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Health Risk Assessment for Rasp Mine Proposal, Broken Hill

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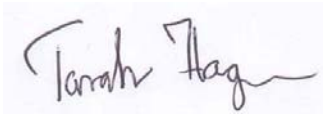

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About Toxikos Pty Ltd

Toxikos Pty Ltd is a consulting company formed on December 1st 2000 to provide clients with independent excellence in toxicology and health based risk assessment. Its charter is to assist industry and government make science based decisions regarding potential effects and management of environmental and occupational chemicals. For over twelve years, prior to and since the establishment of Toxikos, staff have provided toxicology and health risk assessment advice to clients in a wide range of industries and government in Australia, New Zealand and South Africa.

About the author: Dr Roger Drew is one of the Directors and Principal consultants of Toxikos Pty Ltd. He has primary degrees in biochemistry and pharmacology and postgraduate degrees in toxicology. Postdoctoral training was undertaken at the National Institutes of Health, National Cancer Institute in the USA and he spent twelve years teaching medical students and conducting toxicological research at Flinders University of South Australia. He was corporate Toxicologist to ICI/Orica Pty Ltd for twelve years before creating Toxikos Pty Ltd. Dr Drew is the only consultant toxicologist in Australia certified by the American Board of Toxicology.

Dr Drew has been a toxicology consultant to Australian Federal and State Authorities; a member of several standing expert committees of the National Health & Medical Research Council of Australia and the National Occupational Health and Safety Commission of Australia. He has been a member of many expert task groups of the World Health Organization for the International Programme on Chemical Safety.

Dr Drew is Adjunct Associate Professor in the Department of Epidemiology and Preventive Medicine, Monash University and teaches various aspects of toxicology to undergraduate and postgraduate students at local Universities. He is a member of several professional toxicology societies and is a recognised national and international expert in toxicology and risk assessment. He is currently on the editorial board of the international scientific journal "Regulatory Toxicology and Pharmacology".

Executive summary

Broken Hill Operations Pty Ltd (BHOP) is intending to expand the Rasp Mine located on the western side of Consolidated Mining Lease 7 (CML7) in Broken Hill. There is however concern that if the Rasp project proceeds, dust emissions from its operations may undo some the lead related public health gains that have been achieved in recent years. Specifically, there is concern blood lead concentrations in children living in areas historically designated as high risk zones may increase.

Toxikos Pty Ltd has been commissioned by BHOP to undertake a human health risk assessment (HHRA) on dust emissions from the mine. The HHRA has been done according to the requirements of the Director General (NSW department of Planning) and the enHealth Council of Australia (enHealth 2004).

The operational life of the proposed underground mine is 15 years including 1 year for start up and one year for closure. Toxikos was provided with the results of dust air dispersion modelling which predicted dust and lead concentrations at discrete locations around the mine. There are two major sources of dust; firstly that from so called 'free areas', i.e. those areas not impacted by the day to day mine operation, and secondly dust arising from above ground ore processing activities. If the mine proceeds, 'free areas' will have dust control management of at least 80% efficiency.

For a large number of sensitive receptor locations around the lease site Toxikos was provided with airborne concentrations of lead and other metals in total suspended particulates (TSP) and fine particulates (PM_{10}), and also annual deposition rates of lead and metals at the receptors. For each of these data for lead the relative proportion of 'free area' dust and 'mine process activity' dust was provided. The bioaccessibility of lead in these dust sources was determined which allowed receptor specific bioaccessibility in airborne dust and soil to be estimated according to the source apportionment of the lead. Bioaccessibility is that part of the lead in soil or dust that will become solubilised in gastrointestinal juices and then be subject to absorption; in children about 50% of this is absorbed but only about 10 – 15% in adults.

A number of worst case or high end exposure assumptions were incorporated into estimations of lead exposure at representative receptors. Not the least of which was calculation of lead accumulation in receptor soil resulting from deposition of TSP and assuming no loss of the deposited lead over the 15 year operation period of the proposed mine.

This assessment follows the broad guidelines for assessing human health risks from environmental hazards as articulated by the enHealth Council of Australia. The risk characterisation has been undertaken in two ways:

- Comparison of conservative intake of lead and other metals with their respective tolerable daily intakes (TDI) established by either Australian health authorities or the World Health Organisation (WHO).
- Prediction of blood lead levels in children.

Risk characterisation using the tolerable daily intake (TDI):
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 influenced in part on age related 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 intake estimations included ingestion (which also incorporates hand mouth activity), inhalation and dermal exposure pathways to environmental media containing the lead. These were soil at 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 reticulated water supply, and 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 of this (again 95 – 98%) was the result of background intake assumptions. 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 (i.e. background soil lead concentrations) 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 50% 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 HRA, the incremental hazard quotients were summed to give a cumulative exposure hazard index. This was only 0.1, for 0.5 – 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.

