CHAPTER 9 COMMUNITY HEALTH

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9 COMMUNITY HEALTH

This chapter provides a summary of the human health risk assessment which includes the evaluation of existing health risks given current lead exposures and the analysis of incremental risks due to the Project taking into account the management measures proposed.

9.1 INTRODUCTION

A human health risk assessment (HHRA) has been undertaken for the Project by Toxikos Pty Ltd covering background environmental lead concentrations and characterising incremental risks due to the Project. This Chapter provides an overview of the scope, assessment methodology and main findings of the HHRA. Further details are provided in the *Health Risk Assessment for Rasp Mine Proposal Report* (2010) and an associated Addendum Report (2010) presented as *Annexure I(A)* and *Annexure I(B)* respectively.

9.1.1 Issue Identification

Lead, due to local minerialsation, occurs in the natural environment within the City of Broken Hill. Broken Hill has a long history of lead and zinc mining with past mining and smelting practices having resulted in widespread surface soil contamination of areas around the mines. As a result, the local population has experienced high blood lead levels, which according to modern criteria, were unacceptable (GWAHS 2008, Boreland et al. 2008a, Burke et al. 2003, Lesjak and Boreland 2008, Lyle et al. 2006).

In 1994, Broken Hill City Council undertook targeted remediation of public spaces and selected residences (Lyle et al. 2001, 2006). NSW Health also contributed towards remediation efforts. Recent initiatives include an education campaign to minimise lead exposure by modifying personal hygiene behaviour and advice that tank water should not be used for drinking or cooking. Such initiatives have resulted in a steady decline in children's blood lead levels (GWAHS 2008, Boreland et al. 2008a, Burke et al. 2003, Lesjak and Boreland 2008).

The major health concerns arising from the Project are associated with lead and lead exposures therefore these formed the specific focus for the HHRA. The HHRA addresses the requirements of the Director General (NSW Department of Planning), with all potential pathways to lead exposure from the Project having been addressed. The HHRA also takes into account long-term exposure to lead and other metal emissions from the Project. The HHRA conforms to the assessment principles articulated in the Australian *'Guidelines for assessing human health risks from environmental hazards'* (enHealth Council 2004).

Different mineralogical forms of lead are absorbed differently into the body and are a major factor in determining blood lead concentrations (US EPA 2006, ATSDR 2007). Lead from the Rasp Mine is predominantly in the form of weathered galena (anglesite, or lead sulphate) and has been found to have a lower potential to be available for absorption by the human body (Refer to *Section 9.3*). The HHRA aimed to assess health risks associated with the portion of lead able to be absorbed and influence blood lead concentrations.

9.1.2 Scenarios Considered

Dust from the mine lease areas are classified into two main source groups for the purpose of the HHRA. Firstly, dust emitted from the so called 'free areas', i.e. existing areas which are wind exposed and which will not be impacted by the day to day mine operation. Secondly, dust arising due to Project-related activities including truck and light vehicle movements, ore stockpiles, and processing activities. If the Project proceeds, dust from 'free areas' will be controlled with a minimum 80% efficiency (for details of dust controls refer *Chapter 8 Air Quality*).

The HHRA has been conducted on three exposure scenarios, all related to dust from the mine lease. These scenarios are as follows

- **Scenario 1 (S1)** evaluates off-site health risks from metals in dust arising from 'free areas' of the mine lease (i.e. those open areas not impacted by day to day mining and ore processing activities).
  - **S1a** : Existing free areas in current state.
  - **S1b** : Existing free areas with dust control to 80% efficiency, referred to as 'free areas (80% control)'.
- **Scenario 2 (S2)**: Assesses off-site health risks due to dust arising from Project activities including both mining and processing activities.
- **Scenario 3 (S3)**: Assesses off-site health risks from the cumulative impact of dust from the free areas (80% control) plus those in dust from mining and processing activities (ie S1b plus S2).

Health risks arising after 15 years of mine operation are considered for each of the three scenarios. This was achieved by estimating the accumulation of metals in residential soils from annual average deposition rates predicted by air dispersion modelling. This modelling, undertaken as part of the air quality assessment, is documented in Chapter 8. To provide a conservative (upper bound) estimate of the metals accumulating in soils it was assumed that all deposited metals remain in the soils with no provision made for removal due to wind erosion or runoff.

9.1.3 Risk Assessment Method Overview

The air quality assessment documented in Chapter 8 predicted total suspended particulate (TSP) concentrations, airborne concentrations of particulate matter with an aerodynamic diameter of less than 10 microns (PM_{10}), dust deposition rates and airborne concentration and deposition of metals in dust. Airborne dust and metal concentrations and dust deposition rates were evaluated against specific criteria used to assess the likely impact on the surrounding community. These predicted dust concentrations measured against required criteria are documented in Chapter 8. These concentrations were used as the basis for the HHRA.

Data output from the air quality modelling for input to the HHRA included concentrations of metals in airborne TSP and PM_{10}, and annual deposition rates of metals in TSP. Metals covered are: lead, arsenic, barium, silver, berilium, cadmium, chromium, iron, copper, manganese, mercury, nickel, selenium and zinc.

The HHRA evaluates risks from lead and other metal exposures via inhalation, ingestion and dermal contact exposure routes. Potential health impacts are assessed following two risk characterisation methods:

- Calculated metal intakes are compared with the tolerable daily intake (TDI) for each metal. Depending on the metal being assessed, intake estimations were derived from estimations of accumulation of metal in receptor soil, available information on background intakes and predicted metal concentration in PM_{10}. Where scenario specific information was not available, default assumptions commonly used in screening risk assessments were used (enHealth 2004). This approach was employed in a recent risk assessment for lead at the Port of Esperance (Golders Associates 2009).
- Blood lead concentrations in children are modelled at selected worst case receptors and receptors representative of historically designated risk zones around the mine. The US EPA Integrated Exposure Uptake and BioKinetic (IEUBK) model was used to predict blood lead concentrations associated with accumulated soil lead concentrations. IEUBK has been used to assist derivation of the Health Investigation Level (HIL) for lead in soil.

9.1.4 Receptor Selection

Airborne metal concentrations and deposition rates were predicted for a number of sensitive receptor sites, as illustrated in *Figure 9-1* and documented in *Section 8.2.3*. For ease of report reading and presentation within this Chapter, HHRA results are presented in summary tables for selected receptors (*Table 9-1*). These receptors were selected after consideration of:

- proximity to mine activity,
- the historical lead health risk zones in Broken Hill used by the Greater Western Health Services to organise and interpret lead biomonitoring data (Refer to *Figure 9-4*), and
- predicted dust deposition (low, medium, high) from the mine lease site.

Receptors 3 and 8 are predicted to be the most highly impacted receptors, and therefore represent plausible 'worst case.'

Table 9-1 Receptors selected for ease of reporting in the HHRA

Selected Receptors	
Residences	**Other Receptors**
1. Piper Street North	11. Alma Bugdlie Pre-School
2. Piper Street Central	12. Playtime Pre-School
3. Eyre Street North	14. Broken Hill High School
4. Eyre Street Central	17. Broken Hill Public School
5. Eyre Street South	18. Rainbow Pre School
8. Old South Road	
9. South Road (2)	
10. Garnet & Blende Streets	
23. Eyre Street North (3)	
32. Crystal Street (2)	
36. Crystal Street (5)	
38. Gypsum Street (1)	

Figure 9-1: Representative Sensitive Receptors in the Vicinity of the Project

9.2 EXPOSURE ESTIMATIONS

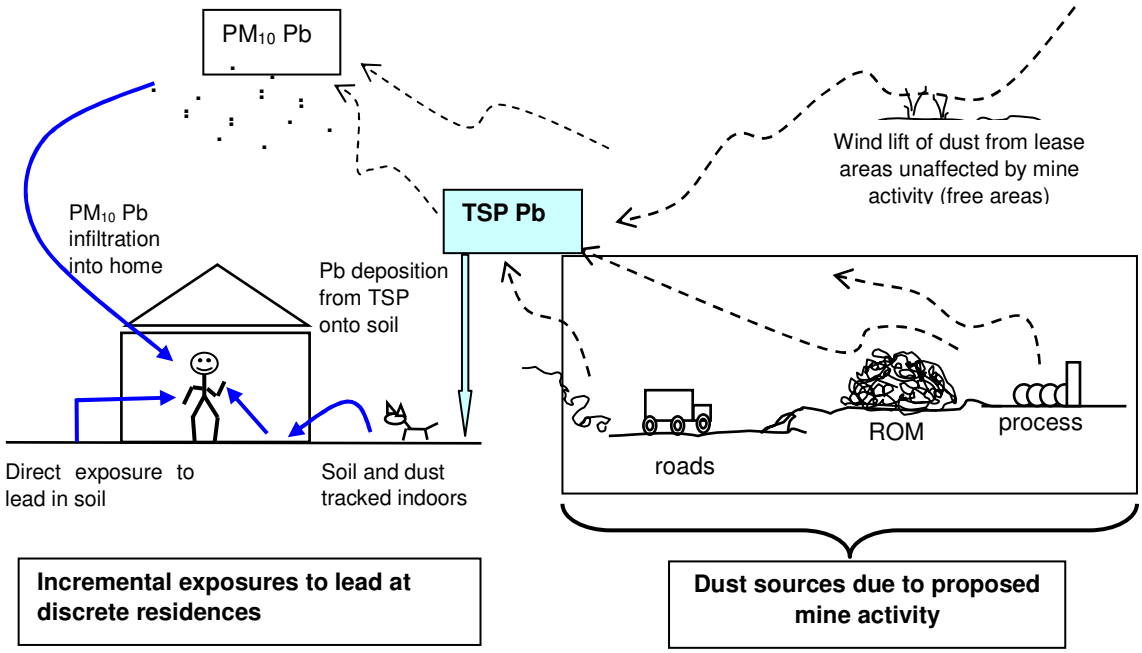
9.2.1 Exposure Pathways

Exposure pathways considered for lead exposure estimates are summarised in *Figure 9-2* and *Figure 9-3*. Pathways considered to be major for the scenarios being investigated are denoted with a bolded arrow in *Figure 9-3*. These pathways were also included in exposure estimations for the other metals.

The major off-site exposure pathway to mine derived metals in dust is incidental ingestion of soil that has had mine site dust deposited on it. The dust deposition occurs from mine derived TSP transported by wind. The sources contributing to the TSP are grouped into Project-related emissions (e.g. paved and unpaved active roads, ore handling, the ore stockpile, crushing, ventilation shafts etc.) and wind blown dust from existing exposed areas of the mine lease surface not involved with the day to day operation of the proposed mine (so called 'free areas').

At receptors, such as residences, exposure to mine derived metals may be via ingestion of soil, ingestion of indoor dust which has either been tracked in or which has settled after infiltration from outdoor air, and inhalation of indoor and/or outdoor airborne dust.

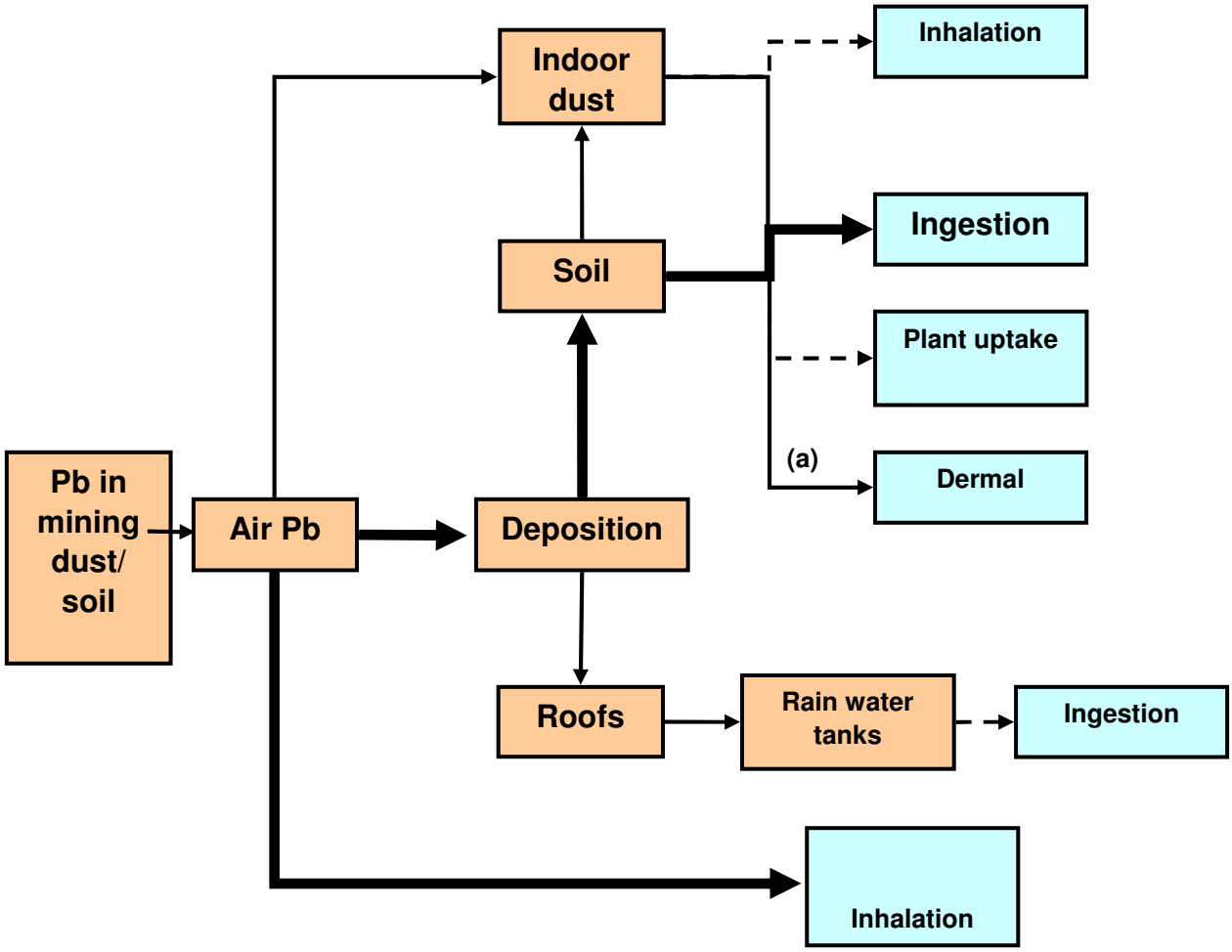
Figure 9-2: Conceptual representation of dust/lead sources and residential exposure pathways



Ingestion of tank water is not considered a complete exposure pathway because reticulated water is supplied and the local public health authority and Broken Hill City Council (BHCC) have undertaken an education campaign alerting residents to the risks of consuming tank water, and advising it not be drunk or used for food preparation.

