to an inhalation value (relevant for the protection of public health, including the use of safety factors) for use in this assessment. The value presented in the above table has been converted from an acceptable dose in mg/kg/day to an acceptable air concentration assuming a body weight of 70kg and inhalation of 20 m^3/day (as per (USEPA 2009a))
U5S:	No guideline available for individual polycyclic aromatic hydrocarbon, hence a surrogate compound has been used for the purpose of screening. The surrogate compound is a polycyclic aromatic hydrocarbon of similar structure and toxicity. In relation to the surrogates adopted in this evaluation, acenaphthene has been adopted as a surrogate for acenaphthylene, fluoranthene has been adopted as a surrogate for phenanthrene

Review of the acute assessment presented in **Table 4-2** indicates that the maximum short-duration peak (1 hour average) concentrations of volatile organic compounds (assessed as the key individual volatile organic compounds and as a sum of all the individual volatile organic compounds) in air surrounding the site are below the relevant acute health based guidelines. On this basis no further detailed assessment of the peak emissions of volatile organic compounds from the project is warranted.

Review of the chronic assessment presented in **Table 4-3** indicates that the maximum long-term average (annual average) concentrations of volatile organic compounds and polycyclic aromatic hydrocarbons (assessed as the key individual volatile organic compound and polycyclic aromatic hydrocarbon compounds and as a sum of all the individual volatile organic compounds and polycyclic aromatic hydrocarbons) in air surrounding the site are below the relevant long-term (chronic) health based guidelines. These are guidelines that are based on the protection of public health for inhalation exposures all day (24 hours), every day (365 days per year) for a lifetime (at least 70 years). Hence comparison against these guidelines is conservative for a project that may occur for up to 3 years. On this basis no further detailed assessment of the emissions of individual volatile organic compounds and polycyclic aromatic hydrocarbons from the project is warranted.

4.4 Review of particulate matter

4.4.1 General

Particulate matter (PM) is a widespread air pollutant with a mixture of physical and chemical characteristics that vary by location (and source). Unlike many other pollutants, particulates comprise a broad class of diverse materials and substances, with varying morphological, chemical, physical and thermodynamic properties, with sizes that vary from <0.005 μm to >100 μm. Particulates can be derived from natural sources such as crustal dust (soil), pollen and moulds, and other sources that include combustion and industrial processes. Secondary particulate matter is formed via atmospheric reactions of primary gaseous emissions. The gases that are the most significant contributors to secondary particulates include nitrogen oxides, ammonia, sulfur oxides, and certain organic gases (derived from vehicle exhaust; combustion sources; agricultural, industrial and biogenic emissions).

Numerous epidemiological studies[6] have reported significant positive associations between particulate air pollution and adverse health outcomes, in particular mortality as well as a range of adverse cardiovascular and respiratory effects.

4.4.2 Particulate size and composition

The potential for particulate matter to result in adverse health effects is dependent on the size and composition of the particulate matter.

The size of particulates is important as it determines how far from an emission source the particulates may be present in air (with larger particulates settling out close to the source and smaller particles remaining airborne for greater distances) and also the potential for adverse effects to occur as a result of exposure.

The common measures of particulate matter that are considered in the assessment of air quality and health risks are:

[6] Epidemiology is the study of diseases in populations. Epidemiological evidence can only show that this risk factor is associated (correlated) with a higher incidence of disease in the population exposed to that risk factor. The higher the correlation the more certain the association. Causation (i.e. that a specific risk factor actually causes a disease) cannot be proven with only epidemiological studies. For causation to be determined a range of other studies need to be considered in conjunction with the epidemiology studies.

- **Total suspended particulates (TSP):** This refers to all particulates with an equivalent aerodynamic particle[7] size below 50 microns (µm) in diameter[8]. It is a fairly gross indicator of the presence of dust with a wide range of sizes. Larger particles (termed "inspirable", comprise particles around 10 microns (µm) and larger) are more of a nuisance as they will deposit out of the air (measured as deposited dust) close to the source and, if inhaled, are mostly trapped in the upper respiratory system[9] and do not reach the lungs. Finer particles (smaller than 10 µm, termed "respirable") tend to be transported further from the source and are of more concern with respect to human health as these particles can penetrate into the lungs. Hence not all of the dust characterised as total suspended particulates is relevant for the assessment of health impacts, and total suspended particulates as a measure of impact, has not been further evaluated in this assessment. The assessment has only focused on particulates of a size where significant associations have been identified between exposure and adverse health effects.
- **PM_{10}, particulate matter below 10 µm in diameter, $PM_{2.5}$, particulate matter below 2.5 µm in diameter and PM_1, particulate matter below 0.1 µm in diameter (termed ultrafine particles):** These particles are small and have the potential to penetrate beyond the body's natural clearance mechanisms of cilia and mucous in the nose and upper respiratory system, with smaller particles able to further penetrate into the lower respiratory tract[10] and lungs. Once in the lungs adverse health effects may result (OEHHA 2002). It is well accepted nationally and internationally that monitoring for PM_{10} is a good method of determining the community's exposure to potentially harmful dust (regardless of the source) and is most commonly measured in local and regional air quality monitoring programs. Smaller particulates such as $PM_{2.5}$ and PM_1, however, are of most significance with respect to evaluating health effects as a higher proportion of these particles penetrate deep into the lungs. Urban air, that has a significant contribution from combustion sources, tends to have a significant proportion of $PM_{2.5}$ and PM_1 in ambient air.

Evaluation of size alone as a single factor in determining the potential for particulate toxicity is difficult since the potential health effects are not independent of chemical composition. There are certain particulate size fractions that tend to contain certain chemical components, such as metals in fine particulates (<$PM_{2.5}$) and crustal materials (like soil) in the coarse mode (PM_{10} or larger).

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[7] The term equivalent aerodynamic particle is used to reference the particle to a particle of spherical shape and particle of density 1 g/cm^3

[8] The size, diameter, of dust particles is measured in micrometers (microns, µm).

[9] The upper respiratory tract comprises the mouth, nose, throat and trachea. Larger particles are mostly trapped by the cilia and mucosa and swept to the back of the throat and swallowed.

[10] The lower respiratory tract comprises the smaller bronchioles and alveoli, the area of the lungs where gaseous exchange takes place. The alveoli have a very large surface area and absorption of gases occurs rapidly with subsequent transport to the blood and the rest of the body. Small particles can reach these areas, be dissolved by fluids and absorbed.

In addition, different sources of particulates have the potential to result in the presence of other pollutants in addition to particulate matter. For example combustion sources, prevalent in urban areas, result in the emission of particulate matter (more dominated by $PM_{2.5}$) as well as gaseous pollutants (ozone, nitrogen dioxide, carbon monoxide and sulfur dioxide).

There is strong evidence to conclude (USEPA 2012; WHO 2003, 2013) that fine particles (< 2.5 µm, $PM_{2.5}$) are more hazardous than larger ones (coarse particles), primarily on the basis of studies conducted in urban air environments where there is a higher proportion (as a percentage of all particulates) of fine particulates and other gaseous pollutants present from fuel combustion sources, as compared to particulates derived from crustal origins. Toxicological and controlled human exposure studies indicate that primary particles generated from fossil fuel combustion processes may be a significant contributor to adverse health outcomes with several physical, biological and chemical characteristics of particles found to elicit cardiopulmonary responses. Amongst the characteristics found to be contributing to toxicity in epidemiological and controlled exposure studies are high organic carbon content, metal content, presence of polycyclic aromatic hydrocarbons, presence of other organic components or endotoxins and both small (< 2.5 µm) and extremely small size (< 1 µm) (USEPA 2009b; WHO 2003, 2006a). Where soil/rock is being handled the potential presence of and exposure to silica (as a fraction of particulates in air) also requires consideration.

A significant amount of research, primarily from large epidemiology studies, has been conducted on the health effects of particulates with causal effects relationships identified for exposure to $PM_{2.5}$ (acting alone or in conjunction with other pollutants) (USEPA 2012). A more limited body of evidence suggests an association between exposure to larger particles, PM_{10} and adverse health effects (USEPA 2009b; WHO 2003). The health effects identified from these studies has been specifically related to $PM_{2.5}$ or PM_{10} as these are the most commonly adopted robust and widespread measures of particulate matter available in urban air environments.

4.4.3 Health effects

Health effects that have been associated with exposure to PM_{10} and $PM_{2.5}$ relate to exposure over both the short term (hours or days where effects may occur on the same day or after a day or two) and long term (lifetime) and include (Anderson et al. 2004; NEPC 2010; OEHHA 2002; USEPA 2009b; WHO 2003, 2013):

- Respiratory and cardiovascular morbidity, such as aggravation of asthma, respiratory symptoms and an increase in hospital admissions.
- Mortality from all causes, and specifically cardiovascular and respiratory diseases and from lung cancer.

There is good evidence of the effects of short-term exposure to PM_{10} on respiratory health, but for mortality and cardiovascular effects the evidence of effects for PM_{10} exposure is weaker. For these health effects $PM_{2.5}$ (particles in the 2.5‑10 µm range) is a stronger risk factor (particles in the 2.5‑10 µm range).

In short-term studies (based on 24-hour particulate levels), groups with pre-existing respiratory, lung or heart disease, as well as elderly people, were more susceptible to the morbidity and mortality effects of ambient particulate matter exposure (Esworthy 2013; WHO 2013). In longer term studies it has been suggested that the socially disadvantaged and poorly educated populations respond more strongly in terms of mortality (Esworthy 2013; WHO 2003, 2013).

Based on the available studies, there is no evidence of a safe level of exposure or a threshold below which no adverse health effects occur (NEPC 2010; WHO 2013).

At present, at the population level, there is not enough evidence to identify differences in the effects of particles with different chemical compositions or emanating from various sources (NEPC 2010; WHO 2013). The evidence for the hazardous nature of combustion-related particulate matter (from both mobile and stationary sources that dominate urban air where most of the epidemiological studies are conducted) is more consistent than that for particulate matter from other sources, and dominate the epidemiological studies used to develop relationships between exposure and adverse health effects.

Particulates that are derived from specific sources, such as diesel emissions, are known to comprise other compounds such as volatile organic compounds and polycyclic aromatic hydrocarbons that are known to also be associated with adverse health effects. The presence of these other compounds has been addressed separately however the presence of these (and likely other compounds) compounds and other co-pollutants (also derived from combustion sources) adds to the complexity of utilising data from urban air epidemiological studies for assessing health effects from particulate matter.

Recently, outdoor air pollution has been classified by the International Agency for Research on Cancer (IARC 2013) as carcinogenic (Group 1) to humans based on sufficient evidence that exposure to outdoor air pollution causes lung cancer. Particulate matter, a major component of outdoor air pollution, was evaluated separately and also classified as carcinogenic to humans (Group 1).