In summary, it is concluded that since conservative high end 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 mine are unlikely to result in health effects for the surrounding community.

Blood lead levels:

The National Health and Medical Research Council (NHMRC) of Australia has 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.

The US Integrated Exposure Uptake and Biokinetic model (IEUBK) was used to predict blood lead levels in children due to accumulation of lead in soil over 15 years of mine operation and 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, and inhalation of airborne outdoor and indoor lead.

The blood lead modelling for various age groups showed the 1 -2 year old child as potentially having 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, it is also consistent with the risk characterisation using the TDI.

For the most affected receptor (R8) the incremental increase in blood lead after 15 years of mine operation is predicted to be 0.75 µg/dL (this receptor is located within the mine site lease boundary), and for receptor 3 (the second most affected receptor) the increase is 0.31 µg/dL. These predictions 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 at the receptors incur no loss 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 blood 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 has been constructed. The matrix consists of low, medium and high existing soil concentrations determined from 2004 – 2008 soil lead data and location of receptors in historically established risk zones of Broken Hill. These assumed existing soil lead concentrations 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 the lead deposition in those areas 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, compared with blood lead concentrations that may occur if the mine lease site is left in its present condition, a net benefit on blood lead concentrations is

anticipated. This is due to the additional dust controls that would occur if the mine proceeds.

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

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1. Introduction

Toxikos Pty Ltd has been commissioned by Broken Hill Operations Pty Ltd (BHOP) to undertake a human health risk assessment (HHRA) for the expansion of the Rasp Mine located on the western side of Consolidated Mining Lease 7 (CML7) in Broken Hill, NSW. BHOP intends to mine the Western and Centenary Mineralisation within the mine lease area (Figure 1.1) over 15 years using underground mining methods. This timing includes one year for plant construction and one year for mine closure.

A detailed description of the proposed mine can be found in the ENVIRON (2010a) report. Ore processing will involve an above ground stock pile of ore, and crushing and flotation plants to produce lead and zinc concentrates which will be transferred by rail for shipment via Port Pirie or Newcastle. Only a relatively small portion of the mine lease site is impacted by the mining operations, the remaining 'free areas' are not traversed by personnel or vehicles other than in the normal course of land management. It is intended that if the mine proceeds the free area will be managed for dust lift off with 80% efficiency. In addition it is noted the mine proposal commits to real time meteorological and dust monitoring to manage and minimise off-site dust impacts.

ENVIRON Pty Ltd has conducted a detailed analysis of dust lift off from the mine lease site and that associated with the mine processes (ENVIRON 2010a) and air dispersion modelling techniques were used to predict the fate of the dust over nearby areas. Apart from generating dust concentration contours around the mine lease, concentrations of airborne dust and metals in the dust were predicted at discrete receptor locations (Figure 1.2). Most of these 'receptors' are close to the mine site since they are the most likely to be impacted by mine activity.

For this HHRA, ENVIRON Pty Ltd supplied Toxikos with contour plots and specific receptor information for the following parameters (Appendix A2):

- Concentrations of air borne dust (TSP and PM_{10}).
- Concentrations of metals (Pb, As, Ag, Ba, Be, Cd, Cr, Cu, Fe, Mn, Hg, Ni, Sb, Zn) in TSP and PM_{10}.
- Annual deposition rate of metals from TSP.

Figure 1.1: Location of key operational components of the proposed Rasp mine.
Reproduced from the Environ Air Quality report (Environ 2010a).