Plant uptake of lead from home-grown produce was not considered a complete exposure pathway based on information obtained from the literature (Norberg *et al.*, 2007). Lead is not readily translocated to shoots of plants which are the edible part of most vegetables. For root vegetables, changes in soil lead have been found to result in small and slow uptake by vegetables such as radishes and potatoes. Furthermore, home grown produce makes up a small percentage of total fruit and vegetable intake by the general population; and finally, plants take up metals in soluble form, with take up of lead in this form shown to be low (1.4 to 7.3%).

Figure 9-3: Conceptual Site Model: Pathways considered in exposure estimates



9.2.2 Background Environmental Lead Concentrations in Broken Hill

A review of available data on soil lead concentrations in residential areas of Broken Hill and published literature on soil lead concentrations and blood lead levels was undertaken. A comprehensive summary from this review is provided in the HHRA documentation (*Annexure I*), with key observations presented in this subsection.

Recent data on soil lead concentrations in residential areas of Broken Hill were not located in the published literature. According to NSW Department of Health, monitoring lead contamination in soil at Broken Hill was conducted by the BHCC in the early 1990's and since then there has been no new systematic soil monitoring or data made available. Soil lead levels from the BHCC study in 1992 are reported in Boreland et al. (2002). One soil sample from the top 10cm of natural soil was collected from each city block for lead analysis. The samples were collected from undisturbed vacant land or, where this was not possible from nature-strips. Geometric mean soil lead concentrations ranged from about 250 to 2,300 µg of lead per gram of soil, with higher soil lead

levels observed to occur in the vicinity of mining areas. Boreland et al. (2002) found the higher the average soil lead in an area the higher the mean movement of lead indoors. Houses in areas with high soil lead had indoor lead levels three times that of houses in areas with lower soil lead. As would be expected the movement of lead indoors was influenced by the condition of the house with lowest levels observed in well sealed dwellings.

Soil data gathered by the BHCC in 1992 is suggested to represent soil lead levels prior to the implementation of an extensive remediation program in the area (Boreland and Lyle, 2006).

The lack of a single source of lead, and its widespread distribution in and around the city, meant that cleaning up the whole town and preventing the further release of lead into the environment was not feasible as a primary strategy. The situation required a more targeted approach that dealt with specific sources that could be linked to children with a high blood lead level, including remediation of public land (Lyle et al. 2006).

Boreland and Lyle (2006) indicate the remediation of homes of children with high blood lead levels commenced in 1994. As part of the remediation, yard soils with high lead levels were capped or removed. The study investigated the impact of house remediation on indoor levels of lead and unfortunately soil lead before and after remediation was not reported.

Extensive land remediation work in the highest lead-risk zones was largely completed by 1997, with final works undertaken in 2003 and 2004. Industrial and public land, including footpaths and vacant blocks, as well as some residential blocks considered to pose a hazard, adjacent to mining leases were comprehensively targeted; all were within two streets of the mines. Land remediation mainly consisted of covering contaminated soil with an appropriate material (clean soil, clay, mulch, concrete, crushed metal), or planting of hardy local native shrubs and grasses to stabilise soil (Boreland et al. 2008a).

Boreland and Lyle (2006) and Boreland et al. (2006) evaluated the effect of residential remediation on lead loadings of houses in Broken Hill, and the relationship of children blood lead with indoor lead levels. The homes in the Boreland and Lyle (2006) study were those of children with blood lead levels between 15 – 30 µg/dL (103 families) and ≥ 30 µg/dL (14 families)[1]. Indoor lead loading was 70–100% higher in areas with high soil lead levels and 90–150% higher in homes that were poorly sealed before remediation. The condition of the home and its location were significant predictors of indoor lead loadings. Both soil lead and the movement of lead from outside to inside were found to be influenced by proximity and wind direction from the former open-cut mines.

Boreland et al. (2006) found indoor lead was a significant predictor of blood lead but it only accounted for a small proportion of the variation in blood lead levels between children. Although, because of limited statistical power, the authors could not assign causality between blood lead and indoor dust they speculated that home remediation would have the most benefit in lowering blood lead for children who lived in homes with high indoor lead loadings.

An earlier study Gulson et al. (1995b) found that although lead in soil and house dust was dominated by ore body sources, the isotope fingerprinting of blood lead in children indicated a significant proportion of the latter lead was from different sources such as leaded petrol and paint.

Measurements of soil lead levels measured during the period 2004 to 2008 were undertaken either because a family residing within Broken Hill requested the sampling or because a child had elevated blood lead levels. The information from this sampling was made available to Toxikos by the Greater Western Health Services (Boreland 2010) for use in the HHRA as geometric means, 95% confidence intervals, minimum and maximum soil lead levels by district.

[1] The National Health and Medical Research Council (NHMRC) of Australia recently determined that all Australians should have a blood lead level of less than 10 µg/dL in order that public health impacts of environmental lead exposure be minimised (NHMRC, 2009).

Boreland et al. (2009a) defined five risk zones in describing hazards posed for soil lead concentrations based on the 1992 soil lead concentration measurements documented in Lyle et al. (2006). These risk zones are illustrated in *Figure 9-4*. Soil lead concentrations recorded during the 1992 BHCC study, and soil lead levels more recently recorded (2004-2008) are presented by risk zone in *Table 9-2*.

Due to the targeted nature of the 2004 – 2008 data, it may not be representative of soil lead levels. Nevertheless, the 95th percentile confidence limits of the recent data (2004-2008) indicate there has not been much reduction in soil lead levels since the 1992 survey, except perhaps for risk zone 1. There has been a decrease in the geometric means of the risk zones in comparison to 1992 survey data. Additionally, the geometric means seem to indicate that risk zones 2 and 3 have merged (since concentrations are similar in these two zones). The same holds true for risk zones 4 and 5.

Figure 9-4: Lead risk zones in Broken Hill (after Boreland et al. 2009a).

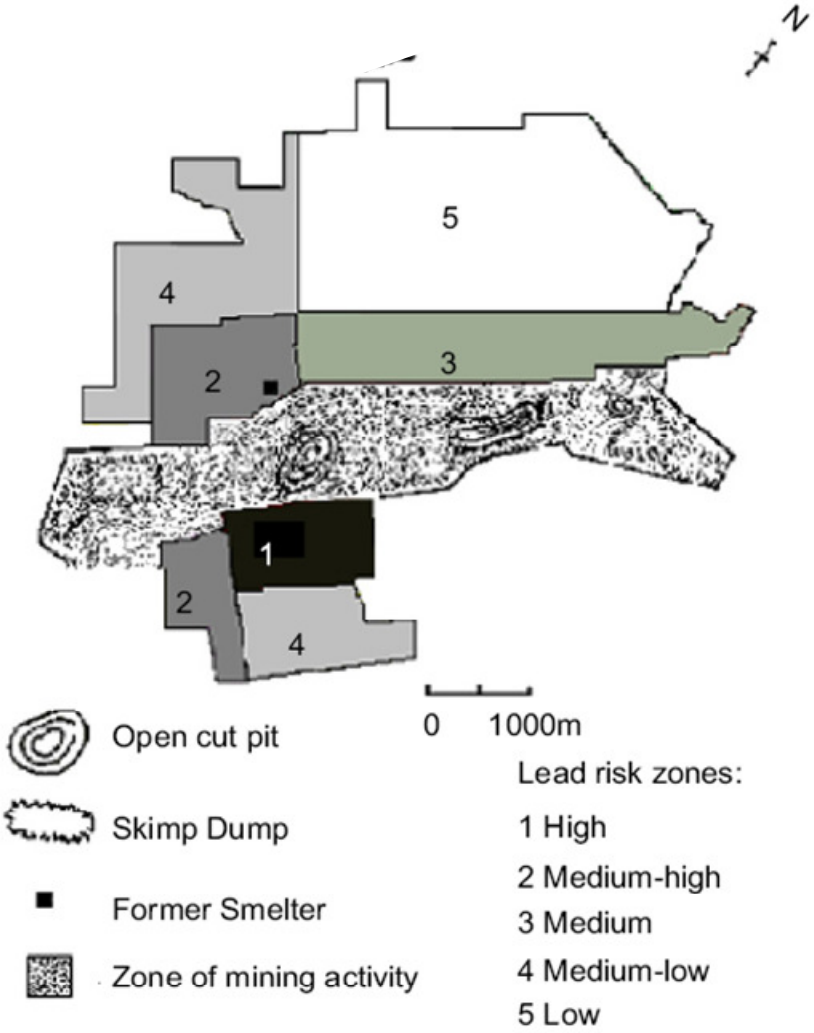


Table 9-2 Receptors selected for ease of reporting in the HHRA

Risk Zone	Soil Lead and Indoor Dust Levels Measured in 1992 (as published by Lyle et al. 2006)[a]		Soil Lead Measured during 2004-2008 (as obtained from Boreland 2010)
	Geometric Mean Soil Lead (mg/kg)	Geometric Mean Indoor Dust (µg/m^2/30 days)	Geometric Mean Soil Lead (mg/kg) [b]
1	1967	946	1350
2	794	717	540
3	621	490	580
4	365	216	260
5	262	201	220

[a] Soil lead concentration data is from the BHCC survey undertaken in 1992 as reported by Lyle et al. (2006) and described according to city districts by Boreland et al. (2002). These concentrations are prior to remediation work undertaken in the late 1990's and completed in 2003/4 (Boreland et al. 2008a).

[b] Soil lead levels for the 2004-2008 dataset (n=148 houses) as provided for Broken Hill districts by Boreland (2010). For comparison with the Lyle et al. (2006) soil lead data in this table the data provided by Boreland (2010) was translated to risk zones as per Boreland et al. (2009a) by averaging the geometric mean values for the districts within a risk zone. The average geometric means were rounded to the nearest ten.

Information on the extent of soil remediation was not located, however it appears residential remediation was limited to locations where children had high blood lead and may not have included many houses that had high soil concentrations. If this was the case then areas with the lower soil lead concentrations may not have been remediated and there may be many places where soil lead is already above the NEPM Health Investigation Level (HIL).

Background Soil Lead Concentrations used in the HHRA

In the HHRA, background soil lead concentrations were assumed from data reported for soil sampling undertaken in 2004-2008, which is the most recent soil data available. It should be appreciated that this data has not been derived via a systematic soil survey; rather the data has been gathered as part of the management response to children with high blood lead levels. Therefore the data is more reflective of high end soil concentrations rather than being representative of the specific risk zone.

The latest data suggests that sampled areas previously classified into five risk zones have been reduced to three risk zones: (a) risk zone 1, (b) risk zones 2 and 3 have merged into one (denoted risk zone 2+3 in this assessment), and (c) zones 4 and 5 have merged into one (denoted risk zone 4+5 in this assessment).

Geometric mean soil lead concentrations from the 2004-2008 soil sampling (from Boreland 2010) are summarised in *Table 9-2*. Background concentrations of soil lead used in the TDI assessment require a specific number for intake calculations. Based on the upper 95% confidence limits from the 2004-2008 data, the background soil lead levels were conservatively assumed as follows:

- Risk zone 1: Background concentration of soil lead assumed to be 2,000 mg/kg.
- Risk zones 2+3: Background concentration of soil lead assumed to be 1,000 mg/kg.
- Risk zones 4+5: Background concentration of soil lead assumed to be 500 mg/kg.