In 2012, IARC evaluated exhaust from diesel engines (consisting mostly of particulate matter) and classified these emissions as carcinogenic (Group 1) to humans.

4.4.4 Assessment of potential health issues from exposure to particulate matter

General

For many of the key health effects associated with exposures to PM_{10} and $PM_{2.5}$ the exposure-response relationship is linear (where there is no threshold below which no adverse effects have been identified) (NEPC 2010). This means that any exposure to particulate matter has the potential to be associated with an effect. Guidelines have been established in Australia (and internationally) to determine a level at which cumulative exposure (i.e. exposure to particulates from all sources) are likely to minimise the potential for adverse impacts in a population. The available guidelines are discussed and further considered below.

However as there is no threshold for adverse effects it is also important that any incremental exposure to particulate matter derived from the project is also assessed.

Cumulative impacts

Air quality goals for PM_{10}, and advisory goal for $PM_{2.5}$, have been established by NEPC (NEPC 2002, 2003) that are based on the protection of human health and well-being. The goals apply to average or regional exposures by populations from all sources, not to localised “hot-spot” areas such as locations near industry, busy roads or mining. They are intended to be compared against ambient air monitoring data collected from appropriately sited regional monitoring stations.

In addition, the assessment of impacts from any development requires consideration of air quality goals/guidelines that are outlined in the Environment Protection Authority’s "Approved Methods for the Modelling and Assessment of Air Pollutants in NSW" (DEC 2005b). The guidelines are primarily derived from the NEPC, with the exception of an annual average PM_{10} guideline which is derived from older goals adopted by the Environment Protection Authority (EPA 1998). The air quality goals relate to total particulate matter burden in the air and not just the particulate matter from the project, hence use of these criteria requires consideration of background levels of particulate matter and other local sources. Similar to the NEPC criteria, these guidelines do not apply to localised “hot-spot” areas such as locations near industry, busy roads or mining. However, in the absence of alternative measures, Environment Protection Authority does apply these criteria to assess the potential for impacts to arise at such locations, particularly for new projects.

Table 4-4 presents a summary of the current NEPC and Environment Protection Authority’s air quality goals and guidelines for particulate matter.

Table 4-4 Air quality goals for particulates

Pollutant	Averaging period	Criteria	Reference
PM_{10}	24-hour	50 µg/m^3 Maximum of 5 days exceedance per year	(DEC 2005b; NEPC 2003)
	Annual	30 µg/m^3	(DEC 2005b)
$PM_{2.5}$	24-hour	25 µg/m^3	Advisory goal [11] (NEPC 2003)
	Annual	8 µg/m^3	

The AQIA presents a detailed assessment of cumulative PM_{10} and $PM_{2.5}$ impacts associated with the project. The assessment undertaken concluded the following in relation to Scenario 2:

[11] The $PM_{2.5}$ criteria established by the National Environment Protection Council are advisory goals. The goals have been derived on the basis of available health based information that relates exposure to $PM_{2.5}$ to adverse health effects. However, as $PM_{2.5}$ had not been routinely monitored in the community at the time when the criteria were being considered, existing urban (and regional) levels were not known, and the ability to meet the advisory goals could not be determined in individual states. Hence these criteria were not established as standards as defined in the National Environment Protection Council Act 1994. The relevance of any exceedance of these goals will be fully assessed once a sufficient database of monitoring data is available. They are, however, goals that are based on the protection of population health.

- For PM_{10}, one exceedance of the 24-hour average criteria is predicted, likely attributable to materials handling activities both on the flats and within the quarry void. No exceedances of the annual average PM_{10} criteria have been predicted.
- For $PM_{2.5}$, two exceedances of the 24-hour $PM_{2.5}$ advisory NEPM criterion were predicted, attributed to the high background concentration considered which exceeded the advisory level in isolation, as well as materials handling activities both on the flats and within the quarry void. In addition, exceedance of the annual average $PM_{2.5}$ advisory standard is predicted. It is noted that background concentrations contributed up to 95% of the advisory standards in the area.
- Both the predicted 24-hour and annual average PM_{10} and $PM_{2.5}$ concentrations could be minimised through a comprehensive dust management program to be implemented during the project.

Incremental impacts

Methodology

As there is no safe level for particulate matter in ambient air, the incremental impact of $PM_{2.5}$ and PM_{10} emissions to air from the project have been evaluated in more detail utilising the methodology outlined in **Appendix A**. The assessment of incremental impacts has addressed the following health endpoints:

Table 4-5 Health Impact Assessment Health Endpoints for Exposure to $PM_{2.5}$ and PM_{10}

Health Endpoint	Effect duration	Age group	Particulate fraction assessed
Primary health endpoints			
Mortality – all causes	Long-term	≥30 years	$PM_{2.5}$
Hospitalisations – Cardiovascular	Short-term	≥65 years**	$PM_{2.5}$
Hospitalisations – Respiratory	Short-term	≥65 years**	$PM_{2.5}$
Secondary health endpoints			
Mortality – all causes	Short-term	All ages	PM_{10}
Mortality all causes	Short-term	All ages	$PM_{2.5}$
Mortality – Cardiopulmonary	Long-term	≥30 years	$PM_{2.5}$
Mortality – Cardiovascular	Short-term	All ages	$PM_{2.5}$
Mortality – Respiratory	Short-term	All ages	$PM_{2.5}$
Lung cancer	Long-term	All ages	Diesel particulate matter (DPM)*

* Diesel particulate matter assumed to be 100% of $PM_{2.5}$ which is an overly conservative estimate as not all $PM_{2.5}$ will be derived from diesel emissions for this project for Scenario 2. Much of the $PM_{2.5}$ will be derived from dust generated from the handling and disposal of rock/soil. The emissions from haulage roads are assumed to all be from diesel trucks and hence the assumption adopted is less conservative.

** Not relevant for exposures in a workplace, only calculated for residential receptors and aged care facilities

Following this approach, the following information has been used to calculate an annual risk (on an individual basis) associated with specific primary and secondary health indicators (identified as robust associations related to exposure to $PM_{2.5}$ or PM_{10}):

- Estimates of the changes in particulate matter exposure levels (i.e. incremental impacts) due to the project (as provided by the AQIA). For the sensitive receivers considered in this assessment, the maximum incremental impacts associated with the project are as follows:
    - Scenario 2 – impacts from the site:

- the maximum annual average $PM_{2.5}$ incremental impact = 1.4 µg/m^3 which is located within Hornsby TAFE and 0.7 µg/m^3 for residential receptors with best practice mitigation measures
- the maximum annual average PM_{10} incremental impact = 2.4 µg/m^3 which is located within Hornsby TAFE and 1.1 µg/m^3 for residential receptors with best practice mitigation measures

o Scenario 2 – impacts from trucks on haulage route roads to/from the site:
- the maximum annual average $PM_{2.5}$ incremental impact = 0.14 µg/m^3
- the maximum annual average PM_{10} incremental impact = 0.14 µg/m^3

- Baseline incidence of the key health endpoints that are relevant to the population exposed (refer to **Section 3.4.3**);
- Exposure-response relationships expressed as a percentage change in health endpoint per µg/m^3 change in particulate matter exposure, where a relative risk (RR) is determined (refer to **Appendix A**); and
- Consideration of the duration of exposure for the assessment of health effects associated with long-term exposures:
  - Long-term effects on health are assessed using annual average exposure concentrations as an estimation of a long-term (lifetime) population exposure. Where the duration of the exposure to the impacts from the project is less than lifetime (as is the case for this project where it is expected to occur over a 33 month period only) the calculated long-term annual risk is adjusted to reflect the less than lifetime exposure. The adjustment relates to long-term exposures for populations over 30 years of age. Assuming a lifetime average of 82 years (enHealth 2012b) the adjustment factor relevant to a 33 month exposure (2.75 years) out of 52 years (>30 years of age) is 0.053.
  - Long-term effects from exposure to diesel particulate matter relates to incremental lifetime risk of lung cancer, based on the use of an inhalation unit risk. This relates to increased lifetime risks (averaged over a 70 year period, the duration of a lifetime over which carcinogenic effects are of importance) and hence for exposures that are less than a lifetime in duration the incremental risk can be calculated by accounting for the duration of exposure. For this calculation the duration of exposure is 2.75 years which results in an adjustment factor of 0.039 (when compared to a 70 year lifetime considered in the cancer risk calculation).

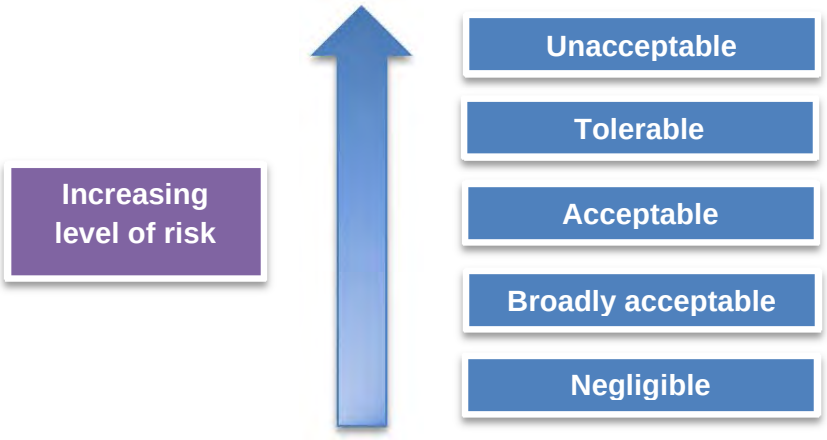
  It is noted that no adjustment for the exposure duration can be included where short-term effects of exposure to particulates is assessed. The short-term effects calculation relates to effects that may occur on the day or shortly after the short-term (daily) change in exposure. The calculation undertaken for these effects at any individual receptor is based on daily risks that are then summed over each day for a year to obtain an annual risk. This risk is relevant for the period of exposure.

The calculations undertaken provide an annual risk for individuals exposed to increased PM emissions from the project at specific locations (such as the maximum, or at specific sensitive receiver locations).

Acceptability of Risk

The acceptability of incremental risks associated with the impacts evaluated in this assessment is the subject of some discussion in relation to exposures to particulate matter. More specifically there are no guidelines available that relate to an acceptable level of risk for a small population (associated with impacts from a specific activity or project).

While various terms have been applied, it is clear that the two ends of what is a spectrum of risk are the “negligible” level and the “unacceptable" level. Risk levels intermediate between these are frequently adopted with varying terms often used to describe the levels. When considering a risk derived for an environmental impact it is important to consider that the level of risk that may be considered acceptable will lie somewhere between what is negligible and unacceptable, as illustrated below.