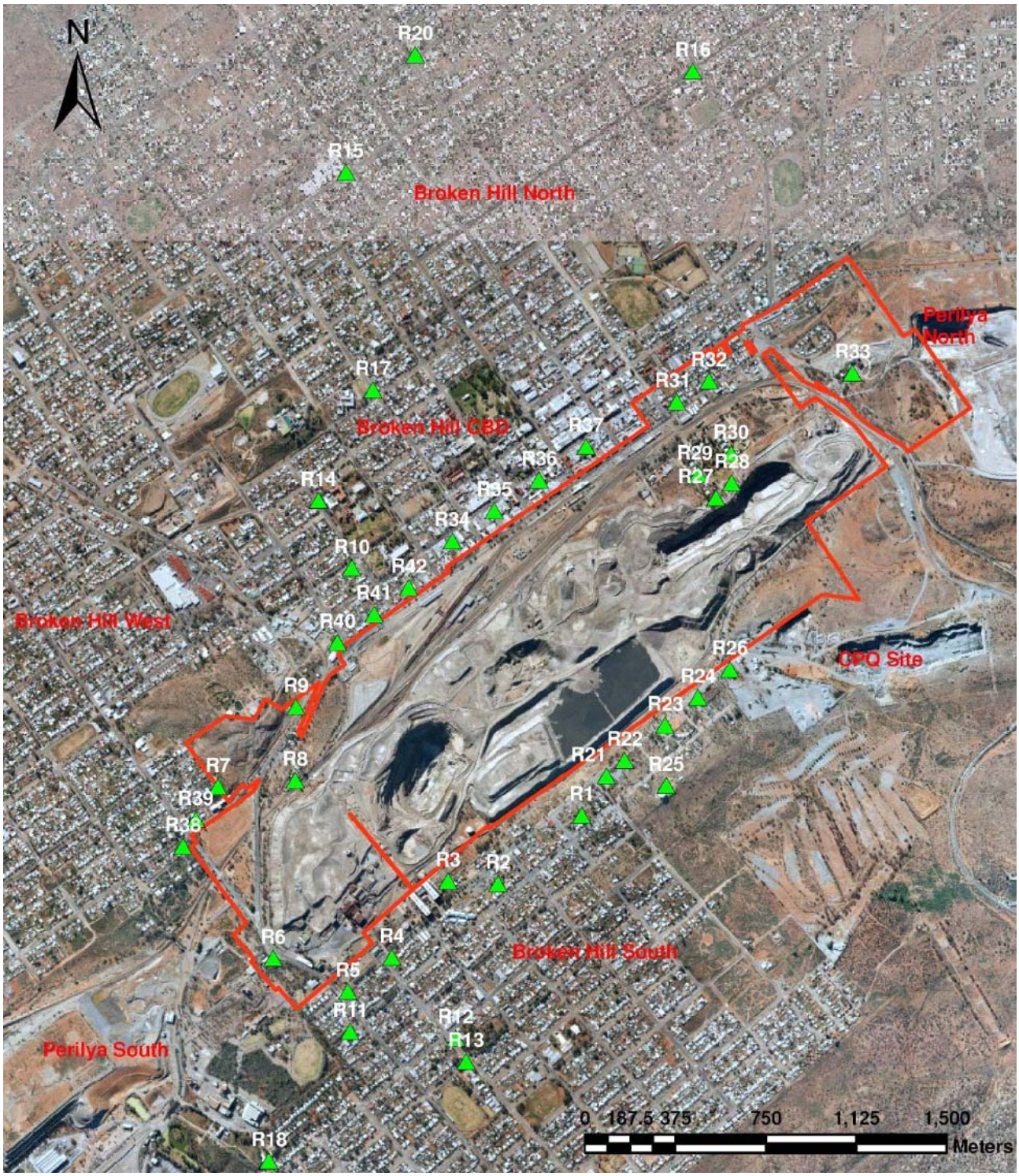


Figure 1.2: Wider area map of Broken Hill with receptor locations addressed by dispersion modelling of mine site derived dust.

1.1 Scope – issue identification

Broken Hill has a long history of lead and zinc mining; quite apart from lead being in the human environment due to the local mineralisation, past mining and smelting practices have resulted in widespread surface soil contamination of areas around the mines. Smelting is no longer done at Broken Hill. As a consequence the population had in the past 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). Starting in 1994 the City of Broken Hill undertook targeted remediation of public spaces and selected residences (Lyle et al. 2001, 2006). This, and other initiatives such as an education campaign for modification of personal behaviour to minimise L\lead exposure and advice that tank water should not be used for drinking or cooking, has 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; Section 5.1).

The major health concerns arising from the proposed mine are associated with lead, this is the specific focus of the HHRA. The HHRA addresses the requirements of the Director General (NSW department of Planning); all potential pathways to lead exposure from the proposed mining operations have been addressed. The HHRA also takes into account long-term exposure to lead and other metal emissions from the proposed mining operations. The HHRA conforms to the assessment principles articulated in the Australian *'Guidelines for assessing human health risks from environmental hazards'* (enHealth Council 2004)[1].

1.2 Scenarios considered

All the scenarios considered in this risk assessment have considered health risks after 15 years of mine operation. This was achieved by estimating the accumulation of metals in residential soils from annual average deposition rates predicted by dispersion modelling of various dust sources at the mine site (ENVIRON 2010).

The heath risk assessment has been conducted on three exposure scenarios which are related to the mine lease dust source. These are designated 'S1', 'S2' and 'S3'; scenario 1 consists of two sub-scenarios, denoted by the letters 'a' or 'b'. The scenarios are as follows

[1] These guidelines were originally released in 2002, they were reissued in 2004.