9.2.3 Incremental Soil Concentrations at Receptors due to the Mine Site

As indicated previously, soil lead concentrations at receptor sites were quantified for the following mine site related scenarios:

- **S1a** : Existing free areas in their current state
- **S1b** : Free areas with dust control to 80% efficiency
- **S2**: Project-related sources
- **S3**: Cumulative mine site related sources, including emissions from free areas with 80% control (S1b) plus Project-related emissions (S2)

Soil lead and other metal concentrations accumulating over 15 years of mine operation were considered for each of the scenarios.

The concentration of lead and other metals in soil was calculated based on the annual average deposition rates (mg/m²/annum) predicted by air dispersion modeling (as documented in Chapter 8). A soil mixing depth of 2 cm was adopted in estimating soil metal concentrations at receptor locations after 15 years of operation of the Rasp Mine. This soil mixing depth was based on a study that profiled dioxin measurements in soil, in which the US EPA (2005) recommend a soil mixing depth of 2 cm for untilled soils (US EPA 2005).

The environmental half life of the metals at each receptor was assumed to be infinite to provide a conservative (upper bound) estimate of soil metal concentrations, i.e. no provision for physical loss of metals from the soil due to wind erosion, percolation by rain or water runoff. The metal deposition rates were therefore multiplied by 15 years to represent soil metal concentrations at the end of the life of mine.

Calculated soil lead concentrations over 15 years of mine operations are presented in *Table 9-3* for each scenario evaluated. As expected, receptors close to operations of the proposed mine accumulate more lead over 15 years of mine operation than do locations further away. Thus, assuming no loss and 2 cm soil mixing depth, receptors R8, R3, R9 and R21 accrue 552 µg lead/g, 225 µg lead/g, 199 µg lead/g, and 205 µg lead/g soil respectively (for Scenario 3), but other receptors only accumulate 2 to 183 µg lead/g soil.

As the absorption of lead in the human body from different materials can vary (refer *Section 9.3*) estimates were made of lead concentrations from the following lead containing dust:

- 'free area' dust – comprising dust from existing exposed areas within the mine leas area and dust from haul roads.
- dust from mine activities ('ore') sources – represents dust from ore handling and stockpiling, crushing and screening and ventilation shaft emissions.

The percentage of lead concentration in the 'free area' dust is presented in *Table 9-3* for Scenario 3. Across the selected receptors, lead from 'free areas' (80% controlled) was calculated to account for between 49% (R12) and 96% (R32) of the total accumulated soil lead concentrations, with the balance derived from 'ore' sources.

The calculated soil concentrations given in *Table 9-3* were used as inputs in the evaluation of risks during the TDI analysis (*Section 9.5*) and the modelling of incremental blood lead concentrations in children (*Section 9.6*).

Table 9-3 Calculated soil lead concentrations for mine site related scenarios (representing 15 years of mine operation; assuming zero loss of lead)

Selected Receptor	Calculated Soil Lead Concentrations (mg/kg) after 15 years			
	Scenario 1a	Scenario 1b	Scenario 2	Scenario 3 (% of lead due to 'free areas',(80% controlled) given in brackets)
Residences				
1. Piper Street North	702	140	25	166 (91%)
2. Piper Street Central	427	85	55	140 (72%)
3. Eyre Street North	566	113	112	225 (63%)
4. Eyre Street Central	321	64	60	124 (65%)
5. Eyre Street South	241	48	45	93 (65%)
8. Old South Road	1646	329	223	552 (64%)
9. South Road (2)	612	122	76	199 (70%)
10. Garnet & Blende Streets	411	82	30	112 (83%)
23. Eyre Street North (3)	601	120	15	135 (94%)
32. Crystal Street (2)	346	69	7	76 (96%)
36. Crystal Street (5)	463	93	15	106 (92%)
38. Gypsum Street (1)	82	16	20	35 (57%)
Other Locations				
11. Alma Bugdlie Pre-School	168	34	28	62 (65%)
12. Playtime Pre-School	39	8	18	26 (49%)
14. Broken Hill High School	89	18	18	36 (67%)
17. Broken Hill Public School	20	4	10	15 (53%)
18. Rainbow Pre School	19	4	9	13 (50%)

9.2.4 Soil Concentrations used for Exposure Calculations

To assess the health risk using TDI analysis for lead, incremental soil concentrations (*Section 9.2.3*) were added to the assumed existing background soil lead concentration described in *Section 9.2.2*.

Incremental increases in blood lead levels in children at specific receptor locations were modelled using the incremental soil lead concentrations (*Section 9.2.3*).

To compare blood lead levels in children exposed to lead from only the free areas (Scenario 1a, no dust control) with the 'cumulative' scenario (S3, mine activity together plus free areas that are 80% dust controlled) in *Section 5.4.2*, the assumed variable background soil lead levels were added to the incremental levels.

For the TDI analyses for other metals, no background soil information was available. Therefore, incremental soil metal concentrations from mine activity and/or free areas were used.

9.3 BIOACCESSIBILITY

9.3.1 Bioaccessibility and Bioavailability

The fraction of an orally administered dose of chemical that reaches the systemic circulation is termed the oral bioavailability or absolute bioavailability (RIVM 2009).

Many substances are able to tightly bind to environmental matrices such as soil or sediment. The bioavailability of the substance from the media (e.g. soil, sediment) is dependent on two major processes:

- *Bioaccessibility:* This is the amount of contaminant released from the media (e.g. during digestion in the gastrointestinal tract) that is available to be absorbed. The solubility of the substance in gastrointestinal media is defined as its bioaccessibility.

- *Absorption:* Usually only part of the bioaccessible fraction is transported across the intestinal epithelium and reaches the systemic circulation. This is the absorbed fraction.

For soil, therefore, the bioaccessible fraction represents the amount of contaminant partitioned from the soil that is dissolved while in the gastrointestinal tract and therefore available for absorption. Overall bioavailability will then depend on how much of this bioaccessible fraction is actually absorbed and able to reach the systemic circulation.

The HHRA used the physiologically based extraction test (PBET) for determining the bioaccessibility of metals from surface soil at the mine site and from mine ore. This involved simulating the leaching of a solid matrix in the human stomach and small intestine under feed and fasting conditions. This test was conducted by enTox at Queensland University. Bruce et al. (2007) have used the PBET model in a risk assessment for assessing the bioavailability of lead and arsenic in mine waste from a Queensland mining operation.

9.3.2 Sampling of Site Dust for Bioaccessibility Analysis

The bioavailability of lead from soil and minerals can be very site specific. To estimate the bioavailability of lead in mine site dust that may be blown to residential locations, surface dust samples were collected from various areas of the site and, together with drill core samples of the Western Mineralisation ore body. These samples were sent to the University of Queensland for determination of bioaccessibility (EnTox 2009). These samples are respectively representative of soil/dust from waste rock left from various stages of mine life (i.e. ‘free areas’) and process related dust (i.e. ‘ore’).

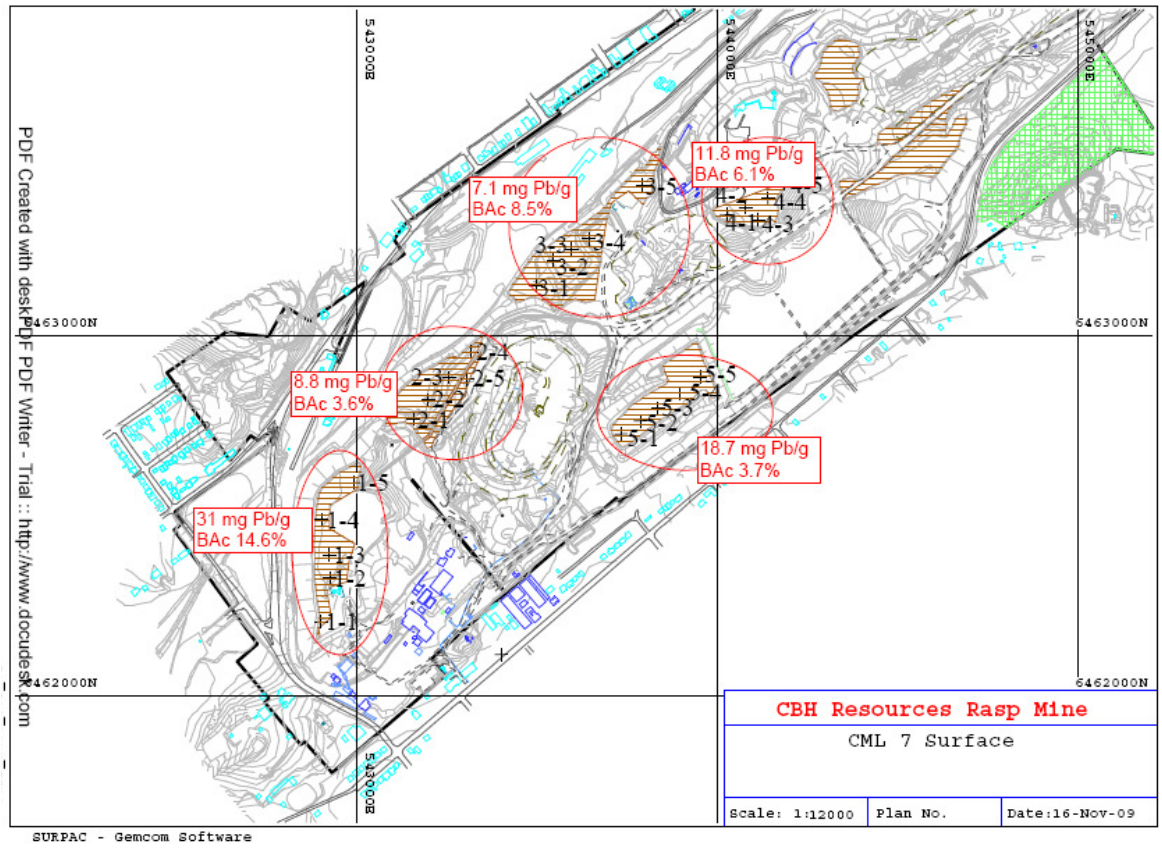
In the air dispersion modelling, the ‘free area’ material type was taken to be representative of dust emitted from roads on site (paved and unpaved) and wider wind exposed areas around mining operations. The ‘ore’ material type was assumed representative of other mining sources including ore handling and crushing, ore stockpile operations, ventilation shaft, the tailings storage facility and concentrate handling.

To analyse the bioaccessibility of lead from ‘free area’ dust five sub-samples of approximately 200 to 300g each were obtained from different locations within five designated areas (1 – 5) of the Rasp Mine site (*Figure 9-4*). Tests were performed on the fines of a 100 µm sieving as this is the particle size fraction subject to potential airlift from the ground.

To analyse the bioaccessibility of lead from ‘ore’ dust four drill core samples were broken up to small pieces with a hammer to roughly simulate the crushing process. The bioaccessibility of lead

in process dust was determined based on fines from a 150 µm sieving of the drill core samples since finer particles are more likely to become airborne and be transferred off-site.

Figure 9-5: Soil Lead Concentrations (mg lead/g soil) and Bioaccessibility (BAc, as percentage) for Soil Sample Composites.



9.3.3 Bioaccessibility Results

The physiologically based extraction test (PBET) was optimized for estimating the bioaccessibility of lead. The test has been formally validated for lead and arsenic (Ruby et al. 1996, 1999), but not for the other metals addressed in this risk assessment. Consequently bioaccessibility data for metals other than lead and arsenic are considered to be uncertain, with the bioaccessibility of such metals therefore assumed to be 100%.

The tests were performed in duplicate for each sample to determine lead and arsenic bioaccessibility, with duplicates showing reasonable concordance with each other. Lead and arsenic bioaccessibility results for surface area dust and mine ore material are summarised in *Table 9-4*.

Weathering of lead sulphide results in the formation of sulphate (Davis et al. 1993). Since this substance is more soluble than lead sulphide it would be expected that the bioaccessibility and bioavailability of weathered lead from the existing surface dust would be greater than that of the fresh ore. Indeed this is what was observed with the tests undertaken for the Rasp Mine site. Average bioaccessibility of lead from surface dusts was 7.3%, whereas that from mine ore was just 1.4%.

Together with mine site source apportionment of incremental lead at discrete receptors, the above average bioaccessibility values were used to calculate the lead bioaccessibility in incremental airborne PM_{10} and deposited lead to soil from TSP at receptor locations. The bioavailability of lead in air, soil and dust was modified for each modelled receptor according to the bioaccessibility mix of the source apportionment. Thus, receptor-specific bioaccessibilities were calculated.

Table 9-4 Lead and Arsenic Bioaccessibility for Surface Area Dust and Mine Ore

Sample	Bioaccessibility (BAc) (%)[a]	
	Lead	Arsenic
Average bioaccessibility from site area dust	7.3	4
Average bioaccessibility from mine ore	1.4	4 (0.4) [b]

[a] Results provided by laboratory as the average of the duplicate samples analysed.