It is not always possible to provide a rigid definition of acceptable risk due to the complex and context-driven nature of any particular project. However for the purpose of this assessment the following have been considered:

- A level less than 10^{-6} (one chance in a million) is considered to be a level of increased risk that would be considered as a negligible risk in the community.
- A level in excess of 10^{-4} for increased risk (one chance in 10,000) is considered to be unacceptable. This level of risk has been generally adopted by health authorities as a point where risk is considered to be unacceptable in the development of drinking water guidelines (that impact on whole populations) (for exposure to carcinogens as well as for annual risks of disease (Fewtrell & Bartram 2001)) and in the evaluation of exposures from pollutants in air (DEC 2005a).
- Between an increased risk level considered negligible (10^{-6}) and unacceptable (10^{-4}) lie risks that may be considered to be tolerable or even acceptable. Tolerable risks are those that can be tolerated (and where the best available, and most appropriate, technology has been implemented to minimise exposure) in order to realise some benefit.

When considering the impacts associated with this project, it is important to consider the health effects being considered and the size of the population exposed. In addition it is important to note that there are a range of benefits associated with the project.

Risk Calculations

On the basis of the approach outlined above, and for the key health endpoints considered in relation to exposure to $PM_{2.5}$ and PM_{10} (derived from the project), incremental risks have been calculated based on data from the AQIA. The calculations have been undertaken for the maximum predicted concentrations at all the sensitive receivers considered.

Table 4-6 presents present a summary of the predicted increased annual risks relevant to the primary and secondary health indicators addressed in this assessment.

The calculations presented in these tables are considered accurate to one significant figure only due to the level of uncertainty within all aspects of the assessment presented.

Table 4-6 Incremental Annual Risks – Exposure to $PM_{2.5}$ and PM_{10}

Health Endpoint	Effect duration	Age group	PM fraction assessed	Risks for Scenario 2 – Best Practice Mitigation			
				Maximum impacts (TAFE)	Maximum impacts (residential)	Average over all sensitive receptors	Maximum Community exposures from haulage roads
Primary health endpoints							
Mortality – all causes	Long-term	≥30 years	$PM_{2.5}$	$5x10^{-6}$	$2x10^{-6}$	$7x10^{-7}$	$5x10^{-7}$
Hospitalisations – Cardiovascular	Short-term	≥65 years*	$PM_{2.5}$	NA	$1x10^{-4}$	$4x10^{-5}$	$3x10^{-5}$
Hospitalisations – Respiratory	Short-term	≥65 years*	$PM_{2.5}$	NA	$2x10^{-5}$	$7x10^{-6}$	$5x10^{-6}$
Secondary health endpoints							
Mortality – all causes	Short-term	All ages	PM_{10}	$1x10^{-5}$	$4x10^{-6}$	$2x10^{-6}$	$6x10^{-7}$
Mortality all causes	Short-term	All ages	$PM_{2.5}$	$9x10^{-6}$	$4x10^{-6}$	$1x10^{-6}$	$9x10^{-7}$
Mortality – Cardiopulmonary	Long-term	≥30 years	$PM_{2.5}$	$5x10^{-6}$	$2x10^{-6}$	$7x10^{-7}$	$5x10^{-7}$
Mortality – Cardiovascular	Short-term	All ages	$PM_{2.5}$	$2x10^{-6}$	$1x10^{-6}$	$3x10^{-7}$	$2x10^{-7}$
Mortality – Respiratory	Short-term	All ages	$PM_{2.5}$	$1x10^{-6}$	$8x10^{-7}$	$2x10^{-7}$	$1x10^{-7}$
Lifetime cancer risk							
Lung cancer	Long-term	All ages	DPM	$2x10^{-6}$	$9x10^{-7}$	$3x10^{-7}$	$2x10^{-7}$

* Not relevant for exposures in a workplace, only calculated for residential receptors and aged care facilities

Based on the calculations presented above the following can be noted:

- Calculated risks associated with exposures to PM_{10} and $PM_{2.5}$ (including DPM) from emissions from haulage route roads are considered to be tolerable and for many health endpoints the risks are considered to be negligible.

- For Scenario 2, where best practice mitigation measures have been considered for the project the following can be noted:
    - Maximum risks associated with exposure to PM_{10} and DPM are less than 1 x 10^{-5} and are considered to be low.
    - Maximum risks associated with exposure to $PM_{2.5}$ typically lie in the range of <1x10^{-6} to 1x10^{-4} and are generally considered to be tolerable. It is noted that the maximum level of risk calculated related to potential cardiovascular hospitalisations for individuals aged 65 years and over associated with short-term changes in $PM_{2.5}$ exposures. For the maximum risk calculation this relates to the maximum at a specific residential receptor where the age and existing health status of the residents is not known. The calculated risk specifically relates to individuals aged 65 years of age and over residing at this location with pre-existing cardiovascular disease. As the population evaluated in this assessment is small (with the impacts limited to areas located immediately adjacent to the quarry and access road) the calculated impacts are not considered to be of significance (i.e. would not result in measurable effects in the population evaluated).

The calculated risks are in the range considered to be negligible to tolerable, where the available guidance requires that mitigation measures be implemented to minimise exposures associated with the project. For this project, air impacts and hence health risks are proposed to be minimised through the implementation of best practice mitigation measures and a comprehensive dust management plan. Such measures are described in detail in the AQIA and would result in lower levels of exposure and risk at all receptors in the surrounding community.

4.4.5 Silica Exposures

Silica is abundant in the earth's crust and is released as fine particulates whenever rock is disturbed. Silica can occur both in crystalline and amorphous forms. There are several crystalline forms: quartz, cristobalite and tridymite; while the amorphous form occurs mainly as diatomaceous earth resulting from deposition of the exoskeletons of living organisms. It is generally only the crystalline forms which are fibrogenic which can cause inflammation resulting in scarring and progressive reduction in lung capacity for which there is no effective treatment (silicosis). The risk and the severity of damage are related to the size and shape of the particles, the concentration of particles and the length of time the person is exposed. Silicosis can only be caused by exposure to crystalline silica particles which are in the respirable size range. This is why only PM_{10} and $PM_{2.5}$ impacts are further reviewed in relation to silica exposures. The International Agency for Research on Cancer (IARC) has classified crystalline silica as carcinogenic to humans.

In relation to this project an assessment of potential silica exposures has been undertaken. The assessment has first determined the likely percentage of crystalline silica that may be present in the PM_{10} and $PM_{2.5}$ impacts predicted. The assessment has then compared the potential silica air concentrations against criteria established that are based on the protection of community health.

The project involves the handling and placement of rock/soil extracted from the construction of the NorthConnex project. This material largely comprises sandstone materials that are rich in quartz and hence there is the potential for community exposures to crystalline silica that will be present in the dust generated. The silica content of Sydney sandstone is reasonably well studied, with published quartz contents between 60% and 70% for "yellow block" sandstone and 70% to 80% for quartz rich (whiter) sandstone which are the materials likely to be extracted during the construction of the tunnel. From these materials an average quartz content of 70% can be assumed.

A number of guidelines are available for occupational exposures to crystalline silica (which is where most exposure occurs and where adverse health effects have been identified). In relation to non-occupational exposures the WHO (WHO 2000a) states "there are no known adverse health effects associated with the non-occupational exposures to quartz".

The guidelines that are available in relation to non-occupational exposures relate to concentrations of crystalline silica present in various particle fraction sizes, In relation to $PM_{2.5}$ and PM_{10} criteria based on the protection of community health are available from the WHO (WHO 2000a) and in guidance from the Victorian EPA (EPA Victoria 2007).

It is noted that these criteria relate to the total community exposure to crystalline silica in air and hence background levels (i.e. the levels normally found in air) also need to be considered. No data has been collected for the area evaluated in this assessment however data is available from the Hunter Valley which has been assumed to be representative (and conservative) for the study area.

Table 4-7 presents a summary of the maximum predicted incremental $PM_{2.5}$ and PM_{10} impacts from the project (for Scenario 2 at the TAFE as well as the maximum residential receptor), the calculated silica concentration in these fractions, the adopted background concentration of silica in air, the total silica concentration and the relevant guidelines.

Table 4-7 Assessment of Silica Impacts

Aspect of assessment	Scenario 2 – Maximum (TAFE)		Scenario 2 – Maximum Residential	
	$PM_{2.5}$	**PM_{10}**	**$PM_{2.5}$**	**PM_{10}**
Incremental dust impact (annual average) µg/m^3	1.4	2.4	0.7	1.1
Incremental silica concentration, µg/m^3	0.98	1.7	0.49	0.77
Background silica concentration#, µg/m^3	0.61	1.9	0.61	1.9
Total silica exposure concentration, µg/m^3	**1.6**	**3.6**	**1.1**	**2.7**
Guidelines				
Victoria EPA*, µg/m^3	3		3	
WHO**, µg/m^3		8		8

Background levels based on measured respirable crystalline silica levels (for the fraction sizes) in the Hunter Valley (potentially conservative for the Hornsby area where ambient particulates are expected to dominated by combustion sources rather than crustal sources [as may be the case for the Hunter Valley]) (Morrison et al)

* Guideline available in the Protocol for Environmental Management: Mining and Extractive Industries. The criteria relates to respirable crystalline silica as $PM_{2.5}$ fraction size and is derived from the California EPA Office for Environmental Health Hazard Assessment (OEHHA) Reference Exposure Level (REL) (OEHHA 2005). It is noted that the OEHHA REL was derived for PM_4 and has been adopted by the Victorian EPA for $PM_{2.5}$.

** Guideline for respirable crystalline quartz as PM_{10}, based on lifetime exposures resulting in silicosis risks of less than 3% in a healthy population, and considered to be consistent with ambient levels of risk. The use of such a guideline is highly conservative for the assessment of silica exposures over a 33 month period, rather than a lifetime (taken to be 70 years for the assessment of cancer risk).

Review of the above table indicates that the potential respirable silica concentrations in air derived from the project are below health based criteria.

US EPA Silicosis Potency Estimates

The US EPA (1996) examined the non-cancer epidemiological literature on crystalline silica induced diseases. From the extensive data available, which examined the medical histories of thousands of miners, they concluded that the cumulative risk of developing silicosis is zero for cumulative exposures of less than 1000 μg/m^3.years.

Cumulative exposure is the average respirable crystalline silica concentration a person is exposed to over a period of time, multiplied by the number of years exposed. For example, an exposure of 1000 μg/m^3.years, would be experienced by an individual exposed to 14.3 μg/m^3 per year for 70 years. For cumulative exposures less than 1000 μg/m^3.years, the US EPA concludes that the risk of developing silicosis is zero.