- **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** : Free areas with dust control to 80% efficiency. Referred to as 'free area(80% control).

- **Scenario 2 (S2)**: Assesses off-site health risks due to dust arising from mine operations (paved and unpaved active roads, ore handling, ore stockpile emissions, ventilation shaft emissions etc). These sources are denoted as 'mine activity' or 'mine process' related in this report.

- **Scenario 3 (S3)**: Off-site health risks from the cumulative impact of metals in dust from the free areas (80% controlled, i.e. S1b) plus those in dust from 'mine activity' (S2).

The 'free areas' emission source represents dust emissions from the open areas within the 'surface rights' lease of the mine site that are not affected by activities associated with the mine proposal.

1.3 Risk assessment method overview

The air quality impact report generated by ENVIRON Pty Ltd (ENVIRON 2010a) assessed the predicted airborne dust concentrations, dust deposition rates and airborne concentrations of metals in dust against specific criteria used to make judgement for likely impact on the surrounding community. Consequently dust *per se* and exposure to airborne metals are not included in this HHRA. The HHRA evaluates the potential health impacts of lead via two risk characterisation methods:

- Comparison of calculated metal intakes with the tolerable daily intake (TDI) for each metal (Chapter 4). For lead intake estimations were derived from estimations of accumulation of lead in receptor soil, available information on background intakes and predicted lead concentration in PM_{10}. Where scenario specific information was not available, default assumptions commonly used in screening risk assessments were used (enHealth 2004). This TDI approach was employed in a recent risk assessment for lead at the Port of Esperance (Golder Associates 2009). Because background intakes for the other metals are not readily available a screening risk assessment was undertaken using

the incremental intakes that may be associated with the proposed mine and comparing these to the relevant TDI for the metal.

Because human uptake of environmental chemicals is influenced 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). In tables and figures in this report and appendices these age ranges are described as 0.5 – 3 yrs, 3 – 13 yrs, 13 – 18 yrs and 18 – 70 yrs.

- Modelling blood lead concentrations in children at selected worst case receptors and receptors representative of historically designated risk zones around the mine (Chapter 5, see Section 1.4 for receptors). The US EPA Integrated Exposure Uptake and BioKinetic (IEUBK) model was used for predicting 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.

The TDI risk characterisation method was used for other metals.

Details of each risk characterisation method are provided in the relevant report chapter.

1.4 Receptor selection for summary tables

For ease of report reading and presentation, selected receptors are presented in summary tables in the text (Table 1.1). These receptors were selected after consideration of:

- proximity to mine activity,
- the historical lead health risk zones in Broken Hill town (Boreland et al. 2002, 2009a; Lyle et al. 2006) (Section 2.2) used by the Greater Western Health Services to organise and interpret lead biomonitoring data (Boreland 2010, Lesjak 2010), and
- predicted dust deposition (low, medium, high) from the mine lease site.

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

Table 1.1: Receptors selected for ease of reporting in this HRA

Selected Receptor
Residences
1. Piper Street North
2. Piper Street Central
3. Eyre Street North
4. Eyre Street Central
5. Eyre Street South
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)
Other Locations
11. Alma Bugdlie Pre-School
12. Playtime Pre-School
14. Broken Hill High School
17. Broken Hill Public School
18. Rainbow Pre School

2. Exposure estimations

2.1 Exposure Pathways

Exposure pathways considered for lead exposure estimates are summarised in Figure 2.1.1 and Figure 2.1.2. In Figure 2.1.2 pathways considered to be major for the scenarios being investigated are denoted with a bolded arrow. These pathways were also included in exposure estimations for the other metals.

In essence, 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 total suspended particulates (TSP) that are transported by wind. The sources contributing to the TSP are conveniently divided into those related to mine process (paved and unpaved active roads, ore handling, the ore stockpile, crushing, ventilation shafts etc) and those that are non-process related (areas of the mine lease surface not involved with the day to day operation of the mine, the so called ‘free areas’).

At the receptor location exposure to the mine derived metal 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, inhalation of indoor and/or outdoor airborne dust.

Equations specific for each risk characterization method are provided in their respective chapters.