[b] Although ore samples give an average BAc for Arsenic of about 0.4%, the BAc of Arsenic was conservatively assumed to be 4% for both surface dust and ore samples, due to only two of the ore samples returning results (i.e. BAc or mine ore made equivalent to site area dust BAc).

For the IEUBK modelling, bioaccessibilities for lead in soil/dust were converted to an absolute bioavailability by multiplying the receptor-specific bioaccessibility by 50%, which corresponds to the bioavailability of soluble lead in water and food (US EPA 1999, ATSDR 2007) (Refer to *Section 9.4.1*). The calculated receptor-specific bioavailabilities were then used as input values for determining blood lead levels. However, in the tolerable daily intake calculations a bioavailability of 100% was used so the overall bioavailability was the same as the bioaccessibility.

Source apportionment information by receptor was not available for arsenic. Therefore, the calculated arsenic bioaccessibility from the PBET test (4%) was applied to all receptors.

9.4 TOXICOLOGICAL INFORMATION FOR LEAD

Detailed toxicological hazard information on lead and other major metals can be found in the HHRA documentation. The metal of most concern for this risk assessment is lead and a brief description of its hazards is provided in this subsection.

9.4.1 Kinetics

With exposure, mainly by ingestion and inhalation, a portion of lead is absorbed and distributed to various body compartments from which it is eliminated at various rates. The absorption and distribution of lead varies depending on duration and intensity of the exposure, age, and various physiological variables (e.g. nutritional status, pregnancy, and menopause) (ATSDR 2007).

Absorption of lead deposited in the respiratory tract is influenced by particle size and solubility, as well as by the pattern of regional deposition within the respiratory tract. Fine particles (<1 µm) deposited in the bronchiolar and alveolar region can be absorbed after extracellular dissolution or can be ingested by phagocytic cells and transported from the respiratory tract (ATSDR 2007). In quantitative studies with human volunteers the proportion of lead particles absorbed was approximately 95% of the deposited lead within the bronchiolar and alveolar region (ATSDR 2007, USEPA 2006). Larger particles (>2.5 µm) that are primarily deposited in the ciliated airways (nasopharyngeal and tracheobronchial regions) can be transferred by mucociliary transport into the oesophagus and swallowed (ATSDR 2007).

The extent and rate of gastrointestinal absorption of ingested inorganic lead are influenced by physiological states of the exposed individual (e.g., age, fasting, nutritional calcium and iron status, pregnancy) and physicochemical characteristics of the lead-bearing material ingested (e.g., particle size, mineralogy, solubility, lead species) (US EPA 2006).

Human studies investigating the absorption of water soluble lead compounds indicate that 40-50% of ingested lead is absorbed in children (2 week old infants to approximately 8 year old children)

while only 3-10% of ingested lead is absorbed by adults (ATSDR 2007, US EPA 2006). The difference is thought to be due to differences in physiological and dietary factors.

Dermal absorption of inorganic lead compounds is considered to be a minor route of entry into the body (ATSDR 2007, US EPA 2006). Few studies have provided quantitative estimates of dermal absorption of inorganic lead in either humans or animals. Those that have been conducted however consistently show the absorption to be very low for both soluble and insoluble lead compounds.

Bioavailability factors selected for the present HHRA are summarised in *Table 9-5*.

Table 9-5 Bioavailability Factors using the HHRA [a]

Exposure Route	Absorption Factor	Basis
Oral	50%	Human studies investigating the absorption of water soluble lead compounds indicate that 40-50% of ingested lead is absorbed in children (2 week old infants to approximately 8 year old children). US EPA 2006, ATSDR 2007.
Inhalation	100%	In quantitative studies with human volunteers the proportion of lead absorbed was approximately 95% of the soluble deposited lead within the bronchiolar and alveolar region. US EPA 2006, ATSDR 2007.
Dermal	0.01%	Quantitative estimates from a comparative study of dermal absorption of inorganic lead in rats showing a dermal absorption for most inorganic lead compounds (both relatively soluble and insoluble) to be less than 0.01% (lead nitrate (0.03%), lead stearate (0.006%), lead sulfate (0.006%), lead oxide (0.005%), and metal lead powder (0.002%). US EPA 2006, ATSDR 2007.

[a] The above bioavailabilities were modified for bioaccessibility as per *Section 9.3.3*.

Throughout life, lead in the body is exchanged between blood and bone, and between blood and soft tissues, with variation in these exchanges reflecting duration and intensity of the exposure, age and various physiological variables (US EPA 2006). The site of accumulation in bone is dependent on the most active areas of calcification at the time of exposure. A larger fraction of the lead body burden of adults resides in bone (approx. 90%) than in children (approx. 70%) (ATSDR 2007, US EPA 2006). Approximately 1% of the lead body burden is found in blood (primarily in red blood cells bound to protein) (US EPA 2006). Bone lead is essentially inert, having a half-life of several decades. A labile compartment exists as well that allows for maintenance of an equilibrium of lead between bone and soft tissue or blood (ATSDR 2007, US EPA 2006). The labile phase, exhibited shortly after a change in exposure occurs, has a half-life of approximately 20 to 30 days (US EPA 2006 p 4-18). A slower phase becomes evident with longer observation periods following a decrease in exposure. The half-life of this slow phase has been estimated to be approximately 3 to 30 years and appears to correlate with finger bone lead levels and is thought to reflect the release of Pb from bone stores to blood (US EPA 2006 p 4-18).

9.4.2 Toxicity

In humans, lead can result in a wide range of biological effects depending upon the level and duration of exposure. Effects may range from inhibition of enzymes to the production of marked morphological changes and death. Such changes occur over a broad range of exposures. For neurological, metabolic and behavioural reasons, children are more vulnerable to the effects of lead than adults (UNEP 2006, ATSDR 2007). *Table 9-6* provides a summary of the blood lead levels associated with a range of effects.

Table 9-6 Effects Associated with Blood Lead Levels [a]

Blood lead (µg/dL)	Duration of exposure	Effect
100–120	*Acute*	Restlessness, irritability, poor attention span, headaches, muscle tremor, abdominal cramps, kidney damage, hallucinations, and loss of memory, encephalopathy in *adults.*
80–100	*Acute*	Restlessness, irritability, poor attention span, headaches, muscle tremor, abdominal cramps, kidney damage, hallucinations, and loss of memory, encephalopathy in *children.*
50–80	Chronic	Signs of chronic toxicity including tiredness, sleeplessness, irritability, headaches, joint pain, and gastrointestinal symptoms in adults.
>50	Chronic	Depressed haemoglobin in adults.
40–60	Chronic	Muscle weakness, gastrointestinal symptoms, lower scores on psychometric tests, disturbances in mood, and symptoms of peripheral neuropathy in occupationally exposed adults.
30–40	Chronic	Reduced fertility in adults.
>40	Chronic	Depressed haemoglobin in *children*, neurobehavioural effects in *adults*.
>30	Chronic	Depressed nerve conduction velocity in *children*, liver damage in *adults*.
>15	Chronic	Depressed vitamin D in *children*.
<10	Chronic	Neurodevelopmental effects in *children*; inhibition of sexual maturation in *children*; depressed δ-aminolevulinic acid dehydratase and glomerular filtration rate in *adults*.
<5	Chronic	Depressed δ-aminolevulinic acid dehydratase in *children*, neurobehavioural effects in elderly *adults*.

[a] From NZ MfE (2010) which is based on ATSDR (2007).

The evidence for carcinogenicity of lead and several inorganic lead compounds in humans is inadequate. Classification carcinogenicity by IARC is Class 2B *'The agent (mixture) is possibly carcinogenic to humans. The exposure circumstance entails exposures that are possibly carcinogenic to humans'* (IARC 1987).

The critical effect of particular concern for environmental exposures to the general public is the effect of lead on the central nervous system. Epidemiological studies suggest that low level exposure of the foetus and developing child may impair the learning capacity and the neuropsychological development. Studies of children indicate a correlation between higher lead contents in the blood and a lower IQ (WHO 1995, ATSDR 2007). The studies used to investigate these endpoints are not precise and the outcomes are influenced by such things as genetics, socio-economic status and early life experience/environment (NHMRC 2009).

Given the imprecise nature and outcomes the NHMRC (2009) concluded that it is not possible to make a definitive statement on what constitutes a 'safe level' or 'level of concern' for blood lead and recommend a blood lead level below 10 µg/dl for all Australians.

9.4.3 Interactions with Other Metals

Interactions of lead with other metals vary; depending on the endpoint measured, the tissue analysed, the animal species, and the metal combination (US EPA 2006 p E20). Two of the most commonly reported lead-element interactions are between lead and calcium and between lead and

zinc. Both calcium and zinc are essential elements in organisms and the interaction of lead with these ions can lead to adverse effects both by increased lead uptake and by a decrease in calcium and zinc required for normal metabolic functions (US EPA 2006 p E23).

9.4.4 Susceptibility of Children

Children, in general and especially low socioeconomic status children, have been well-documented as being at increased risk for lead exposure and lead-induced adverse health effects. Children and developing organisms in general, are more susceptible to lead toxicity than adults. As reviewed by the ATSDR (2007) the factors that result in higher susceptibility can include:

- Children exhibit more severe toxicity at lower exposures than adults, as indicated by lower blood lead concentrations and time-integrated blood lead concentrations. The mechanism for this increased vulnerability is not completely understood but is thought to be related to key processes (e.g. cell migration and synaptogenesis) during brain development.

- Children also absorb a larger fraction of ingested lead than do adults; thus, children will experience a higher internal lead dose per unit of body mass than adults at similar exposure concentrations. Absorption of lead appears to be higher in children who have low dietary iron or calcium intakes; thus, dietary insufficiencies, which are not uncommon in lower socioeconomic children, may contribute to their lead absorption.

- Infants are born with a lead body burden that reflects the burden of the mother. During gestation, lead from the maternal skeleton is transferred across the placenta to the foetus and additional lead exposure may occur during breast feeding.

- Behavioural patterns of children, particularly hand to mouth activity, can result in higher rates of ingestion of soil and dust, both of which are often important environmental depots for lead leading to a higher potential for intake of lead.

9.5 RISK CHARACTERISATION – TOLERABLE DAILY INTAKE (TDI) ANALYSIS

Metal intake from all complete exposure pathways was calculated and compared to the tolerable daily intake (TDI) set by WHO/JECFA (1986, 2000). This is a standard risk assessment procedure (enHealth 2004).

Human uptake of environmental chemicals is not only reliant upon the concentration of the chemical in environmental media but also on age dependent behaviour factors. For example, children are more likely to accumulate soil on their hands and they have greater hand-mouth transfer rates. Due to age dependent ventilation rates and differing body weight throughout life, intake (i.e. dose) varies.

The TDI comparison was performed using daily intake of lead for:

- infants/toddlers (0.5 up to 3 years),
- children (3 up to 13 years),
- adolescents (13 up to18 years), and
- adults (18 up to 70 years).

In addition to the age interval specific comparisons, the overall lifetime weighted daily intake over all life stages, called the time weighted average daily intake or TWADI, has been compared to the TDI.

The incremental risks due to metals other than lead were found to be negligible, as documented in *Section 9.5.3*. For this reason, the overview of the exposure calculations provided in this section makes reference primarily to lead.

9.5.1 Exposure Calculations

For the TDI analysis, three exposure pathways are considered for residents:

- Soil ingestion
- Dermal absorption of metal from soil
- Inhalation of airborne dust delivered from mine site

Pathway-specific equations were applied in the exposure calculations. In addition to parameters taken into account in such calculations, as outlined below for lead, reference was made to exposure time (hr/day), exposure frequency (day/year), age-interval specific exposure duration (years), and age-specific body weight and averaging time.

Soil ingestion

Consideration was given to the ingestion pathway for the incidental ingestion of soil/dust. Estimated daily intake via ingestion of soil/dust was calculated based on soil lead concentrations (from air dispersion modelling), age-specific rates of soil ingestion, bioaccessibilities of metal in soil (*Table 9-4*), and bioavailability of ingested metal. Bioavailability of ingested lead was assumed to be the same as the bioavailability in the study from which the TDI was derived (i.e. 100%).

Dermal absorption

Factors taken into account in calculating dermal adsorption included the soil lead concentration (from air dispersion modeling), soil adherence rates, age-specific surface skin areas, bioaccessibility of lead in soil and absolute bioavailability of dermally absorbed lead.

Dermal absorption of inorganic lead is much lower than by ingestion or inhalation (ATSDR 2007, US EPA 2006). Given that lead from the Rasp Mine is predominantly in the form of weathered galena (anglesite, or Pb sulphide), and has low bioaccessibility (*Table 9-4*) the absorption of mining lead through the skin is expected to be negligible. In these considerations the absolute bioavailability of lead via the dermal pathway was taken to be 0.01%.