For this project, the following has been calculated:

- The total respirable crystalline silica concentration (as annual average $PM_{2.5}$) is 1.6 μg/m^3
- The duration of the project is 33 months, which is 2.75 years
- The total silica exposure associated with the project is calculated to be 4.4 μg/m^3.years
- Silica exposures relevant to the remaining 75.25 years (assuming a lifetime of 78 years) to ambient/background levels of 0.61 μg/m^3 is 46 μg/m^3.years
- Total lifetime exposure from background plus the project is calculated to be 50.4 μg/m^3.years

The total cumulative risk of silicosis is well below 1000 μg/m^3.years and is therefore considered to be zero.

Overall assessment

Hence no adverse health impacts are expected as a result of exposure to silica emissions from the project.

4.5 Uncertainties

The modelling of particulate impacts involves the use of a number of assumptions in relation to the carrying out of the project and activities that result in the emission of dust to air. In addition, determining the dispersion of particulate matter from the project to the surrounding environment has utilised air dispersion models. While the approach adopted in the AQIA utilised published peer-reviewed emission estimation techniques, the currently available site-specific data on the carrying out of the project, site-specific meteorology and terrain data and approved models for the quantification of impacts in the surrounding areas, the overall approach adopted is generally conservative to ensure that where uncertainties are present, the impact is overestimated.

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Section 5. Review of noise and vibration impacts

5.1 Overview of the noise and vibration assessment

5.1.1 General

This section presents a summary of the technical working paper: noise and vibration (AECOM, 2015) (NVTP) that relates to the project. The assessment has been reviewed to determine if the predicted impacts have the potential to affect the health of the surrounding community and, if impacts are predicted, if they can be effectively mitigated.

The NVTP provides a more detailed evaluation of all the activities, and the duration of those activities, that may give rise to noise or vibration impacts in the surrounding community.

To undertake the noise assessment required for the project, the existing background noise quality is required as the guidelines that relate to noise impacts from a specific project are based on levels allowable above background (refer to **Section 3.5.2** for further detail). Background noise levels were measured at 4 locations in the study area, at locations considered representative of the local suburban area. Two additional roadside locations were monitored to determine existing road traffic noise levels. Noise assessment criteria were then established addressing activities in the quarry as well as road traffic.

Noise levels that are measured, or modelled, refer to noise levels over a specified period of time and are presented as L_{A1}, L_{A10}, L_{A90}, L_{Amax} and L_{Aeq} levels of the noise environment. The L_{A1}, L_{A10} and L_{A90} levels are the levels exceeded for one percent, 10 per cent and 90 per cent of the sample period respectively. The L_{Amax} is indicative of maximum noise levels due to individual noise events. The L_{A90} is taken as the rating background noise level (RBL). The L_{Aeq} is the energy averaged noise level over a defined period.

5.1.2 Noise assessment criteria

Noise issues in NSW are managed by the NSW Environment Protection Authority (EPA). The EPA has prepared a number of guidance documents with regard to the types of noise that are considered in relation to the project. The NSW Road Noise Policy (RNP) (NSW DECCW 2011) and the Interim Construction Noise Guideline (ICNG) (NSW DECC 2009) are relevant to the assessment of noise generated by this project. In these policies there is discussion of the need to balance the economic and social benefits of activities that may generate noise with the protection of the community from the adverse effects of noise. The noise assessment criteria adopted relate to levels of noise that can be tolerated or permitted above background before some adverse effect (annoyance, discomfort, sleep disturbance or complaints) occurs.

For the assessment of noise impacts from the project a range of guidelines and criteria have been adopted:

Construction noise

The ICNG has been adopted for the assessment of noise generated by the project. In relation to these guidelines, noise impacts from the project are predicted at sensitive receivers and compared with the criteria, referred to as management levels, outlined in the ICNG. Where an exceedance occurs, the guidelines advise that the proponent apply all feasible and reasonable work practices to minimise impacts. The management levels are based on levels of noise above background that may result in reactions (or complaints) by the community. The levels are based on some reaction (noise affected) and a strong reaction (highly noise affected).

During standard work hours (Monday to Friday 7am to 6pm and Saturday 8am to 1pm) the construction noise management level is set at the background level plus 10 dB(A). Where construction noise levels reach 75 dB(A) residential receivers can be considered as 'highly noise affected' and the proponent should, in consultation with the community, consider restricting hours to provide respite periods.

Levels of noise allowable outside standard work hours, particularly at night, are lower. For works undertaken outside of standard work hours the construction noise management level is set at the background level plus 5 dB(A). It is noted that no works outside of standard work hours are proposed as part of the project.

For non-residential areas noise management levels are outlined in the ICNG.

The assessment of noise impacts from the project has been undertaken based on 4 noise catchment areas (assumed to have background noise levels consistent with the background noise monitoring location within that catchment area).

Construction Road Noise

The ICNG does not provide direct reference to an appropriate criterion to assess the noise arising from construction traffic on public roads. However, as the RNP is the current state policy document for assessing road traffic noise and in the absence of an alternative guidance it has been used to assess the noise arising from construction traffic movements generated by the project. The RNP does not require assessment of noise impact to commercial or industrial receivers. Based on this guidance the external noise criteria (applied 1 m from the external façade of a building) is set at:

- 60 dB(A) $LA_{eq,15hr}$ between 7 am and 10 pm and 55 dB(A) $LA_{eq,9hr}$ between 10 pm and 7 am for arterial/sub-arterial roads.
- 55 dB(A) $LA_{eq,15hr}$ between 7 am and 10 pm and 50 dB(A) $LA_{eq,9hr}$ between 10 pm and 7 am for local roads (including Bridge Road).

It is noted that as no works or haulage to the site is proposed outside of standard work hours the night time criteria (10 pm to 7 am) are not applicable to the project.

In cases where existing traffic noise levels are above the noise assessment criteria, the primary objective is to reduce these through feasible and reasonable measures to meet the assessment criteria. In assessing feasible and reasonable mitigation measures, an increase of up to 2 dB represents a minor impact that is considered barely perceptible to the average person.

Vibration criteria

Guidelines for vibration from project activities that are based on structural damage and human comfort (as tactile vibration or regenerated noise) have been adopted in the assessment.

In relation to human comfort, intermittent vibration has been evaluated on the basis of the Environment Protection Authority guideline Assessing Vibration: A Technical Guideline (Department of Environment and Conservation, 2006), which is based on vibration dose values (VDV). The criteria for VDV are based on the potential for annoyance (based on the level of vibration over the assessment period). Guidelines for continuous and impulsive vibration are dependent on the time of day and the activity taking place. The criteria established for these vibration types are based on the potential for adverse comment (complaint) and disturbance to building occupants.

The structural damage guidelines adopted are the German Standard DIN 4150 (as there are no Australian Standards available).

Ground-borne noise

Noise from activities such as vibratory rolling or tunnelling are assessed on the basis of criteria outlined in the ICNG for the day-time and night-time. These criteria are based on amenity and sleep disturbance when people are at home.

5.2 Impacts

5.2.1 Noise impacts

The assessment of noise impacts has considered the activities proposed to be undertaken (and equipment to be used) as well as the work hours and overall duration of the activities.

During standard work hours the assessment has identified a number of sensitive receivers in the community (including some classrooms at Hornsby TAFE) where noise management levels (NML) are exceeded. No sensitive receivers are identified as highly noise affected. The exceedance of the NML is typical of a construction project and highlights the need for a detailed Construction Noise and Vibration Management Plan (CNVMP) to ensure that all feasible and reasonable noise management and mitigation measures are employed. The NVTP has outlined a range of management and mitigation measures to be considered in the project.

Cumulative noise impacts from the project as well as maintenance pumping of the quarry void (by council) have also been assessed. In general the predicted noise impacts are not changed by the additional consideration of pumping activities. The exception to this is during the conveyer construction phase where noise levels could increase, however the increase would be by less than 1dB(A), and the cumulative impacts are unlikely to affect sensitive receivers.

No out of hours work is proposed to be undertaken and hence no noise impacts are assessed for these periods of time.

Review of the impact of road traffic (for the initially proposed peak number of 50 movements per hour into the site) on noise levels has identified that the predicted increase for sub-arterial and arterial roads during the morning and afternoon peak periods (less than 2 dB) meets the recommended noise goal. However, noise level increase for Bridge Road, the only affected local road, is predicted to be exceeding the 2 dB(A) increase criterion.

A sensitivity analysis was undertaken to determine a more manageable number of movements that would achieve compliance. To achieve compliance, it was considered that a maximum of 10 movements per hour would be required. However this number of movements is not considered feasible to meet project requirements of efficient spoil disposal in line with the rate of spoil generation at the tunnel excavation sites. To ensure the potential traffic noise impact from the project is minimised as far as practicable, while still allowing a reasonable volume of material to be hauled in to the site, the maximum number of movements per hour into the site was decreased to 35. This decrease reduced the exceedance on bridge Road to 7 to 10 dB(A) depending on the time of day. Whilst these levels are still non-compliant, they are considered a realistic balance between the project's objectives and ensuring noise impacts are minimised as far as reasonably and feasibly possible.

Additional feasible and reasonable noise management and mitigation measures would be considered for all residual noise exceedances and recommendations have been provided in the NVTP. Further noise mitigation management measures, including hoarding would be investigated by the Contractor and presented in the CNVMP. Residents would also be consulted to ensure that the most appropriate combination of noise mitigation will be implemented to minimise the impacts on sensitive receivers.

As a result of the assessment undertaken a CNVMP would be prepared. The CNVMP would address the following:

- Identification of nearby residences and other sensitive land uses.
- Description of approved hours of work.
- Description and identification of all project activities, including work areas, equipment and duration.
- Description of what work practices (generic and specific) would be applied to minimise noise and vibration. This would include feasible and reasonable mitigation measures to manage predicted noise and vibration impacts.
- A complaints handling process.
- Noise and vibration monitoring procedures.
- Overview of community consultation required for identified high impact works.
- Cumulative noise impacts of the maintenance pumping of the quarry void.

5.2.2 Vibration impacts

The assessment undertaken considered that given the site layout and existing offset distances to sensitive receivers it is unlikely that the proposed activities would result in exceedance of vibration criteria relevant to structural damage or human comfort. Where work is required to be undertaken during the site establishment phase closer to sensitive receivers a review of the equipment proposed to be used, with alternative equipment (with lower potential for vibration impacts) would be implemented/used.

Impacts associated with vibration are to be addressed, mitigated or managed, using measures to be outlined in the Construction Noise and Vibration Management Plan.