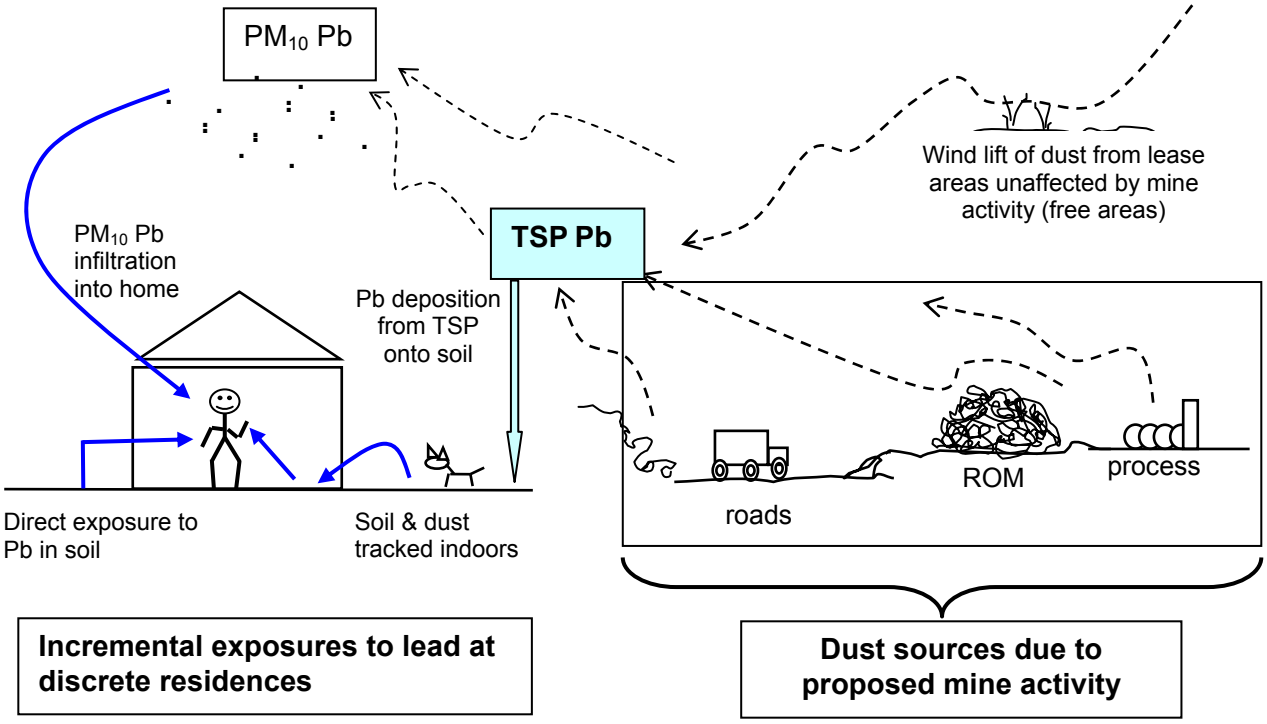


Figure 2.1.1: Conceptual representation of dust/lead sources and residential exposure pathways.
The assessment for incremental health risk has been performed on three exposure scenarios.
S1: Dust from mine lease sources not affected by mine operations (i.e. free areas) either not-controlled for dust lift off (S1a) or 80% of the area with dust control (S1b).
S2: Dust sources attributable only to mine activity (i.e. roads + run of mine (ROM) + process + ventilation shafts etc).
S3: The combination of S1b & S2.