Inhalation of airborne dust delivered from mine site

Inhalation exposures to airborne dust from the mine site were calculated based on the modelled concentration of airborne lead in the PM_{10} fraction, the age-specific inhalation rate and the bioaccessibility of the lead from tests done on site area dust and ore samples (Refer to *Section 9.3.3*). The absolute bioavailability of inhaled lead was taken to be 100%.

Background intake

Given that the hazard index calculated for metals other than lead was determined to be trivial (*Section 9.5.3)* a more detailed consideration of background intakes of such metals was not warranted.

Background intake of lead from all sources was incorporated into the intake calculations for this metal. Age-specific background intake of lead consists of intake from soil, air, food, water and consumer goods.

The intake from soil was based on the upper 95% confidence limit soil lead concentrations sourced from Boreland 2010 and varied by risk zone, as discussed in *Section 9.2.2*.

The intake of lead in background air was based on average airborne lead concentrations measured at the Rasp Mine (0.2 µg/m³), as documented in *Section 8.1.5* and age-specific inhalation rates.

All background intakes of lead from soil and air have been calculated using the bioaccessibility of the "free areas" profile (i.e. 7.3%). This is based on the assumption that all existing lead at

receptor locations has the same characteristics and bioaccessibility as the lead in the free areas, even though existing lead at receptor locations may have been derived from multiple sources.

Background dietary intakes were obtained from Food Standards Australia New Zealand (FSANZ 2003) and are for the general Australian population; it should be noted it is uncertain whether these values are representative of the Broken Hill population. FSANZ (2003) reported ranges for mean dietary intakes of Pb for 9-month old infants, 2-year old toddlers, 12-year old children, and 25-34 year old adults. From this data background intake of lead from diet was assumed to be at or about the top of the range for the age group, i.e. 1 µg/kg/d for 0.5-3 year olds, 0.9 µg/kg/d for 3-13 year olds, and 0.4 µg/kg/d for the other two age groups.

In terms of lead intake from water, it was assumed that tank water is not used for drinking water purposes, as recommended by the local authorities (NSW Gov 2009). The maximum lead concentration measured in the reticulated water supply in 2009 was <0.5 µg/L (Countrywater 2009). This concentration was used to calculate background intakes of water, assuming age-specific body weights and drinking water intakes.

The intake of lead through the use of consumer goods was assumed to be zero since there are many regulations in place that limit the amount of lead in consumer goods such as toys, crayons, and cosmetics.

Total age-specific daily intake

As indicated, total daily intakes were calculated for the following age groups: infants/toddlers (0.5 up to 3 years), children (3 up to 13 years), adolescents (13 up to18 years), and adults (18 up to 70 years).

Total daily intakes comprised the addition of intakes via soil ingestion, dermal absorption and inhalation of airborne lead from the mine site, with background intake of lead.

Lifetime time-weighted average intake (TWADI)

In calculating the TWADI, age-interval specific daily intakes were multiplied by the averaging time (days) of such intervals and the product summed across age groups. This sum was then divided by the averaging time for lifetime exposure (i.e. 70 years x 365 days/year = 25,550) to obtain the overall lifetime weighted daily intake over all life stages for comparison to the TDI.

Hazard quotients

The hazard quotient (HQ) was calculated by dividing the calculated age-interval specific or lifetime time-weighted average intake of a metal with the relevant TDI for that metal. A summary of the TDIs taken from reputable sources for application in the HHRA is given in *Table 9-7*.

Table 9-7 TDIs Applied in the HHRA

Metal	TDI (µg/kg/d)	Reference
Antimony	6	WHO (2008)
Arsenic	2	JECFA (1983a; 1988a)
Barium	20	RIVM (2001)
Beryllium	2	WHO (2001)
Cadmium	1	JECFA (1988b)

Metal	TDI (µg/kg/d)	Reference
Chromium (Cr as Cr^{III})	3	UK EA (2002)
Copper	140	WHO (2008)
Iron	800	JECFA (1983b)
Lead	3.5 (based on the PTWI of 25)	JECFA (1986, 1999)
Manganese	140	US EPA IRIS (1996)
Mercury	2	WHO (2003)
Nickel	12	WHO (2008)
Silver	400	NHMRC (2004)
Zinc	500	NHMRC (2006)

PTWI - Provisional tolerable weekly intake.

9.5.2 Exposure Assumptions

Exposure calculations rely on a number of assumptions for exposure in the absence of site-specific data. Exposure parameters used in the study for each life stage are documented in the HHRA documentation. Where possible the HHRA incorporated exposure factors from contemporary Australian studies. Where this was not appropriate, data from recently published US Exposure Factors Handbooks (US EPA 2008, 2009a) were applied, with reasoning provided as to the selection of such data.

Age-specific physiological parameters (e.g. body weight, inhalation rate etc.) for each age group were assumed to correspond to the mean age in the age group. For example, for the 0.5-3 year life stage, physiological parameters were assumed to be equivalent to those of a 2 year old child ([0.5 + 3]/2 = 1.75 yrs, rounded to 2 yrs).

9.5.3 Metal Hazard Quotients and Hazard Index (excluding Lead)

The TDI analysis was carried out with 13 additional metals (other than lead) for which the air dispersion modelling provided data for metal concentration in PM_{10} and deposition to soil. The hazard quotients for these metals, as given in *Table 9-8*, were calculated to be very low for all mine site related emission scenarios. The hazard quotients in the table are calculated for the most sensitive life stage. The highest hazard quotients were calculated for manganese, chromium and iron; these being 0.008, 0.007, and 0.007, respectively. That is the estimated intake is just 0.7 to 0.8% of the respective TDI for these metals.

The hazard index, which is the sum of hazard quotients, is a screening risk assessment technique commonly used to judge whether there is concern for additive effects between chemicals. If the hazard index is less than unity (1.0) it indicates that even if all the metals acted on the same organ or interacted in some other way to cause health effects the risk of these effects would still be low. The hazard index calculated across all metals other than lead was determined to be 0.05 for Scenario 3 (*Table 9-8*).

The incremental increase in exposure to metals other than lead was concluded to be negligible and unlikely to materially change current background exposures to these metals. The grounds for this conclusion being reached are twofold: (i) the incremental hazard quotients of the thirteen individual metals, listed in *Table 9-8*, are each less than 0.01 (i.e. each is less than 1% of the TDI), and (ii) the combined hazard index for such metals is only 0.05.

Table 9-8 Hazard Quotients for the Intake of Metals Other than Lead for a Child at Receptor 8 given Mine Site Related Emission Scenarios

Metal	Hazard Quotients by Scenario		
	S1b	S2	S3
Arsenic	0.003	0.003	0.003
Silber	0.00002	0.00002	0.00002
Barium	0.0003	0.0003	0.0003
Beryllium	0.003	0.003	0.003
Cadmium	0.006	0.006	0.006
Chromium	0.007	0.002	0.007
Copper	0.001	0.0003	0.001
Iron	0.005	0.002	0.007
Mercury	0.004	0.003	0.004
Manganese	0.008	0.0001	0.008
Nickel	0.001	0.0005	0.001
Antimony	0.002	0.001	0.003
Zinc	0.002	0.004	0.006
		Hazard Index	**0.05**

9.5.4 Lead Intakes and Hazard Quotients

Table 9-9 summarises total lead intakes by risk zone for each of the four age groups. From the table it is evident that the vast majority (>95%) of lead intake is from assumed background (diet, assumed existing soil lead concentrations, water and air). Of this, intake from diet and existing soil lead each contribute about 50%.

Intake from the mine lease area (existing free areas and mine activity related, i.e. Scenario 3) is a minor contributor to overall intake, comprising less than 10% of the TDI for all age groups (*Table 9-9*).

Mean intakes as a percentage of the TDI decreases with increasing age (*Table 9-9*). Calculated lead intake by the infant/toddler is the highest of all the age groups (about 60% of the TDI) and therefore represents the most sensitive population sector. However since the exposure assumptions are conservative and background intake assumptions were for high end exposures the fact that total lead intake for this age group is less than the TDI indicates low risk.

Incremental intake of lead due to mine site related emissions are depicted by pathway for an infant/toddler and an adult for two example receptors, Receptors 3 and 8, in *Figure 9-6*. Ingestion is noted to account for over 90% of total intake in the majority of cases.

The mean lead intakes estimated for Scenario 3 – including background, incremental mine site related and total intakes – are given as a percentage of the TDI for each Risk Zone in *Figure 9-7*.

Table 9-9 Total Lead Intake (Background + 'Cumulative' Incremental Intake from the Mine Site, i.e. Scenario 3) given in µg lead/kg bw/d [a]

Assumed Existing Soil Lead Concentration (mg/kg) and Corresponding Risk Zone [b]		Receptors located within Risk Zone		Age (yrs)			
				0.5-3	3-13	13-18	18-70
2000	RZ 1	R1-R4 R8[c]	**Range**	2.06-2.21	1.36-1.43	0.56-0.58	0.51-0.53
			Mean Intake	2.1	1.38	0.57	0.52
			Mean intake as % TDI	**60**	**39**	**16**	**15**
			% total intake from background	96	97	98	98
			% total intake from mine lease 'cumulative' exposure	4	3	2	2
1000	RZ 2	R10, R14 R38-R39	**Range**	1.54-1.57	1.14-1.15	0.49-0.5	0.46-0.47
			Mean Intake	1.55	1.14	0.49	0.46
			Mean intake as % TDI	**44**	**33**	**14**	**13**
			% total intake from background	99	99	99	99
			% total intake from mine lease 'cumulative' exposure	1	1	1	1
1000	RZ 3	R5-R7 R9, R11 R21-R37 [c] R40-R42	**Range**	1.55-1.62	1.14-1.18	0.49-0.5	0.46-0.47
			Mean Intake	1.58	1.16	0.5	0.47
			Mean intake as % TDI	**45**	**33**	**14**	**13**
			% total intake from background	96	98	98	99
			% total intake from 'cumulative' exposure	4	3	3	1
500	RZ 4	R12-R13 R18 [c]	**Range**	-	-	-	-
			Mean Intake	1.29	1.0	0.46	0.44
			Mean intake as % TDI	**37**	**29**	**13**	**1**
			% total intake from background	99.6	99.8	99.8	99.9
			% total intake from mine lease 'cumulative' exposure	0.4	0.2	0.2	0.1
500	RZ 5	R15-R17 R19-R20	**Range**	1.28-1.29	-	-	-
			Mean Intake	1.28	1.0	0.46	0.44
			Mean intake as % TDI	**37**	**29**	**13**	**13**
			% total intake from background	99.9	99.9	99.9	99.9
			% total intake from 'cumulative' exposure	0.1	0.1	0.1	0.1

[a] The intakes in the table are for the mean of the receptors nominated as being in each risk zone.

[b] 'Existing' soil concentrations are based on the upper 95% confidence limit soil lead concentrations sourced from Boreland 2010 and varied by risk zone, as discussed in **Section 9.2.2**

[c] Receptor 8 is located on the mine lease however for the purposes of calculating hazard quotients has been assumed to be in risk zone 1. R21-R33 have been assumed to be in risk zone 3. R18 has been assumed to be in risk zone 4.