5.3 Health outcomes relevant to noise

Environmental noise has been identified (I-INCE 2011; WHO 2011) as a growing concern in the growth of urban areas because it has negative effects on quality of life and well-being and it has the potential for causing harmful physiological health effects. With increasingly urbanised societies impacts of noise have the potential to increase within the community.

Deciding on the most effective noise management option in a specific situation is not just a matter of defining noise control actions to achieve the lowest noise levels or meeting arbitrarily chosen criteria for exposure to noise. The goal should be to achieve the best available compromise between the benefits to society of reduced exposure to community noise versus the costs and technical feasibility of achieving the desired exposure levels. On the one hand there are the rights of the community to enjoy an acceptably quiet and healthy environment. On the other are the needs of the society for a new or upgraded facilities, industries, roads, recreation opportunities, etc, all of which typically produce more community noise (I-INCE 2011; WHO 2011).

Sound is a natural phenomenon that only becomes noise when it has some undesirable effect on people or animals. Unlike chemical pollution, noise energy does not accumulate either in the body or in the environment but it can have both short-term and long-term adverse effects on people. These health effects include (WHO 1999, 2011):

- Sleep disturbance.
- Annoyance.
- Hearing impairment.
- Interference with speech and other daily activities.
- Children's school performance (through effects on memory and concentration).
- Cardiovascular health.

Other effects for which evidence of health impacts exists, but for which the evidence is weaker, include:

- Effects on mental health (usually in the form of exacerbation of existing issues for vulnerable populations rather than direct effects).
- Effects on the performance of cognitive tasks.
- Some evidence of indirect effects such as impacts on the immune system.

Often, annoyance is the major consideration because it reflects the community's dislike of noise and their concerns about the full range of potential negative effects.

There are many possible reasons for noise annoyance in different situations. Noise can interfere with speech communication or other desired activities. Noise can contribute to sleep disturbance, which can obviously be very annoying and has the potential to lead to long-term health effects. Sometimes noise is just perceived as being inappropriate in a particular setting without there being any objectively measurable effect at all. In this respect, the context in which sound becomes noise can be more important than the sound level itself.

Different individuals have different sensitivities to different types of noise and this reflects differences in expectations and attitudes more than it reflects any differences in underlying auditory physiology. A noise level that is perceived as reasonable by one person in one context (for example in their kitchen when preparing a meal) may be considered completely unacceptable by that same person in another context (for example in their bedroom when they are trying to sleep). In this case the annoyance relates, in part, to the intrusion from the noise. Similarly a noise level, which is considered to be completely unacceptable by one person, may be of little consequence to another even if they are in essentially the same room. In this case the annoyance depends almost entirely on the personal preferences, lifestyles and attitudes of the listeners concerned.

It is against this background that regulators in various communities have established sound level criteria above which noise is deemed to be unacceptable and below which it is deemed to be acceptable. Any assessment of noise impacts needs to consider the relevant criteria established for a new or existing (or upgraded) facility or activity. Where there are impacts in excess of these guidelines an assessment of noise mitigation is required to be undertaken.

In relation to the project, potential noise impacts have been assessed against Australian (more specifically New South Wales) criteria that have been established on the basis of the relationship between noise and health impacts. The criteria developed for use in the assessment for control of noise come from policy documents developed by the NSW Government including the NSW Interim Construction Noise Policy, and the NSW Road Noise Policy. These policies are based on the health effects of noise, and are based on guidance and reviews published in the following:

- World Health Organisation- Guidelines on Community Noise – Health effects of noise (WHO 1999).
- World Health Organisation – Night Noise Guidelines for Europe (WHO 2009).
- Environmental Health Council of Australia - The health effects of environmental noise – other than hearing loss (enHealth 2004).

Various attempts have been made to assess the effect (measured by average reported annoyance, sleep disturbance or a similar type of effect) from community noise (measured by long term average sound levels) to develop exposure-response relationships. As individual reactions to noise are so varied, these studies need large sample sizes to obtain reasonable correlation between the noise exposure and the response. Any dose-response relationship determined from large studies over a range of communities and cultures will not necessarily represent the reaction of individuals or small communities.

These exposure-response relationships are of value for macro-scale (i.e. whole urban environment scale) strategic assessment purposes where individual differences are not important, however they are not useful when considering potential impacts to a small population located close to a specific project/activity. Hence these macro-scale relationships cannot be applied (in any meaningful way) in this assessment.

As guidelines/criteria are available for noise and vibration impacts associated with this project, that are based on the protection of health (including annoyance), the assessment of potential health impacts has focused on whether the guidelines/criteria established can be met. Noise levels that do not comply with these guidelines/criteria may have the potential to have negative health outcomes for the community adjacent to the project. The guidelines in relation to construction noise provides management levels that require assessment of noise impacts and implementation of practical and feasible management measures to minimise impacts. Where this process is followed, and where project works are only expected to occur for a relatively short period of time (as is the case with the proposed project, when compared to permanent operations) no adverse health effects are expected to occur in the community.

Currently, the worst case assessment predicts that noise criteria would be exceeded at a number of properties without additional noise mitigation measures. Detailed consideration of feasible and reasonable mitigation measures will determine the suite of measures that will achieve the most effective outcome for affected receivers. These measures will be detailed within the CNVMP and implemented to manage predicted impacts at sensitive receivers. Consultation with the affected community would also occur prior to and during the project.

Where the proposed noise management and mitigation measures are adopted, no adverse health impacts are exposure in the local community.

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Section 6. Conclusions

An assessment of health impacts associated with emissions to air as well as noise and vibration resulting from the project has been undertaken.

In relation to impacts to air quality, potential health impacts have been evaluated on the basis of appropriate health based guidelines (that are protective of public health), or, in the case of exposure to $PM_{2.5}$ and PM_{10} conducting a detailed assessment of the impact of the emissions on key community health indicators. All predicted concentrations of carbon monoxide, nitrogen dioxide, key individual volatile organic compounds and polycyclic aromatic hydrocarbons are below health based guidelines.

For the assessment of potential impacts of $PM_{2.5}$ and PM_{10} the following has been determined:

- Potential concentrations of silica that may be present in $PM_{2.5}$ and PM_{10} dust emissions are not considered to be of concern for community health.
- Calculated risks associated with exposures to PM_{10} and $PM_{2.5}$ (including DPM) from emissions from trucks on the haulage route roads are not considered to be of concern for community health.
- In relation to the proposed activities at the Hornsby Quarry site, where best practice mitigation measures are considered, risks are considered to be tolerable.
- Where risks are determined to be tolerable, it is expected that mitigation measures would be implemented to minimise exposures associated with the project. For this project, air impacts and hence health risks are proposed to be further minimised through the implementation of best industry practice dust management and mitigation measures. Such measures would result in lower levels of exposure and risk at all receptors in the surrounding community.

In relation to noise and vibration, potential impacts associated with the project have been considered. The worst case assessment predicts that noise criteria would be exceeded at a number of properties without additional noise mitigation measures. Feasible and reasonable mitigation measures would be detailed within the CNVMP to manage and mitigate predicted noise levels at sensitive receivers. In addition consultation with the affected community would also occur prior to and during the project. Where the proposed noise management and mitigation measures are adopted, no adverse health impacts are expected in the local community.

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Appendix A Methodology for Detailed Assessment of Exposure to $PM_{2.5}$ and PM_{10}

A1 Summary of adverse health effects

Adverse health effects associated with exposure to particulate matter have been well studied and reviewed by Australian and International agencies. Most of the studies and reviews have focused on population-based epidemiological studies in large urban areas in North America, Europe and Australia, where there have been clear associations determined between health effects and exposure to $PM_{2.5}$ and to a lesser extent, PM_{10}. These studies are complemented by findings from other key investigations conducted in relation to the characteristics of inhaled particles; deposition and clearance of particles in the respiratory tract; animal and cellular toxicity studies; and studies on inhalation toxicity by human volunteers (NEPC 2010).

Particulate matter has been linked to adverse health effects after both short-term exposure (days to weeks) and long-term exposure (months to years). The health effects associated with exposure to particulate matter vary widely (with the respiratory and cardiovascular systems most affected) and include mortality and morbidity effects.

In relation to mortality: for short-term exposures in a population this relates to the increase in the number of deaths due to existing (underlying) respiratory or cardiovascular disease; for long-term exposures in a population this relates to mortality rates over a lifetime, where long-term exposure is considered to accelerate the progression of disease or even initiate disease.

In relation to morbidity effects, this refers to a wide range of health indicators used to define illness that have been associated with (or caused by) exposure to particulate matter. In relation to exposure to particulate matter, effects are primarily related to the respiratory and cardiovascular system and include (Morawska, Moore & Ristovski 2004; USEPA 2009b):

- Aggravation of existing respiratory and cardiovascular disease (as indicated by increased hospital admissions and emergency room visits).
- Changes in cardiovascular risk factors such as blood pressure.
- Changes in lung function and increased respiratory symptoms (including asthma).
- Changes to lung tissues and structure.
- Altered respiratory defence mechanisms.

These effects are commonly used as measures of population exposure to particulate matter in community epidemiological studies (from which most of the available data in relation to health effects is derived), and are more often grouped (through the use of hospital codes) into the general categories of cardiovascular morbidity/effects and respiratory morbidity/effects. The available studies provide evidence for increased susceptibility for various populations, particularly older populations, children and those with underlying health conditions (USEPA 2009b).

There is consensus in the available studies and detailed reviews that exposure to fine particulates, $PM_{2.5}$, is associated with (and causal to) cardiovascular and respiratory effects and mortality (all causes) (USEPA 2012). Similar relationships have also been determined for PM_{10} , however, the supporting studies do not show relationships as clear as shown with $PM_{2.5}$ (USEPA 2012).

There are a number of other studies that have been undertaken where other health effects have been evaluated. These studies are suggestive (but do not show effects as clearly as the effects noted above) of an association between exposure to $PM_{2.5}$ and reproductive and developmental effects as well as cancer, mutagenicity and genotoxicity (USEPA 2012). IARC (2013) has classified particulate matter as carcinogenic to human based on data relevant to lung cancer.

Other studies have been reviewed to determine relationships/associations between particulate matter exposure (either PM_{10} or $PM_{2.5}$) and a wide range of other health effects and health measures including mortality (for different age groups), chronic bronchitis, medication use by adults and children with asthma, respiratory symptoms (including cough), restricted work days, work days lost, school absence and restricted activity days (Anderson et al. 2004; EC 2011; Ostro 2004; WHO 2006a). While these relationships/associations have been identified the exposure-response relationships established are not as strong as those discussed above. Also the available baseline data does not include information for many of these health effects which means it is not possible to undertake a quantitative assessment.