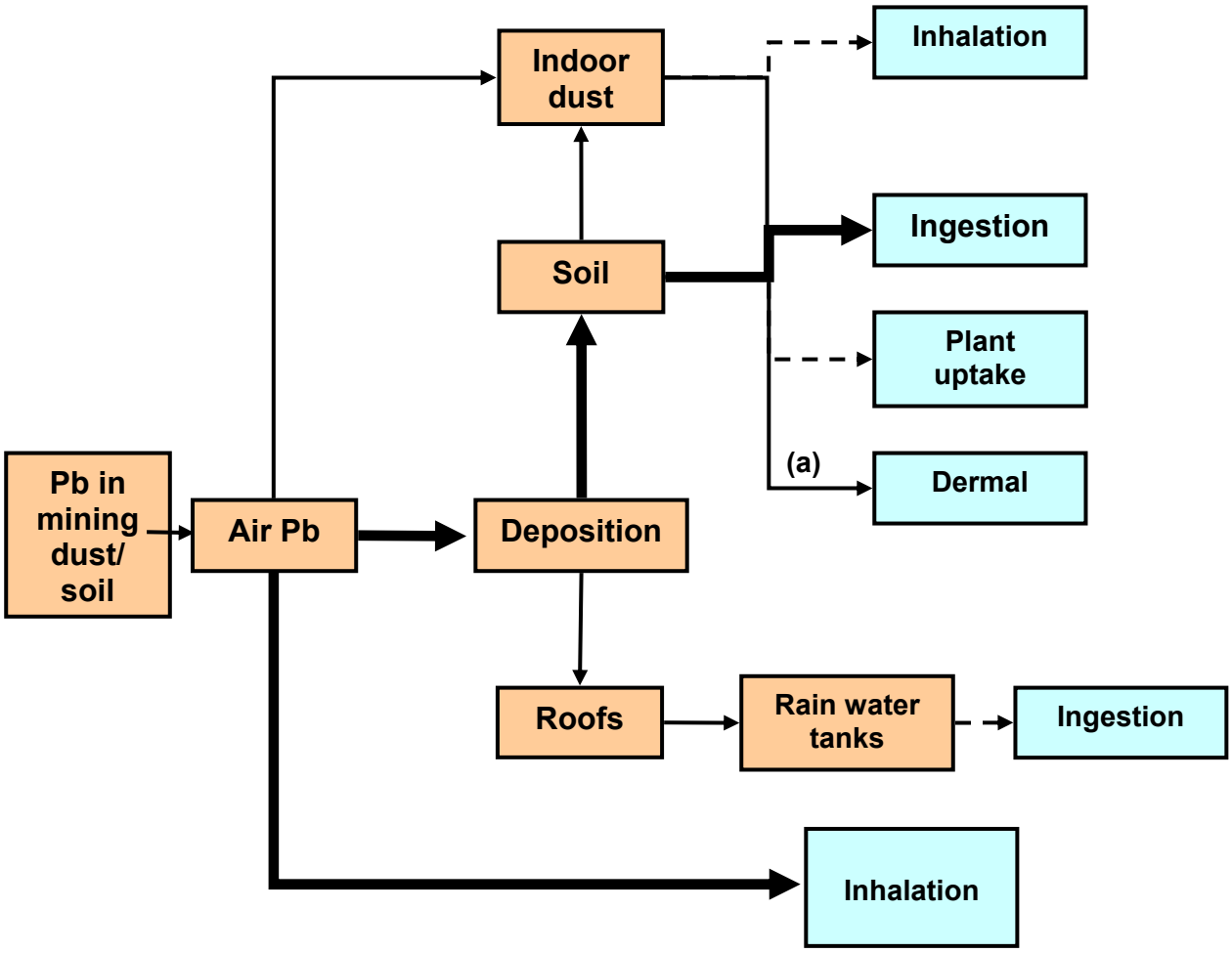


Figure 2.1.2: Conceptual Site Model: Pathways considered in exposure estimates.

Legend

- - - ▸ Incomplete or insignificant according to contemporary knowledge

➡ Major pathway

(a) Not included in IEUBK modeling

Ingestion of tank water is not considered a complete exposure pathway because reticulated water is supplied and the local public health authority and Shire Council 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 because:

- A literature search indicated lead is not readily translocated to shoots of plants (which is the edible part of most vegetables) compared to the lead taken up by the roots (Hong et al. 2008, Kumar et al. 2009, Peralta-Videa et al. 2009, Ciesli'nski and Mercik 1993, Mbila and Thompson 2004). Nevertheless leafy vegetables such as lettuce and cabbage may accumulate lead in their edible portions (Lagerwerff 1971, Nasralla and Ali 1985). Lead accumulation varies with different plants and different soil conditions; data indicate accumulation becomes significant at levels around 500 µg/g [2] (Judel and Stelte 1977, Sterrett et al. 1996).

  For root vegetables, Lagerwerff (1971) showed changes in soil lead only caused small changes in radish. Similarly, Davies (1978) concluded that although they do absorb small amounts of lead the amount taken up by radishes and potatoes is low. In addition a study by Judel and Stelte (1977) showed accumulation of lead from lead acetate (a soluble form of lead) in plants whose roots are generally used for food (i.e. radish, carrots) was greatest in portions not normally eaten.

  The *Handbook on the Toxicology of Metals* (Nordberg et al. 2007) considers the uptake pathway of lead by roots of vegetables to be relatively unimportant. Lead is said to be present in vegetables mainly as the result of deposition from air, rather than uptake from soil.

- Home grown produce only makes up a small percentage of total vegetable intake by the general population. In the ABS survey of 1994 (ABS 1994), 4.3% of total fruit and 5.6%

[2] Judel and Stelte (1977) showed at 20 µg Pb acetate/g soil, there was a slight increase in Pb content of radish, carrots and spinach, but this increased at 100 µg/g soil, reaching high significance at 500 µg/g.