Figure 9-6: Contribution of Intake Pathway to Incremental Intake of Lead due to Mine Site Emissions for an Infant/Toddler and Adult at Receptors 3 and 8

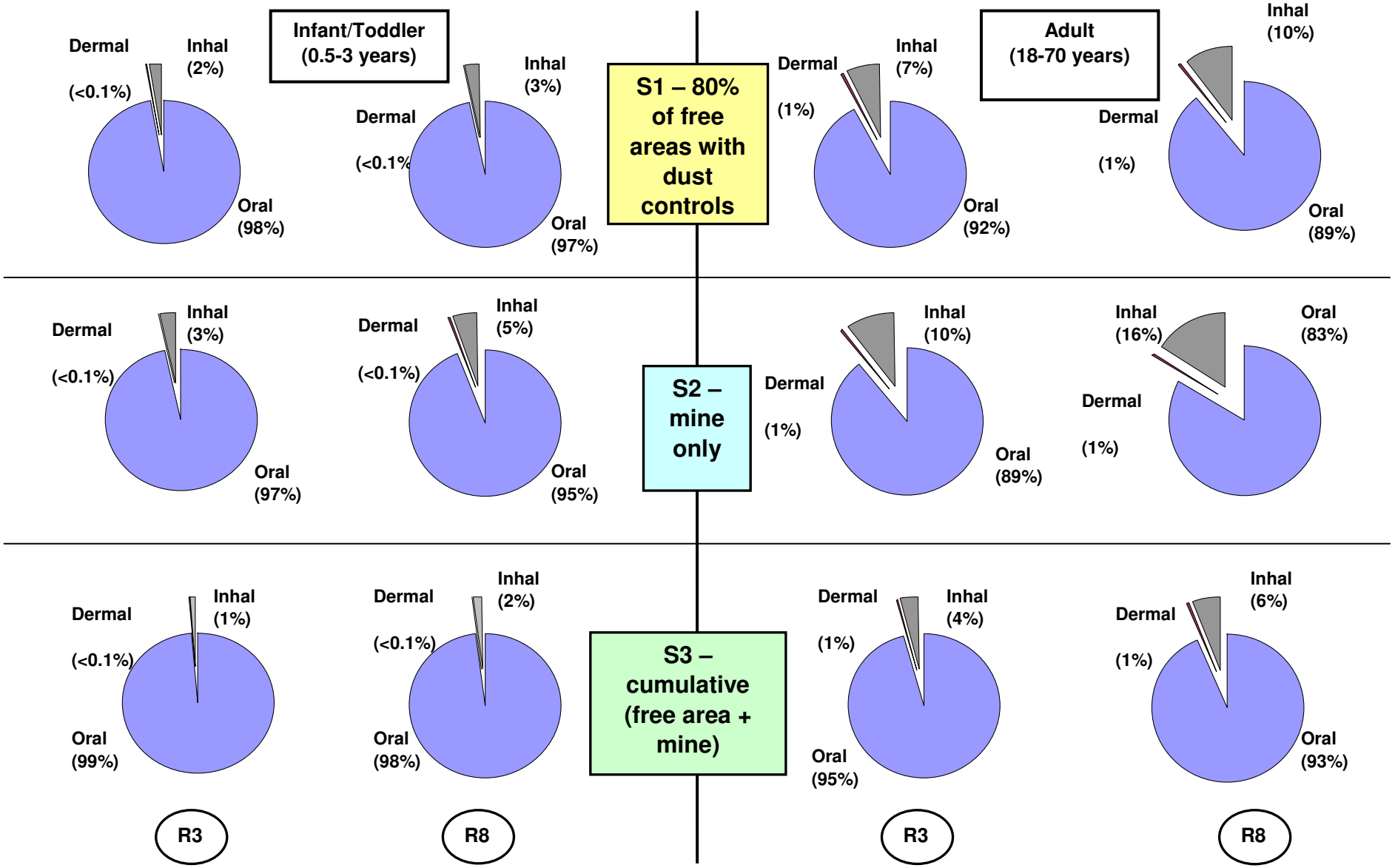
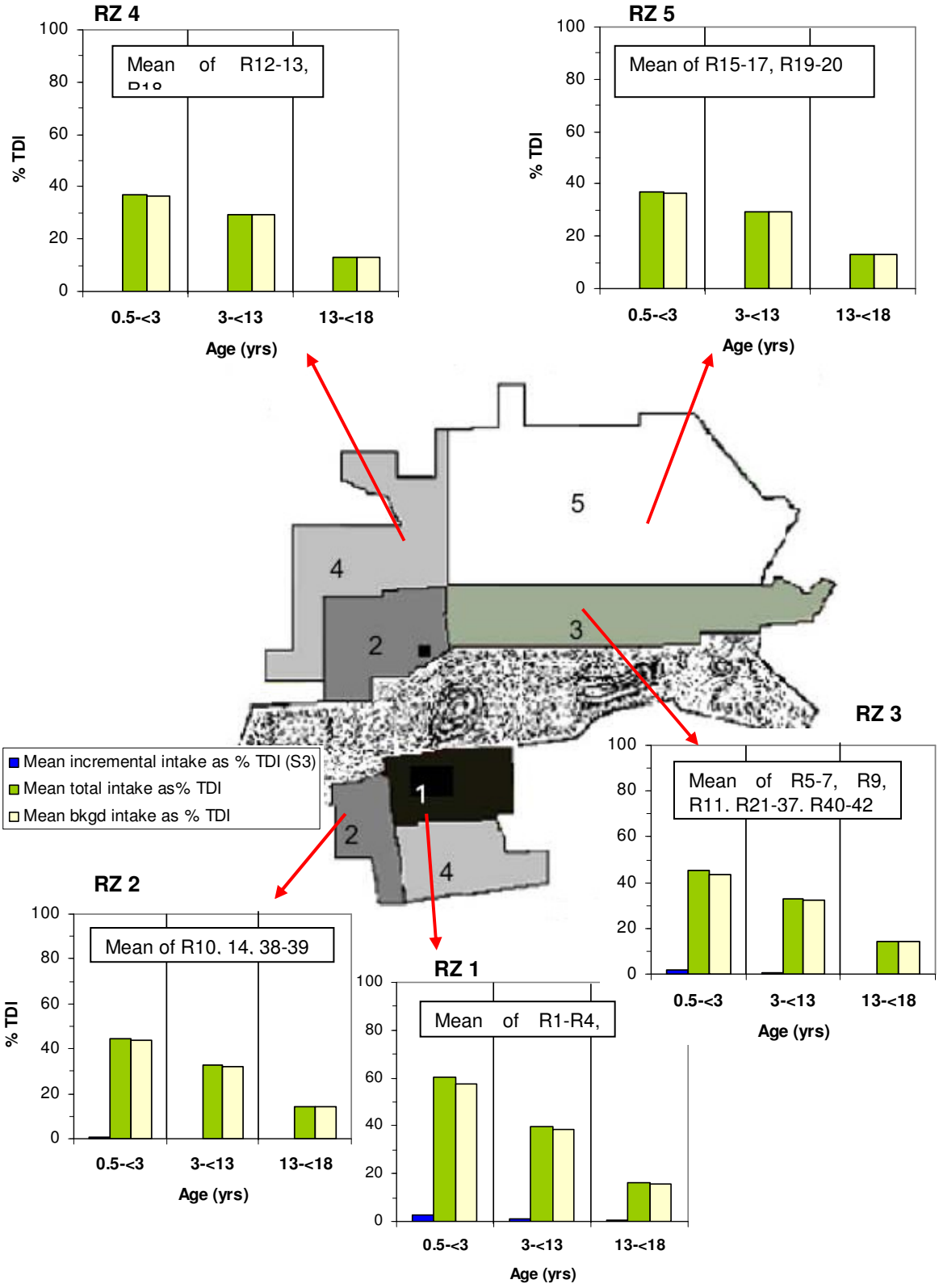


Figure 9-7: Mean Estimated Lead Intakes (Incremental, Background and Total) as a Percentage of the TDI for Scenario 3, given per Risk Zone [a]



[a] Note the incremental increases in some risk zones are so small as not to be seen in the graphs.

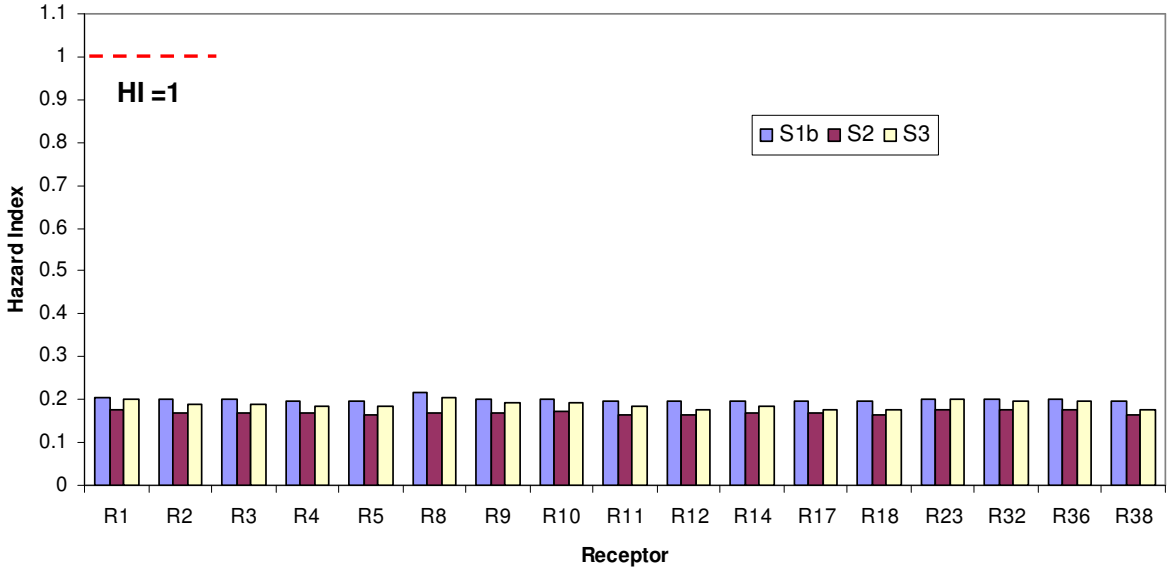
9.5.5 Hazard Index including Lead

Hazard indices were calculated by summing the hazard quotients for all 14 metals (including lead) evaluated in the risk assessment. The hazard quotients for lead were calculated including available background intakes. The hazard quotients for the other metals did not include background due to the incremental levels of such metals being found to result in a negligible fraction of the respective TDIs.

The level of conservatism underlying the assumption of additive effects is noted. Additive or synergistic effects by chemicals are only likely to occur if chemicals were affecting the same tissue types in an equivalent manner. A more detailed discussion of the potential target organs of concern is respect of individual metals is given in the HHRA documentation. For the purpose of the HHRA however, all chemicals included were conservatively assumed to act in an additive manner.

Figure 9-8 shows the combined metal hazard indices at selected receptors for Scenarios 1b, 2, and 3. For conservatism, lead hazard quotients corresponding to intakes with the highest background soil concentrations (i.e. Risk Zone 1) were used in calculating the hazard indices. The resulting hazard indices are all markedly less than 1.0 with the highest being of the order of 0.2. Lead contributes about 98% of the hazard indices due to the assumed high background.

Figure 9-8: Hazard Indices for Metal Intakes (including Lead) by Receptor for Scenarios S1b, S2 and S3 (including Background Intakes in the case of Lead)



9.6 RISK CHARACTERISATION – PREDICTED BLOOD LEAD

The NHMRC (2009) recommends that *all* Australians should have blood lead levels below 10 µg/dL. Given that dose-response data for lead is based on blood concentrations and that the management goal of the NHMRC for public health is expressed as a blood lead level, it is pertinent to consider background blood lead levels in Broken Hill and to predict blood lead concentrations associated with environmental lead exposure from the proposed Rasp Mine. Furthermore it is important that blood lead levels be considered for the most susceptible sub-population, i.e. children.

9.6.1 Background Blood Lead levels

The Greater Western Area Health Service (GWAHS 2009) has published information on blood lead levels for people in Broken Hill, this includes geometric mean blood lead concentrations for antenatal women and children aged 1-4 years old for each year from 1995-2008. Over the last

decade there has been a steady decline in blood lead concentrations in these population sectors. The data for 2005 – 2008 was expanded by GWAHS with additional summary statistics which included the minimum, maximum, 95th percentile, and standard deviation of blood lead levels.

In 2008, antenatal women (n=120) living in Broken Hill had a geometric mean blood lead concentration of 1.3 µg/dL, with a standard deviation of 1.03 µg/dL (*Figure 9-9*).

The geometric mean blood lead level for 1-4 year old children for blood lead measurements from 2005-2008 in Broken Hill was 5.6 µg/L, with a standard deviation of 6 µg/L (*Figure 9-10*). For children it appears as if a plateau has been reached at around 2005, however this may be influenced by the fact that the data is not the result of a random systematic survey of the population.