The detailed assessment of potential health effects associated with exposure to emissions associated with the project has focused on health effects and exposure-response relationships[12] that are robust and relate to $PM_{2.5}$, being the more important particulate fraction size relevant for emissions from combustion sources. These health effects (or endpoints) have been identified and agreed with NSW Health and include the following:

- Primary health endpoints:
  - Long-term exposure to $PM_{2.5}$ on all-cause mortality (≥ 30 years of age).
  - Short-term exposure on the rate of hospitalisation with cardiovascular and respiratory disease (≥ 65 years of age).
- Secondary health endpoints (to supplement the primary assessment):
  - Long-term exposure to $PM_{2.5}$ on cardiopulmonary mortality (≥ 30 years of age).
  - Short-term exposure to $PM_{2.5}$ on mortality (all causes, cardiovascular and respiratory, all ages).
  - Short-term exposure to PM_{10} on mortality (all causes and all ages).

A2 Exposure-response relationships

Mortality and morbidity health endpoints

A quantitative assessment of risk for these endpoints uses a mathematical relationship between an exposure concentration (ie concentration in air) and a response (namely a health effect). This relationship is termed an exposure-response relationship and is relevant to the range of health effects (or endpoints) identified as relevant (to the nature of the emissions assessed) and robust. An exposure-response relationship can have a threshold, where there is a safe level of exposure,

[12] An exposure-response relationship is a quantitative relationship between an exposure concentration of particulate matter in air (what is inhaled) and the health effect evaluated.

below which there are no adverse effects; or the relationship can have no threshold (and is regarded as linear) where there is some potential for adverse effects at any level of exposure.

In relation to the health effects associated with exposure to particulate matter, no threshold has been identified. Non-threshold exposure-response relationships have been identified for the primary and secondary health endpoints considered in this assessment.

A range of exposure-response relationships are available from the many studies that have been undertaken and published. Review of the available studies has been undertaken in Australia for the purpose of developing the NEPC Air Quality Guidelines (Burgers & Walsh 2002; NEPC 2002, 2010), where a range of health endpoints and exposure-response relationships were identified and evaluated. Similar exposure-response relationships have been considered in the development and review of air guidelines established by the WHO (WHO 2005) and the USEPA (USEPA 2012). These organisations have identified which of the available relationships that have been identified are the most robust.

The exposure-response relationships adopted in this assessment have been identified on the basis of the studies considered in the development of the NEPC Air Quality Guidelines as well as updated supporting studies published in the literature.

The assessment of potential risks associated with exposure to particulate matter involves the calculation of a relative risk (RR). For the purpose of this assessment the shape of the exposure response function used to calculate the relative risk is assumed to be linear[13]. The calculation of a relative risk based on the change in relative risk exposure concentration from baseline/existing (ie based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro 2004):

$$RR = exp[\beta(X-X_0)] \quad \text{…Equation 1}$$

Where:

$X-X_0$ = the change in particulate matter concentration to which the population is exposed ($\mu g/m^3$)

β = regression/slope coefficient, or the slope of the exposure-response function which can also be expressed as the per cent change in response per 1 $\mu g/m^3$ increase in particulate matter exposure.

[13] Some reviews have identified that a log-linear exposure response function may be more relevant for some of the health endpoints considered in this assessment. Review of outcomes where a log-linear exposure-response function has been adopted (Ostro 2004) for $PM_{2.5}$ identified that the log-linear relationship calculated slightly higher relative risks compared with the linear relationship within the range 10-30 $\mu g/m^3$,(relevant for evaluating potential impacts associated with air quality goals or guidelines) but lower relative risks below and above this range. For this assessment (where impacts from a particular project are being evaluated) the impacts assessed relate to concentrations of $PM_{2.5}$ that are well below 10 $\mu g/m^3$ and hence use of the linear relationship is expected to provide a more conservative estimate of relative risk.

Based on this equation, where the published studies have derived relative risk values that are associated with a 10 µg/m^3 increase in particulate matter exposure (as presented in **Table A-**1), the β coefficient can be calculated using the following equation:

$$\beta = \frac{\ln(RR)}{10} \quad \text{… Equation 2}$$

Where:

RR = relative risk for the relevant health endpoint as published and listed in **Table 5-1** (µg/m^3)

10 = increase in particulate matter concentration associated with the RR (all the RR presented in **Table 5-1** are associated with a 10 µg/m^3 increase in particulate matter exposure).

Table A-1 presents a summary of the health endpoints considered in this assessment, the relevant health impact functions (from the referenced published studies) and the associated β value relevant to the calculation of a relative risk.

The health impact functions presented in this table have been discussed and agreed with NSW Health as the most current and appropriate for the quantification of potential health effects for the health endpoints considered in this assessment.

Table A-1 Adopted health impact functions and exposure-responses relationships

Health endpoint	Exposure period	Age group	Published relative risk [95% confidence interval] per 10 µg/m^3	Adopted β coefficient (as %) for 1 µg/m^3 increase in PM	Reference
Primary assessment health endpoints					
PM2.5: Mortality, all causes	Long-term	≥30yrs	1.06 [1.04-1.08]	0.0058 (0.58%)	Relationship derived for all follow-up time periods to the year 2000 (for approx. 500 000 participants in the US) with adjustment for seven ecologic (neighbourhood level) covariates (Krewski et al. 2009). This study is an extension (additional follow-up and exposure data) of the work undertaken by Pope (2002), is consistent with the findings from California (1999-2002) (Ostro et al. 2006) and is more conservative than the relationships identified in a more recent Australian and New Zealand study (EPHC 2010).
PM2.5: Cardiovascular hospital admissions	Short-term	≥65yrs	1.008 [1.0059-1.011]	0.0008 (0.08%)	Relationship established for all data and all seasons from US data for 1999 to 2005 for lag 0 (exposure on same-day)(strongest effect identified) (Bell, M. L. 2012; Bell, Michelle L. et al. 2008)
PM2.5: Respiratory hospital admissions	Short-term	≥65yrs	1.0041 [1.0009-1.0074]	0.00041 (0.041%)	Relationship established for all data and all seasons from US data for 1999 to 2005 for lag 2 (exposure 2 days previous)(strongest effect identified) (Bell, M. L. 2012; Bell, Michelle L. et al. 2008)
Secondary assessment health endpoints					
PM10: Mortality, all causes	Short-term	All ages*	1.006 [1.004-1.008]	0.0006 (0.06%)	Based on analysis of data from European studies from 33 cities and includes panel studies of symptomatic children (asthmatics, chronic respiratory conditions) (Anderson et al. 2004)
PM2.5: Mortality, all causes	Short-term	All ages*	1.0094 [1.0065-1.0122]	0.00094 (0.094%)	Relationship established from study of data from 47 US cities for the years 1999 to 2005 (Zanobetti & Schwartz 2009)
PM2.5: Cardiopulmonary Mortality	Long-term	≥30yrs	1.14 [1.11-1.17]	0.013 (1.3%)	Relationship derived for all follow-up time periods to the year 2000 (for approx. 500 000 participants in the US) with adjustment for seven ecologic (neighbourhood level) covariates (Krewski et al. 2009).
PM2.5: Cardiovascular mortality	Short-term	All ages*	1.0097 [1.0051-1.0143]	0.00097 (0.097%)	Relationship established from study of data from 47 US cities for the years 1999 to 2005 (Zanobetti & Schwartz 2009)
PM2.5: Respiratory mortality (including lung cancer)	Short-term	All ages*	1.0192 [1.0108-1.0278]	0.0019 (0.19%)	Relationship established from study of data from 47 US cities for the years 1999 to 2005 (Zanobetti & Schwartz 2009)

* Relationships established for all ages, including young children and the elderly

Exposure to diesel particulate matter

In addition to the above exposure-response relationships, potential exposure to diesel particulate matter (DPM) derived from the project has been evaluated.

Diesel exhaust (DE) is emitted from “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 present in diesel exhaust ranges from hours to days.

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 carrying out of the project include a significant proportion of diesel powered vehicles (100 per cent of the HGVs and 49.9 per cent of the LDVs). 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 (eg episodic) exposure to diesel particulate matter can cause acute irritation (eg eye, throat, bronchial), neurophysiological symptoms (eg 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. Chronic effects include respiratory effects. The review of these effects (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 will also be met if the $PM_{2.5}$ guideline was met. Review of exposure to $PM_{2.5}$ has been assessed separately in relation to the current ambient air guidelines (refer to **Section 4.4.4**) where cumulative impacts of $PM_{2.5}$ for the project have been found to comply with the NEPC $PM_{2.5}$ advisory goal. Hence non-carcinogenic effects associated with exposure to diesel particulate matter are not considered to be of concern.

Review of exposures to diesel particulate matter (USEPA 2002) identified that such exposures are “likely to be carcinogenic to humans by inhalation”. A more recent review by IARC (Attfield et al. 2012; IARC 2012; 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.

Many of the organic compounds present in diesel exhaust are known to have mutagenic and carcinogenic properties and hence 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.

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 1998). 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 one in one hundred thousand excess risk of cancer. This estimate has been widely criticised as overestimating the risk and hence has not been considered in this assessment.

On the basis of the above, the WHO cancer unit risk value (mean value of 3.4×10^{-5} per µg/m^3) has been used to evaluate potential excess lifetime risks associated with incremental impacts from diesel particulate matter exposures. Diesel particulate matter has not been specifically modelled in the AQIA; rather diesel particulate matter is part of the $PM_{2.5}$ assessment. For the purpose of this assessment it has been conservatively assumed that 100 per cent of the incremental $PM_{2.5}$ (from the project only) is derived from diesel sources. This is conservative as not all the vehicles from the project (and emitting $PM_{2.5}$) would be diesel powered (as currently there is a mix of petrol, diesel, LPG and hybrid-electric powered vehicles with the proportion of alternative fuels rising in the future).

A3 Particulate impact assessment

Quantification of impact and risk

The assessment of health impacts for a particular population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro 2004)[14] where the exposure-response relationships (presented in **Table A-1**) have been directly considered on the basis of the approach outlined below.

[14] For regional guidance, such as that provided for Europe by the WHO ((WHO 2006a)) regional background incidence data for relevant health endpoints are combined with exposure-response functions to present an impact function, which is expressed as the number/change in incidence/new cases per 100,000 population exposed per µg/m^3 change in particulate matter exposure. These impact functions are simpler to use than the approach adopted in this assessment, however in utilising this approach it is assumed that the baseline incidence of the health effects is consistent throughout the whole population (as used in the studies) and is specifically applicable to the sub-population group being evaluated. For the assessment of exposures in the areas evaluated surrounding the project it is more relevant to utilise local data in relation to baseline incidence rather than assume that the population is similar to that in Europe (where these relationships are derived).