Sterrett et al. (1996) grew lettuce in one control agricultural soil and five urban garden soils whose Pb concentration ranged from 12 - 5210 µg/g. Lettuce Pb concentrations increased from 2.2 µg/g in control soil to 37 µg/g in metal-rich urban garden soil. On normally fertilized soils Pb uptake by lettuce was not high until soil Pb substantially exceeded 500 µg/g.

of total vegetables produced in Australia were home-grown. Broken Hill is a relatively dry region. Mean annual rainfall (Patton Street and Broken Hill airport) for 1981-2010 was 241-253 mm/year (BoM 2010). Thus the likelihood of vegetables and fruits being grown at home is lower than other parts of Australia, where rainfall is higher[3].

- Plants take up metals in soluble form, bioaccessibility tests mimicking gastrointestinal conditions on samples of surface soil from open areas and on crushed ore showed the bioaccessibility of lead to be low (1.4-7.3%) (EnTox 2009). Although water solubility at soil pH was not undertaken it is expected to be lowered than the solubility in synthetic gastrointestinal juice.

2.2 Background environmental lead concentrations and 'risk' zones

Recent data on soil lead concentrations in residential areas of Broken Hill were not located in the published literature. NSW Department of Health advise [4] monitoring lead contamination in soil at Broken Hill was conducted by the Broken Hill Council in early 1990's and since then there has been no new systematic soil monitoring or data made available.

Soil lead levels from the Broken Hill City Council 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. Based on soil lead levels and proximity to the mine Broken Hill was divided into ten districts (Figure 2.2.1, Table 2.2.1). Districts were ranked and divided into two groups about the median value. Districts with soil lead above the median value were regarded as having high soil lead (mean 1,220 µg/g, range 708 – 2,305 µg/g) and those below the median were designated as having relatively lower soil lead (mean 361 µg/g, range 245 – 521 µg/g). Boreland et al. (2002) found the higher the average soil lead in a district the higher the mean flux of lead indoors. Houses in districts with high soil lead had indoor lead flux levels three times that of houses in districts with lower soil lead. As would be expected the flux of lead indoors was influenced by the condition of the house with lowest levels observed in well sealed dwellings.

[3] For comparison, Sydney (Observatory Hill) had an average rainfall of around 1170 mm/year between 1990 and 2009.

[4] Personal communication with NSW Department of Health by email 10/12/09.