Figure 9-9: Geometric Mean Blood Lead Concentrations of Antenatal Women in Broken Hill, Measured from 1995 to 2008 (GWAHS 2009; Lesjak, 2010)

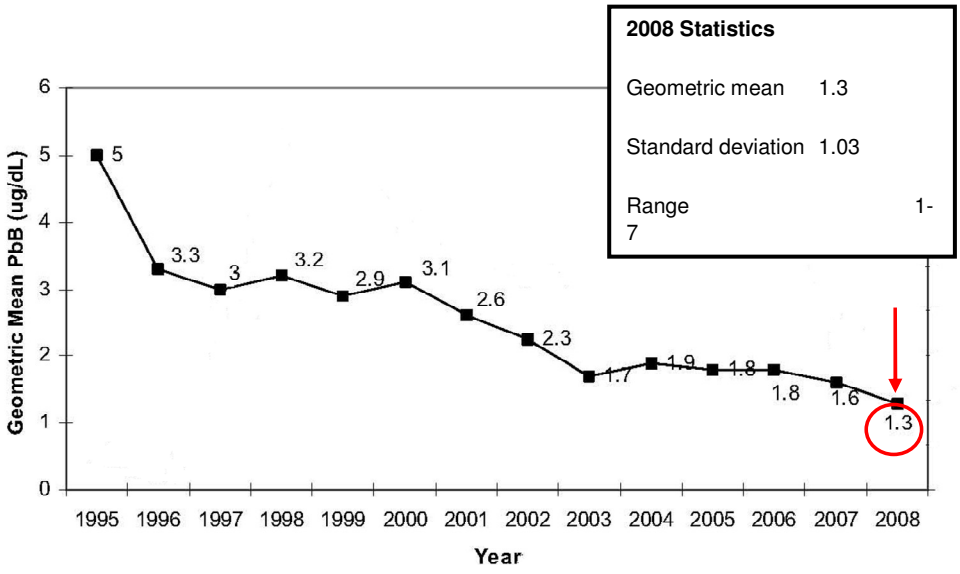
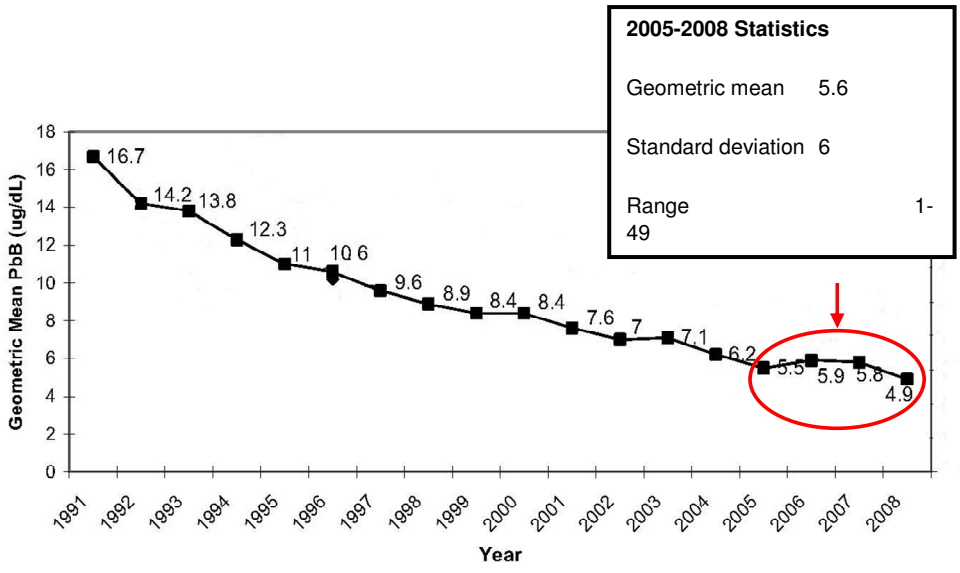


Figure 9-10: Geometric Mean Blood Lead Concentrations of Children Aged 1 to 4 years in Broken Hill, as measured from 1995 to 2008 (GWAHS 2009; Lesjak, 2010)



9.6.2 Modelling of Blood Lead

The US integrated exposure uptake and biokinetic model (IEUBK) was used to predict blood lead levels in children due to exposure to mine emissions and/or assumed background concentrations of lead in soil. This model has been validated and is regularly updated by the US EPA, it is extensively applied in North America to predict blood lead concentrations in children exposed to lead in their environment. The IEUBK model is used in regulatory decision making in the US and in health risk assessments undertaken by the Centre for Disease Control (CDC). The model has also been used in Australia to inform deliberations in establishing a health investigation level (HIL) for lead in soil (NEPM 1999).

An overview of the modelling approach used in the HHRA to predict blood lead levels is given in *Figure 9-11*. The blood lead modelling for children was carried out with the IEUBK model with the help of a number of site-specific inputs. One of these inputs was the maternal blood lead level, which was predicted using the *Adult Lead Methodology* (ALM).

IEUBK Model

The IEUBK model for lead is designed to mimic exposure to lead in air, water, soil, dust, diet, and paint, as well as other sources to predict blood lead levels in children aged 6 months to 7 years old. It caters for exposure via ingestion of soil and indoor dust, diet, and water, inhalation of airborne outdoor and indoor lead.

The four main components of the IEUBK model are:

- an **exposure module** that relates environmental lead concentrations to age-dependant intake via inhalation and ingestion,
- an **absorption (or uptake) module** that relates lead intake to lead uptake ,
- a **biokinetic module** that relates the uptake to tissue concentrations, including blood, and
- a module for **uncertainty** in exposure and for **population variability** in absorption and biokinetics.

The IEUBK model uses the above interrelated modules to estimate blood lead levels in children exposed to lead-contaminated media, and constructs a plausible distribution of blood lead concentrations centered on a geometric mean blood lead concentration. The geometric mean blood lead is predicted from available information about children's exposure to lead. From the distribution, the model estimates the probability (or risk) that a child's or a population of children's blood lead concentrations will exceed a certain level of concern (typically 10 µg/dL) (US EPA 2007a). The probability distribution gives a central estimate of blood lead concentration, which is used to provide the geometric standard deviation (GSD).

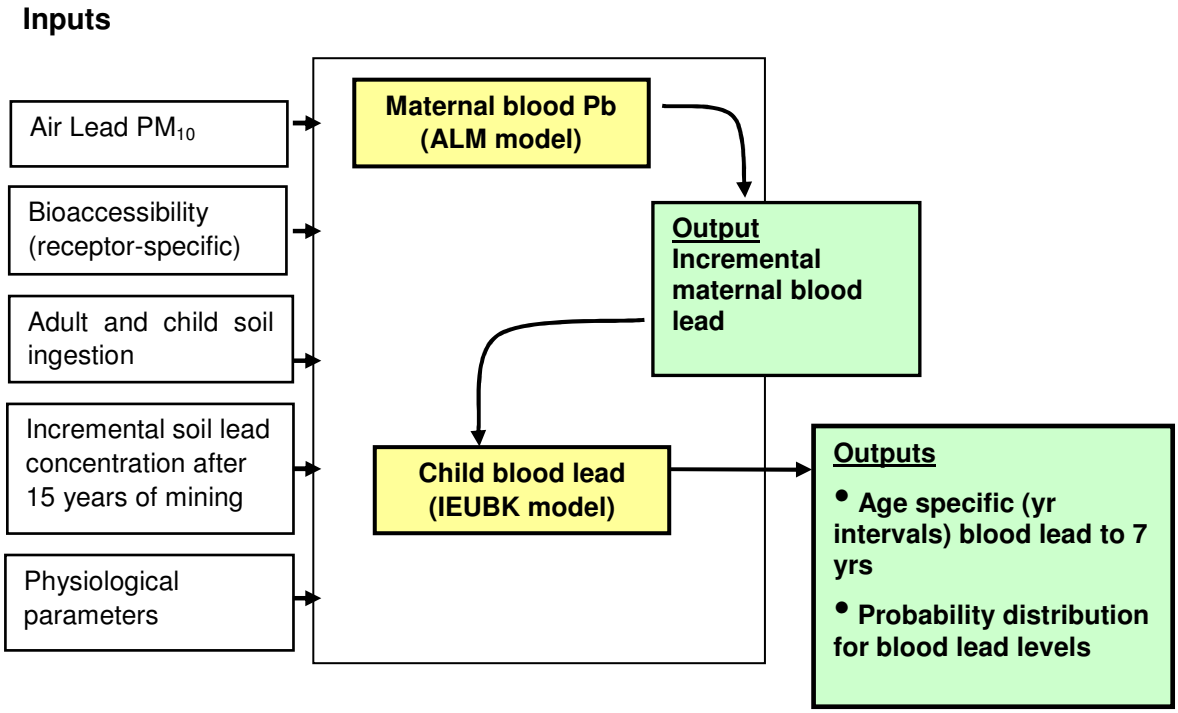
The IEUBK model contains more than 100 input parameters that are initially set to default values, e.g. dietary lead concentrations, consumption rates, inhalation rates, soil ingestion (US EPA 2007a). Almost half of these may be changed by the user. The remaining internal model parameters are set to fixed default values. The defaults represent US averages or plausible central values developed based on years of research. A comprehensive overview of modifiable and non-modifiable parameters in the IEUBK model is given in the HHRA documentation.

During the HHRA a review was undertaken of the default values in the IEUBK model and several parameters modified where site/scenario or Australian specific information was available. Key IEUBK parameters modified for the purpose of the HHRA are documented in the HHRA documentation.

The IEUBK model is designed to evaluate relatively stable exposure situations, rather than rapidly varying exposures. The model cannot be used to predict the effects of short term exposure episodes. The IEUBK model allows changes in exposure to environmental lead concentrations only at one year intervals, and therefore provides outputs only at one year age intervals. The model

therefore assumes that for any year environmental lead concentrations are at steady state during that year. In the HHRA the increase in soil concentrations due to mine site have been calculated at the end of 15 years mine operation, hence the blood lead predictions assume the soil concentrations have been at steady state for up to the seven year age (exposure) that the model is designed to evaluate. This is equivalent to addressing the question – what would the blood lead level be for a child that lived at the location for seven years after the mine closed? This is a maximum exposure scenario since prior to 15 years operation deposited lead at the receptors will be lower than after 15 years.

Figure 9-11: Overview of Methodology for Blood Lead Predictions



Adult Lead Methodology Model

The Adult Lead Methodology (ALM) is a model developed by the US EPA in 1996 and is used to assess adult lead risks from soil at non-residential sites. The methodology focuses on estimating foetal blood lead concentrations in women of child-bearing age exposed to lead contaminated soils. The methodology employs a simplified representation of lead biokinetics to predict quasi-steady state blood lead concentrations among women with relatively steady exposure patterns (US EPA 1996).

In the HHRA the ALM model was used to calculate maternal blood lead levels as an input into the IEUBK model, which was used to estimate the incremental increase in blood lead levels for children aged 0.5-7 years for Scenarios 1a, 1b, and 3. The input parameters for the ALM were modified to reflect site-specific conditions, as documented further in the HHRA documentation.

9.6.3 Predicted Incremental Blood Lead Levels

Incremental blood lead increases were modelled for Scenarios 1a, 1b and 3 for all age groups in the IEUBK model at selected receptors (**Section 9.1.4**).

Predicted incremental increases in blood lead concentration (µg/dL) for each of the above scenarios, assuming no loss of deposited lead from soil over the 15 year period of mine operation, are depicted for the most impacted receptors (R8 and R3) by child age in *Figure 9-12*. Model results indicate that the 1 -2 year old child has the highest incremental blood lead increase. This is consistent with conventional risk assessment wisdom in which this age group is considered to be

the most susceptible to environmental chemicals (enHealth 2004), and also with the information in *Table 9-9* showing lead intake relative to the TDI.

Predicted blood lead levels for a 1-2 year old child at selected receptors for Scenarios 1a, 1b and 3 are illustrated in *Figure 9-13*.

For the most affected receptor (R8) the incremental increase in blood lead after 15 years of mine operation is 0.75 µg/dL, and for R3 (the second most affected receptor) the increase is 0.31 µg/dL.

Figure 9-12: Predicted Incremental Blood Lead Levels by Age Group and Scenario at Receptors 3 and 8, after 15 years of Emissions. (NHMRC 2009 recommends that* all *Australians should have blood lead levels below 10 µg/dL.)