The calculation of changes in health endpoints associated with exposure to particulate matter as outlined by the WHO (Ostro 2004) has considered the following four elements:

- Estimates of the changes in particulate matter exposure levels (ie incremental impacts) due to the project for the relevant modelled scenarios (as provided by the AQIA);
- Estimates of the number of people exposed to particulate matter at a given location (ie population data);
- Baseline incidence of the key health endpoints that are relevant to the population exposed; and
- Exposure-response relationships expressed as a percentage change in health endpoint per $\mu g/m^3$ change in particulate matter exposure, where a relative risk (RR) is determined (refer to Equation 1).

From the above, the increased incidence of a health endpoint corresponding to a particular change in particulate matter concentrations can be calculated using the following:

The attributable fraction/portion (AF) of health effects from air pollution, or impact factor, can be calculated from the relative risk (calculated for the incremental change in particulate matter considered as per Equation 1) as:

$$AF = \frac{RR-1}{RR} \quad \text{… Equation 3}$$

The total number of cases attributable to exposure to particulate matter (where a linear dose-response is assumed) can be calculated as:

$$E = AF \times B \times P \quad \text{… Equation 4}$$

Where:
B = baseline incidence of a given health effect (eg mortality rate per person per year)
P = relevant exposed population

The above approach (while presented slightly differently) is consistent with that presented in Australia (Burgers & Walsh 2002), US (OEHHA 2002; USEPA 2005, 2010) and Europe (Martuzzi et al. 2002; Sjoberg et al. 2009). Where a linear dose-response is assumed (as is the case in this assessment), the calculations are equivalent to the following:

The calculation of an increased incidence (ie number of cases) of a particular health endpoint is not relevant to a specific individual, rather this is relevant to a statistically relevant population. This calculation has been undertaken for populations within the suburbs surrounding the proposed project. When considering the potential impact of the project on the population, the calculation has been undertaken using the following:

- Equation 1 has been used to calculate a relative risk. The relative risk has been calculated for a population weighted annual average incremental increase in $PM_{2.5}$ concentrations. The population weighted average has been calculated on the basis of the smallest statistical division provided by the Australian Bureau of Statistics within a suburb (i.e. mesh blocks – which are small blocks that cover an area of approximately 30 urban residences). For each mesh block in a suburb the average incremental increase in $PM_{2.5}$ concentration has been calculated and multiplied by the population living in the mesh block (data available from the ABS for the 2011 census year). The weighted average has been calculated by summing these calculations for each mesh block in a suburb and dividing by the total population in the suburb (i.e. in all the mesh block).
- Equation 3 has been used to calculate an attributable fraction.
- Equation 4 has been used to calculate the increased number of cases associated with the incremental $PM_{2.5}$ impact evaluated. The calculation is undertaken utilising the baseline incidence data relevant for the endpoint considered and the population (for the relevant age groups) present in the suburb.

The above approach can be simplified (mathematically, where the incremental change in particulate concentration is low, less than 1 µg/m^3) as follows:

$$E=\beta \times B \times \sum_{mesh} (\Delta X_{mesh} \times P_{mesh}) \quad \text{… Equation 5}$$

Where:
β = slope coefficient relevant to the per cent change in response to a 1 µg/m^3 change in particulate matter exposure (as per **Table 5-1**)
B = baseline incidence of a given health effect per person (eg annual mortality rate)
ΔX_{mesh} = change (increment) in PM_{10} or $PM_{2.5}$ exposure concentration in µg/m^3 as an average within a small area defined as a mesh block (from the ABS – where many mesh blocks make up a suburb)
P_{mesh} = population (residential – based on data from the ABS) within each small mesh block

An additional risk can then be calculated as:

$$Risk=\beta \times \Delta X \times B \quad \text{… Equation 6}$$

Where:
β = slope coefficient relevant to the per cent change in response to a 1 µg/m^3 change in particulate matter exposure (as per **Table 5-1**)
ΔX = change (increment) in PM_{10} or $PM_{2.5}$ exposure concentration in µg/m^3 relevant to the project at the point of exposure
B = baseline incidence of a given health effect per person (eg annual mortality rate)

This calculation provides an annual risk for individuals exposed to increased PM emissions from the project at specific locations (such as the maximum, or at specific sensitive receiver locations).

For the assessment of potential lung cancer risks associated with exposure to diesel particulate matter, a non-threshold cancer risk is calculated. Non-threshold carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential non-threshold carcinogen. The numerical estimate of excess lifetime cancer risk is calculated as follows for inhalation exposures (USEPA 2009a):

Carcinogenic Risk (inhalation) = Exposure Concentration in Air x Inhalation Unit Risk

Quantification of short-and long-term effects

The concentration-response functions adopted for the assessment of exposure are derived from long and short-term studies and relate to short or long-term effects endpoints (eg change in incidence from daily changes in particulate matter, or chronic incidence from long-term exposures to particulate matter).

Long-term or chronic effects are assessed on the basis of the identified exposure-response function and annual average particulate matter concentrations. These then allow the calculation of a chronic incidence of the assessed health endpoint.

Short-term effects are also assessed on the basis of an exposure-response function that is expressed as a percentage change in endpoint per $\mu g/m^3$ change in particulate matter exposure. For short-term effects, the calculations relate to daily increases in particulate matter exposures and changes in daily effects endpoints. While it may be possible to measure daily incidence of the evaluated health endpoints in a large population study specifically designed to include such data, it is not common to collect such data in hospitals nor are effects measurable in smaller communities. Instead these calculations relate to a parameter that is measurable, such as annual incidence of hospitalisations, mortality or lung cancer risks. The calculation of an annual incidence or additional risk can be undertaken using two approaches (Ostro 2004; USEPA 2010):

1. Calculate the daily incidence or risk at each receiver location over every 24-hour period of the year (based on the modelled incremental 24-hour average concentration for each day of the year and daily baseline incidence data) and then sum the daily incidence/risk to get the annual risk; or
2. Calculate the annual incidence/risk based on the incremental annual average concentration at each receiver (and using annual baseline incidence data).

In the absence of a threshold, and assuming a linear concentration-response function (as is the case in this assessment), these two approaches result in the same outcome mathematically (calculated incidence or risk). Given that it is much simpler computationally to calculate the incidence (for each receiver) based on the incremental annual average, compared with calculating effects on each day of the year and then summing, this is the preferred calculation method. It is the recommended method outlined by the WHO (Ostro 2004).

The use of the simpler approach, based on annual average particulate matter concentrations should not be taken as implying or suggesting that the calculation is quantifying the effects of long-term exposure.

Hence for the calculations presented in this technical working paper, for both long-term and short-term effects, annual average concentrations of particulate matter have been utilised.

A4 Uncertainties

Exposure-response function

The choice of exposure-response functions for the quantification of potential health impacts is important. For mortality health endpoints, many of the exposure-mortality functions have been replicated throughout the world. While many of these have shown consistent outcomes, the calculated relative risk estimates for these studies do vary. This is illustrated by **Figures A-1** to **A-3** that show the variability in the relative risk estimates calculated in published studies for the US (and Canadian) population that are relevant to the primary health endpoints considered in this assessment (USEPA 2012). A similar variability is observed where additional studies from Europe, Asia and Australia/New Zealand are considered.

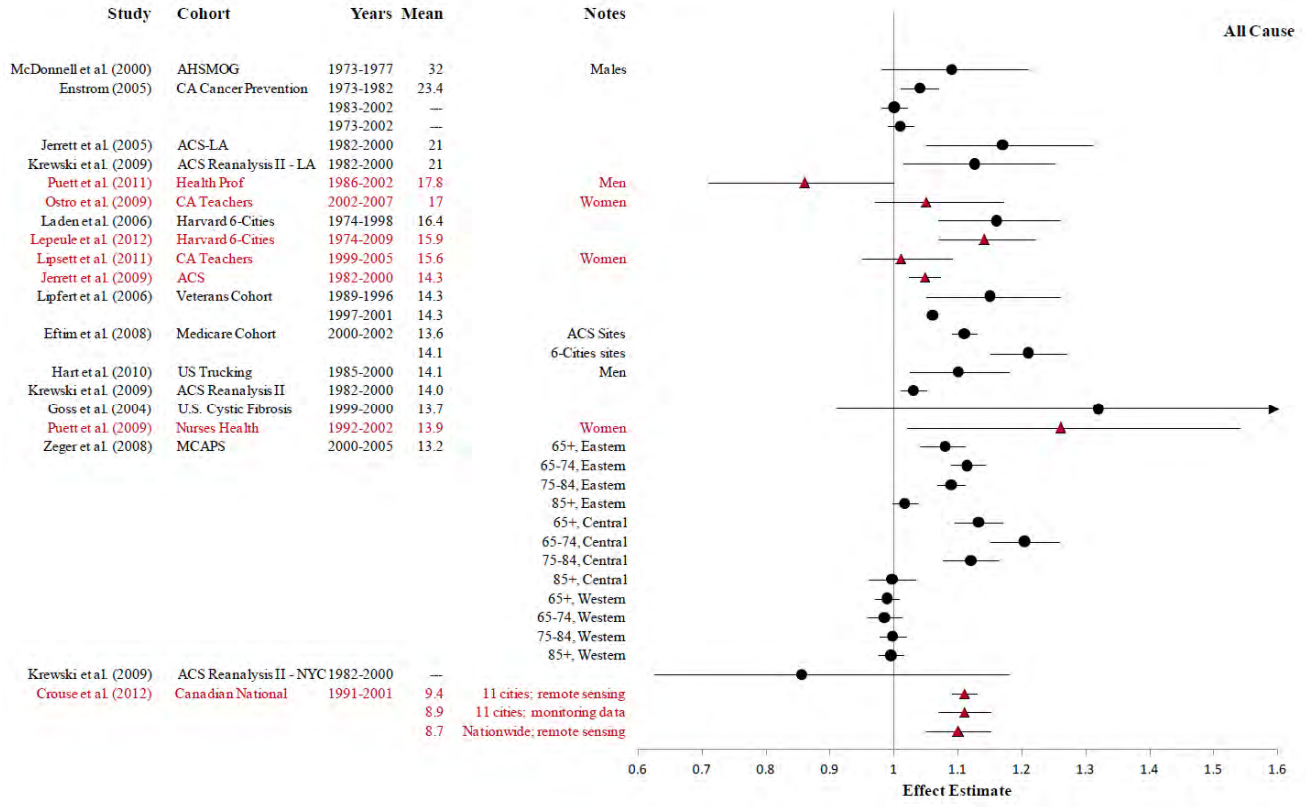

Figure A-1 **All-cause mortality relative risk estimates for long-term exposure to $PM_{2.5}$** (USEPA 2012, note studies in red are those completed since 2009)