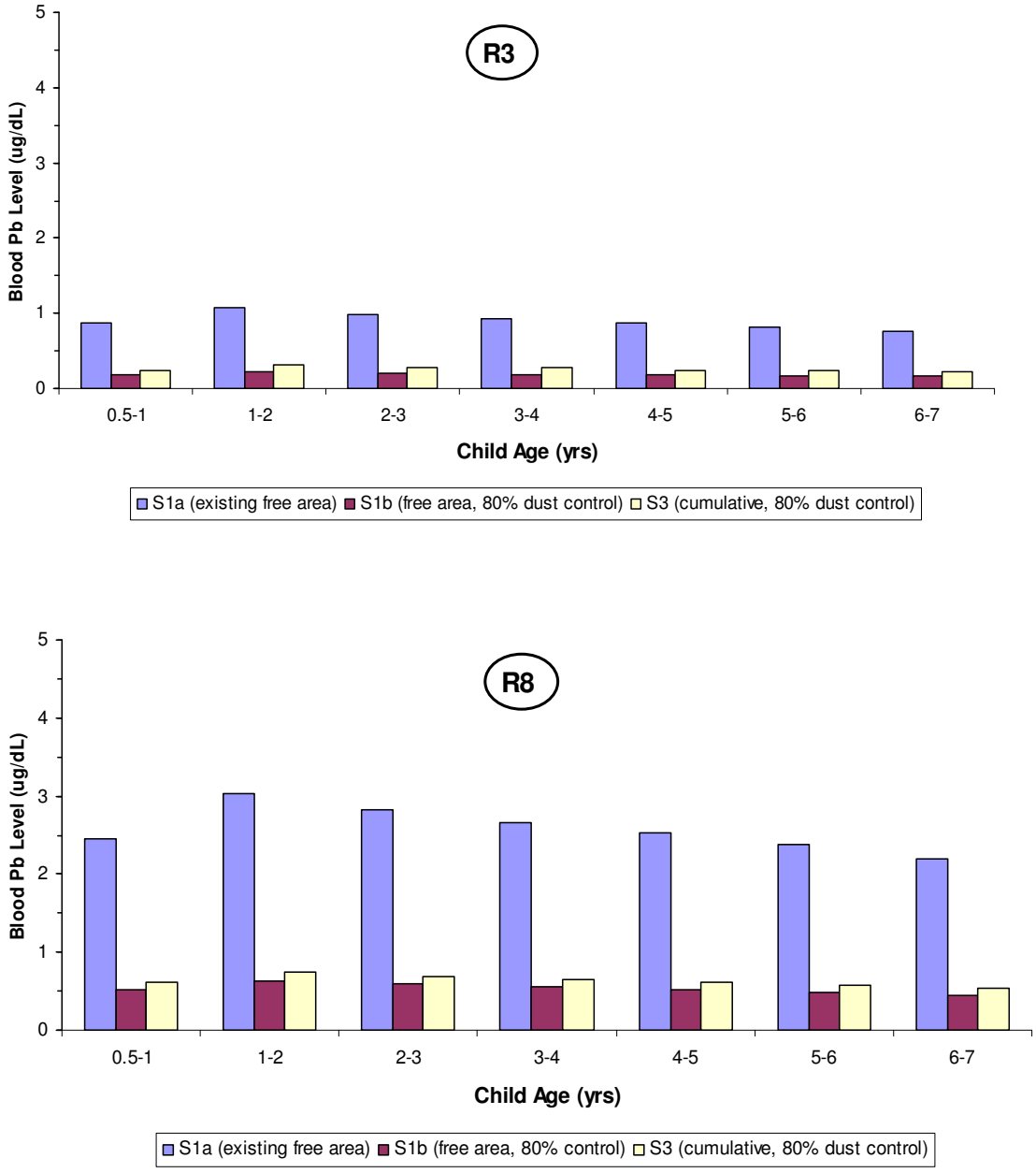
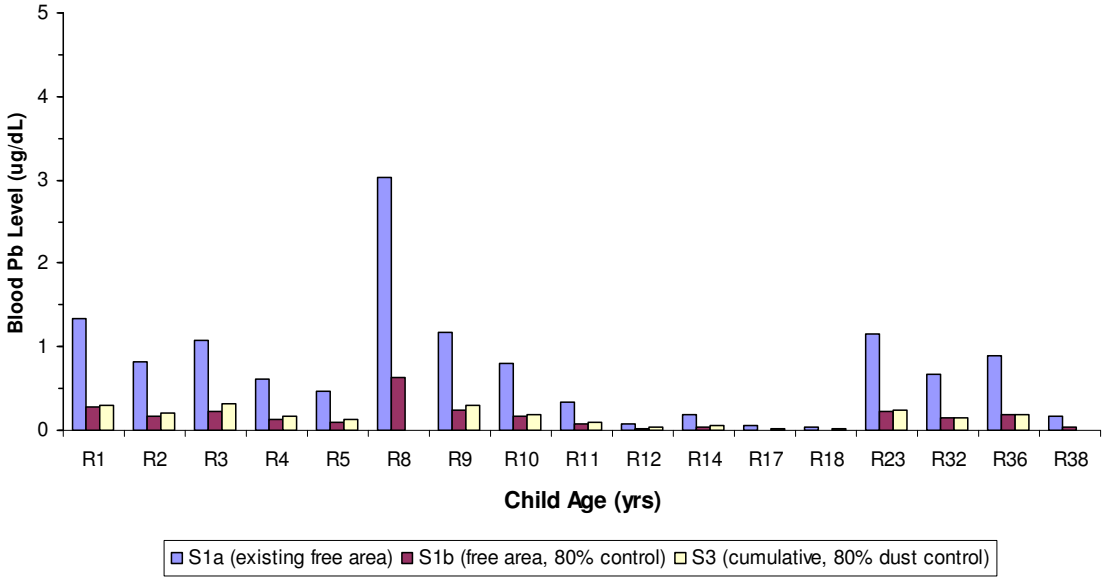


Figure 9-13: Predicted Incremental Blood Lead Levels for a 1-2 year old Child at Selected Receptors, after 15 years of Emissions. (NHMRC 2009 recommends that* all *Australians should have blood lead levels below 10 µg/dL.)



9.6.4 Benefit of Free Area Dust Control on Blood Lead

Model results demonstrate that after 15 years during which the mine lease area is left in its existing condition (i.e. Scenario 1a; if the Rasp mine does not proceed), the predicted incremental increase in blood lead is 2 – 5 times higher than if the mine does proceed (Scenario 3). The difference is due to the additional dust control that the mine operation will bring to the free areas of the lease site (Scenario 1b). The difference is visually depicted for the two most affected receptors in *Figure 9-12* for the different age groups, and in *Figure 9-13* for a 1-2 year old child at the various selected representative receptors.

The concentrations of lead in soil and indoor dust will depend highly on the level of control of the 'free areas.' Controlling dust from the 'free areas' will lessen the annual expected deposition rate of lead to soil at off-site receptors, thereby decreasing the amount of lead being added to background concentrations and hence the incremental increase in blood lead. These benefits on predicted blood lead are expected since dust control of the free areas results in considerable lessening of the deposited dust.

The extent to which control of free area dust will lower predicted blood lead levels relative to levels which may occur if the free areas are not dust controlled, is dependent upon existing exposures to lead. That is, the benefit will depend on existing soil lead concentrations at the receptor locations.

A benefit matrix for amelioration of increases in blood lead concentrations over the life of the proposed mine was constructed during the HHRA. The matrix consists of low, medium and high existing soil concentrations determined from 2004 – 2008 soil lead data and location of receptors in the GWAHS determined risk zones of Broken Hill. These are juxtaposed to low, medium or high lead deposition for receptors in the designated risk zones. The benefit of additional dust control of the free areas was judged as poor, good or very good according to the percentage decrease in predicted blood lead level that would otherwise occur if the mine did not proceed; these terms are respectively linked to decreases in the rise of blood lead levels of 10%, 10 – 20% and >20%.

The greatest benefit of 'free area' dust controls occurs at receptor locations where existing soil lead concentrations are low or medium and lead deposition is medium or high.

9.7 CANCER RISK ASSESSMENT

Lifetime cancer risk for carcinogens which act by directly altering genetic material (i.e. they are genotoxic) is calculated by multiplying the average lifetime chemical exposure by an estimate of the carcinogenic potency of the chemical. The latter is commonly called the unit risk factor or slope factor.

For airborne carcinogens, the unit generally used for unit risk factors is risk per 1 µg/m³ and, depending on the nature of the data used to determine the carcinogenic potency, the numerical value refers to the probability of developing cancer. Thus a lifetime exposure to 1 µg/m³ of a substance may carry a risk of 1 chance in 200 of developing cancer. This probability is expressed as 0.5 in 100 or 0.5 x 10^{-2} per µg/m³.

An official acceptable cancer risk level has not been formally announced by any agency in Australia to the knowledge of Toxikos who undertook the HHRA. In the US a risk of 1 in a million is regarded as being negligible and is the risk level used by the Australian NHMRC for establishing drinking water guidelines for genotoxic carcinogens. The target acceptable risk band adopted in this assessment is 1 x 10^{-6} to 1 x 10^{-5}, i.e. with a lifetime exposure there is a chance of developing a tumour between one in one hundred thousand and one in a million.

Of the fourteen metals identified as being of potential concern in the HHRA, four metals (arsenic, beryllium, cadmium and nickel) are considered genotoxic carcinogens. These four metals were assessed for incremental cancer risk via inhalation exposure. In addition, arsenic was assessed for its potential to induce cancer via ingestion.

Overall the emissions from the mine site do not pose an inhalation or oral cancer risk to persons living around the site.

The highest calculated total cancer risk is approximately 12 x 10^{-7} or one in about eight million. This represents an aggregated risk of the individual inhalation cancer risks for cadmium, beryllium, nickel and arsenic and the incremental oral/dermal risk for arsenic. The predicted risk is within the commonly accepted risk band of between one in a million and one in one hundred thousand.

9.8 UNCERTAINTY ANALYSIS

Uncertainties in the HHRA may influence its accuracy. It is however important to note that the assumptions used to cope with unknown data for specific parameters err on the side of safety and therefore bias the evaluation to an over estimation of health risk. This approach is considered appropriate for an assessment for possible impacts on human health.

Major uncertainties identified in the study were subjected to a quantitative assessment. The sensitivity analysis conducted demonstrated the conservative nature of several of the assumptions used in the study. Further discussion is provided in the HHRA documentation.

9.9 SUMMARY OF MAIN FINDINGS

9.9.1 Risk Characterisation by TDI Analysis

Calculating the intake of a substance from all exposure pathways and comparing the resulting intake to the TDI is a standard risk characterisation procedure commonly performed in human health risk assessments.

Because human uptake of environmental chemicals is dependent in part on age dependent behaviour and physiological factors the calculation of metal intake was estimated for four age

groups; infants/toddlers (0.5 up to 3 years), children (3 up to 13 years), adolescents (13 up to18 years) and adults (18 up to 70 years).

For life stage daily intake of lead the estimations included ingestion, inhalation and dermal exposure pathways to environmental media containing lead. These were soil lead concentrations calculated to be present after 15 years of mine operation assuming no loss of the deposited lead plus assumed background (i.e. existing soil lead concentrations); soil lead concentrations were dependent upon proximity to the mine site both in terms of deposition rate from the proposed mine and existing background soil concentrations. Also included were high end background intake from diet, intake of lead from the Broken Hill articulated water supply, intake by inhaling airborne PM_{10} lead (incremental from dispersion modelling plus background).

Lead intake was greatest for a toddler/child, being about 3 – 4 times greater than an adult. Of the exposure pathways evaluated ingestion contributed 95 – 98% of the total intake; the majority (again 95 – 98%) of this was the result of background intake assumptions for lead. Nevertheless the total daily intake by a child was only approximately 35 – 60% of the TDI for lead, the range being due to the risk zone in which the receptor was located. Compared to the TDI incremental lead intake due to the cumulative exposure from the mine lease area (i.e. exposure to dust from free areas 80% controlled plus mine activities) was negligible for most receptors. Even for the most impacted receptors (R8 & R3) incremental intake was less than 5% of the TDI and much of this was dust from the free areas (80% controlled).

Since at the most affected receptors the total lead intake, including very conservative estimates of background intake from existing soil and diet, was only about 60% of the TDI it is concluded lead exposure resulting from the proposed mine presents little risk to the health of nearby residents.

Despite the fact there is no firm evidence that an additive interaction is expected from the metals evaluated in the HHRA the incremental hazard quotients were summed to give a cumulative exposure hazard index. This was only 0.1 for 0.5 to 3 year olds, with 50% due to lead, and 0.02 for assumed lifetime exposure with 40 – 45% due to lead thus signifying little health risk from combined exposure to metals in dust from the proposed mine site.

In summary, it is concluded:

- *Lead is the metal of most concern regarding potential health effects of dust emissions from the Rasp Mine site.*

- *Since conservative high and exposure assumptions, inclusive of identifiable background exposures, for the most impacted receptor resulted in lead intake by a child that was 60% of the TDI, lead in dust emissions from the proposed Project are unlikely to result in health effects for the surrounding community.*

9.9.2 Predicted Blood Lead Levels

The US integrated exposure uptake and biokinetic model (IEUBK) was used to predict blood levels in children due to exposure to mine emissions and/or assumed background concentrations of lead in soil. This model has been validated and is regularly updated by the US EPA, it is extensively applied in North America to predict blood lead concentrations in children exposed to lead in their environment. It caters for exposure via ingestion of soil and indoor dust, diet, and water, inhalation of airborne outdoor and indoor lead.

The blood lead modelling for various age groups showed the 1 -2 year old child has the highest incremental blood lead increase. This is consistent with conventional risk assessment wisdom in which this age group is considered to be the most susceptible to environmental chemicals and with risk characterisation using the TDI.

For the most affected receptor (R8) the incremental increase in blood lead after 15 years of mine operation is 0.75 µg/dL, and for receptor 3 (the second most affected receptor) the increase is 0.31

µg/dL. These scenarios assume exposure is the result of dust from the free areas (80% controlled) plus dust from mine operation activities, it is also assumed accumulated soil concentrations of deposited lead with no loss incurred over the 15 year period. These increases in blood lead are however 2 – 5 times less than that which is predicted to occur if the lease site is left in its present condition and the proposed mine does not proceed. The difference is due to the additional dust control that the mine operation will bring to the free areas of the lease site.

The extent to which control of free area dust will lower predicted blood lead levels relative to levels which may occur if the free areas are not dust controlled, is dependent upon existing exposures to lead. That is, the benefit will depend on existing soil lead concentrations at the receptor locations. The greatest benefit of 'free area' dust controls occurs at receptor locations where existing soil lead concentrations are low or medium and lead deposition is medium or high.

In summary, with worst case or high end exposure assumptions the predicted increments in child blood lead levels that would occur as a result of mine approval are quite low. Indeed a net benefit on blood lead concentrations is anticipated as a result of the additional dust controls that would occur if the mine proceeds.

9.9.3 Cancer Risk

The total incremental cancer risk associated with genotoxic metals were lower than the cancer risks usually deemed acceptable in Australia (10^{-5} or 10^{-6}). It is therefore concluded that the cancer risks from exposure to the metals of potential concern are very low.