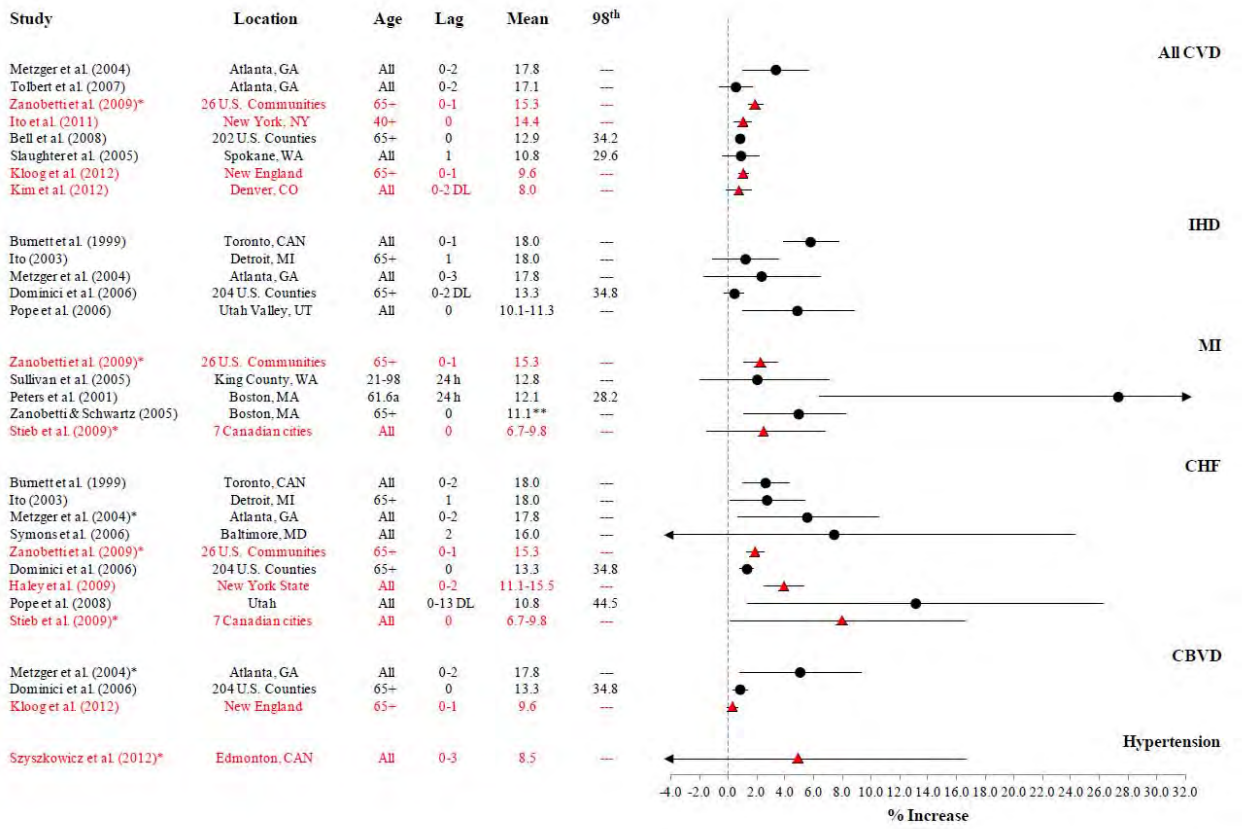

Figure A-2 **Per cent increase in cardiovascular-related hospital admissions for a 10 µg/m^3 increase in short-term (24-hour average) exposure to $PM_{2.5}$** (USEPA 2012, note studies in red are those completed since 2009)

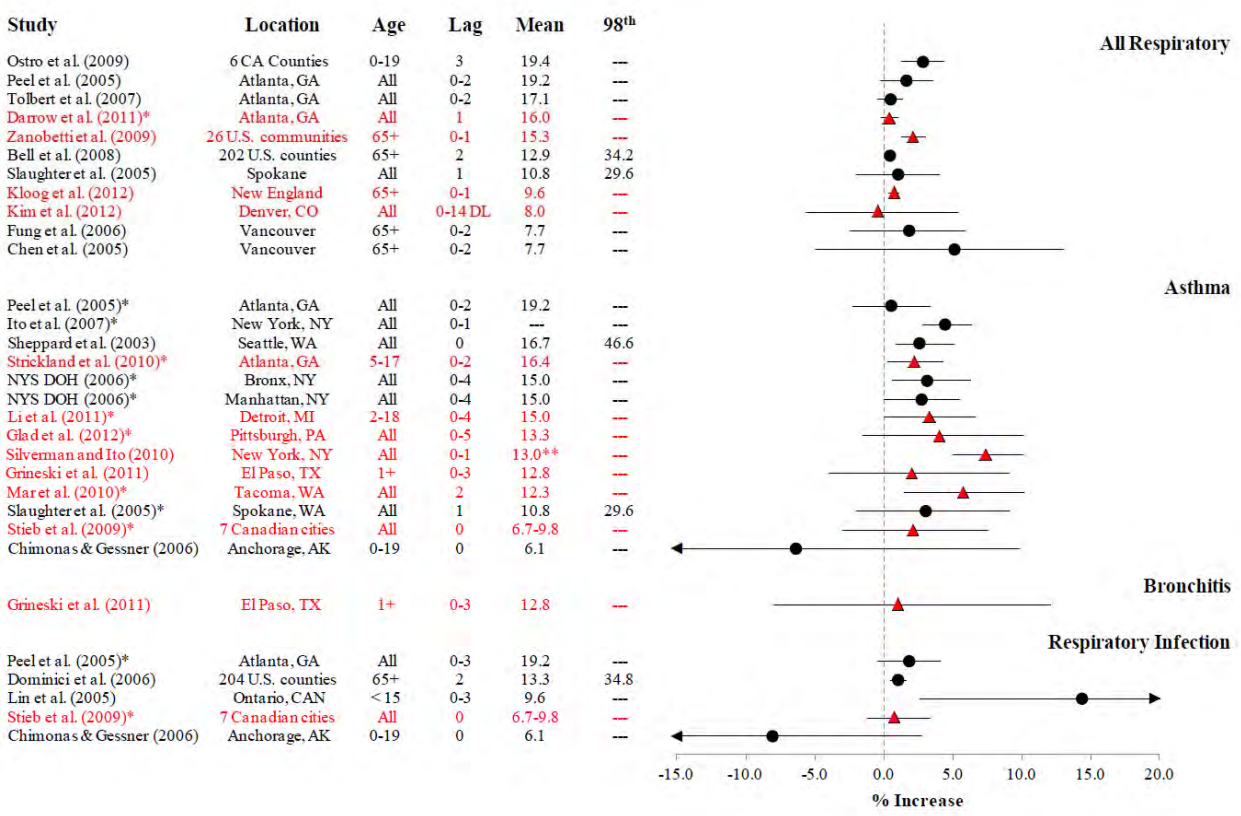

Figure A-3 **Per cent increase in respiratory-related hospital admissions for a 10 µg/m^3 increase in short-term (24-hour average) exposure to $PM_{2.5}$** (USEPA 2012, note studies in red are those completed since 2009)

These figures illustrate the variability inherent in the studies used to estimate exposure-response functions. The variability is expected to reflect the local and regional variability in the characteristics of particulate matter to which the population is exposed.

Based on the available data, and the detailed reviews undertaken by organisations such as the USEPA (USEPA 2010, 2012) and WHO (WHO 2003, 2006b, 2006a) and discussions with NSW Health, the adopted exposure-response estimates are considered to be current, robust and relevant to the characterisation of impacts from PM.

Shape of exposure-response function

The shape of the exposure-response function and whether there is a threshold for some of the effects endpoints remains an uncertainty. Reviews of the currently available data (that includes studies that show effects at low concentrations) have not shown evidence of a threshold. However, as these conclusions are based on epidemiological studies, discerning the characteristics of the particulates responsible for these effects and the observed shape of the dose-response relationship is complex. For example, it is not possible to determine if the observed no threshold response is relevant to exposure to particulates from all sources, or whether it relates to particulates from combustion sources only.

Most studies have demonstrated that there is a linear relationship between relative risk and ambient concentration however for long-term exposure-related mortality a log-linear relationship is more plausible and should be considered where there is the potential for exposure to very high concentrations of pollution. In this assessment the impact considered is a localised impact with low level incremental increases in concentration. At low levels the assumption of a linear relationship is considered appropriate.

Co-pollutants

It is likely that some of the health effects observed relate to both particulate matter and other related/correlated pollutants. Many of the pollutants evaluated come from a common source (eg fuel combustion) hence the use of only particulate matter as an index for the mix of pollutants is reasonable but conservative, particularly where there are multiple sources, or the scenario being evaluated is not from a source type that is likely to have dominated the studies underlying the relative risk values used in the risk assessment.

Selected health outcomes

The assessment of risk has utilised exposure-response functions and relative risk values that relate to the more significant health endpoints where the most significant and robust positive associations have been identified. The approach does not include all possible subsets of effects that have been considered in various published studies. However, the assessment undertaken has considered the health endpoints/outcomes that incorporate many of the subsets, and has utilised the most current and robust relationships.

Application of exposure-response functions to small populations

The exposure-response functions have been developed on the basis of epidemiological studies from large urban populations where associations have been determined between health effects (health endpoints) and changes in ambient (regional) particulate levels. Typically these exposure response functions are applied to large populations for the purpose of establishing/reviewing air guidelines or reviewing potential impacts of regional air quality issues on large populations. When applied to small populations (less than larger urban centres such as the whole of greater Sydney) the uncertainty increases.

In addition it is noted that the exposure-response functions relate changes in health endpoints with changes in regional air quality measurements. They do not relate to specific local sources (which occur within a regional airshed), or daily variability in exposure that may occur as a result of various different activities that may occur in any one day.

Diesel particulate matter evaluation

The health hazard conclusions associated with exposure to diesel particulate matter are based on studies that are dominated by exhaust emissions from diesel engines built prior to the mid-1990s. With current engine use including some new and many older engines (engines typically stay in service for a long time), the health hazard conclusions, in general, are likely to be applicable to engines currently in use. However as new and cleaner diesel engines, together with different diesel fuels, replace a substantial number of existing engines; the general applicability of the health hazard conclusions may require further evaluation. The NEPC (NEPC 2009) has established a program to reduce diesel emissions from the Australian heavy vehicle fleet. This is expected to lower the potential for all diesel emissions over time.

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