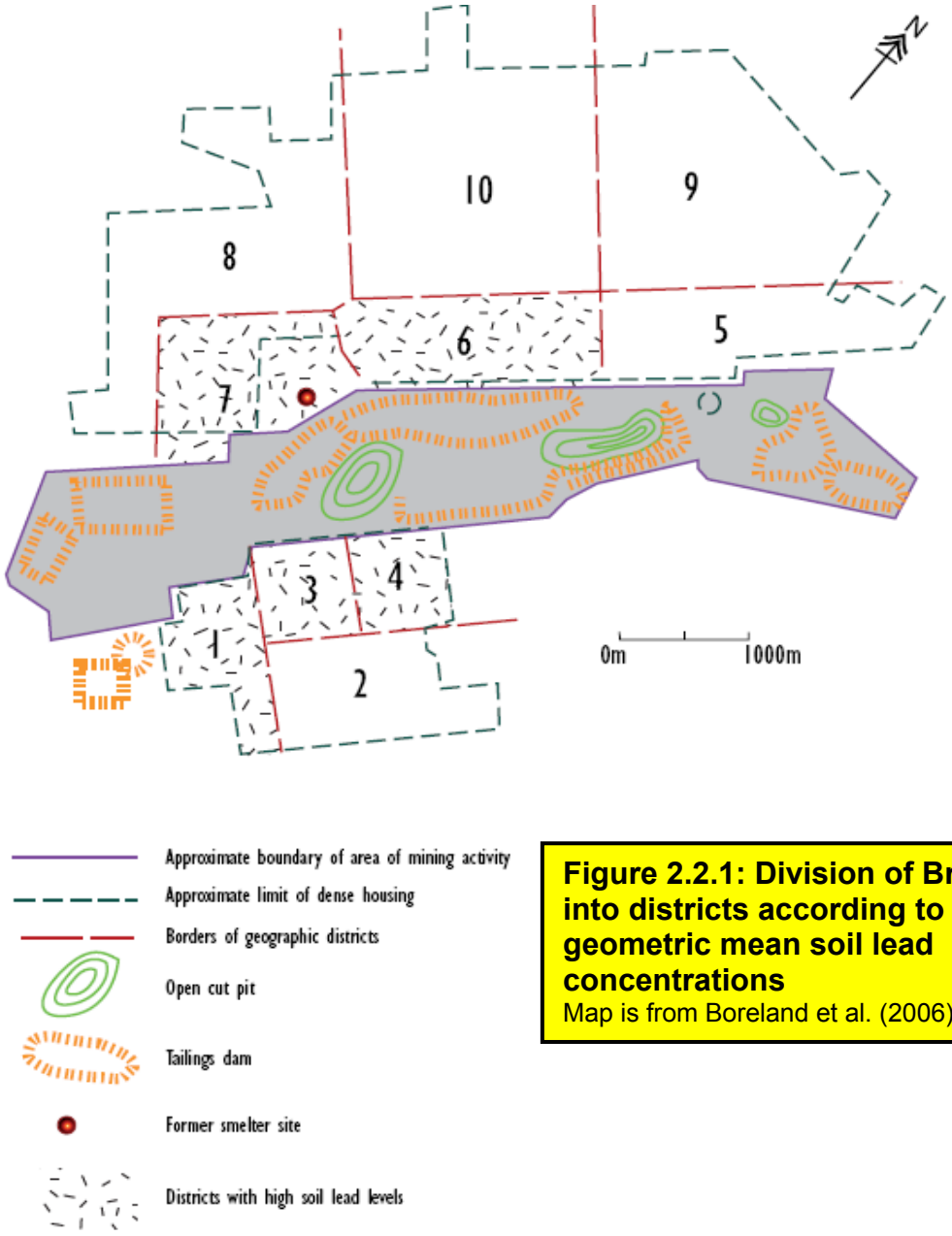


Figure 2.2.1: Division of Broken Hill into districts according to *historical* geometric mean soil lead concentrations
Map is from Boreland et al. (2006)

Table 2.2.1: Background soil lead concentrations by district and approximate corresponding modelled receptor.

District code (Risk Zone)	Soil Pb concentration (µg Pb/g soil) [a]				Receptor(s) in this district
	Boreland et al. 2002 (1992 data)		Boreland (2010) (2004-2008 data)		
	Geometric mean	95% CL range	Geometric Mean	95% CL range	
1 (RZ2)	870	684–1098	480	214-1069	5, 11
2 (RZ4)	510	435–597	340	172-676	12, 13
3 (RZ1)	1,450	1136–1861	1310	941-1831	4
4 (RZ1)	2,310	1642 – 3235	1390	1007-1908	2, 3
5 (RZ3)	520	435 – 624	530	287-971	-
6 (RZ3)	770	581 – 1011	640	324-1269	41, 42, 34, 35, 36, 37, 10, 14
7 (RZ2 & 4)	710	508 – 986	600	340-1048	38, 39, 7, 9
8 (RZ4)	260	229 – 298	180	114-292	-
9 (RZ5)	250	204 – 295	240	178-332	16, 19
10 (RZ5)	270	238 - 309	200	108-369	17, 15, 20

[a] The GM soil lead concentrations have been rounded to the nearest ten.

The 'district' concept for organising environmental lead data was retained by Boreland and Lyle (2006) and Boreland et al. (2006) in the evaluation of residential remediation success on lead loadings of houses in Broken Hill and the relationship of children blood lead with indoor lead fluxes. 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). Lead loading levels recorded prior to remediation were highest on internal windowsills (mean loading, 4460 mg/m^2) and lowest on internal floors (mean loading, 129 mg/m^2). Mean lead loading on all surfaces was significantly reduced by remediation, but with respect to internal floors, only homes with an internal floor loading above 276 mg/m^2 benefited significantly. 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. When two homes with very high soil lead were excluded from the analysis, the effect of high soil lead was insignificant suggesting it was not a major determinant of indoor lead loadings for the majority of homes. Both the soil lead and the indoor flux were likely the result of high ambient air concentrations of lead in dust that were influenced by proximity and wind direction from the former open-cut mines. At the time of the Boreland et al. (2002) study the majority of Broken Hill homes were 50–100 years old and built of wood and clad with corrugated iron or stone.

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.

More recently Boreland et al. (2009a) have amalgamated the 'districts' into risk zones which correlate with potential for greater hazard exposure. This resulted in five risk zones depicted in Figure 2.2.3. The data from the Broken Hill City study, as reported by Boreland et al. (2002), is reproduced in Table 2.2.1 and the approximate positions of the receptors assessed in this report relative to the risk districts are depicted in Figure 2.2.2. It is apparent that many of the discrete receptor locations subject to dispersion modelling, at least historically, have been associated with high soil lead concentrations.

From 2004-2008, measurements of soil lead levels 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[5] to Toxikos as geometric means, 95% confidence intervals, minimum and maximum soil lead levels by district. This information, together with the 1992 survey data are presented in Table 2.2.1.

It is noted that due to the targeted nature of the 2004 – 2008 data that 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 (Table 2.2.1). There has been a decrease in the geometric means of the risk zones (Figure 2.2.3) 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.

[5] Personal communication with the Greater Western Health Services (Boreland 2010).

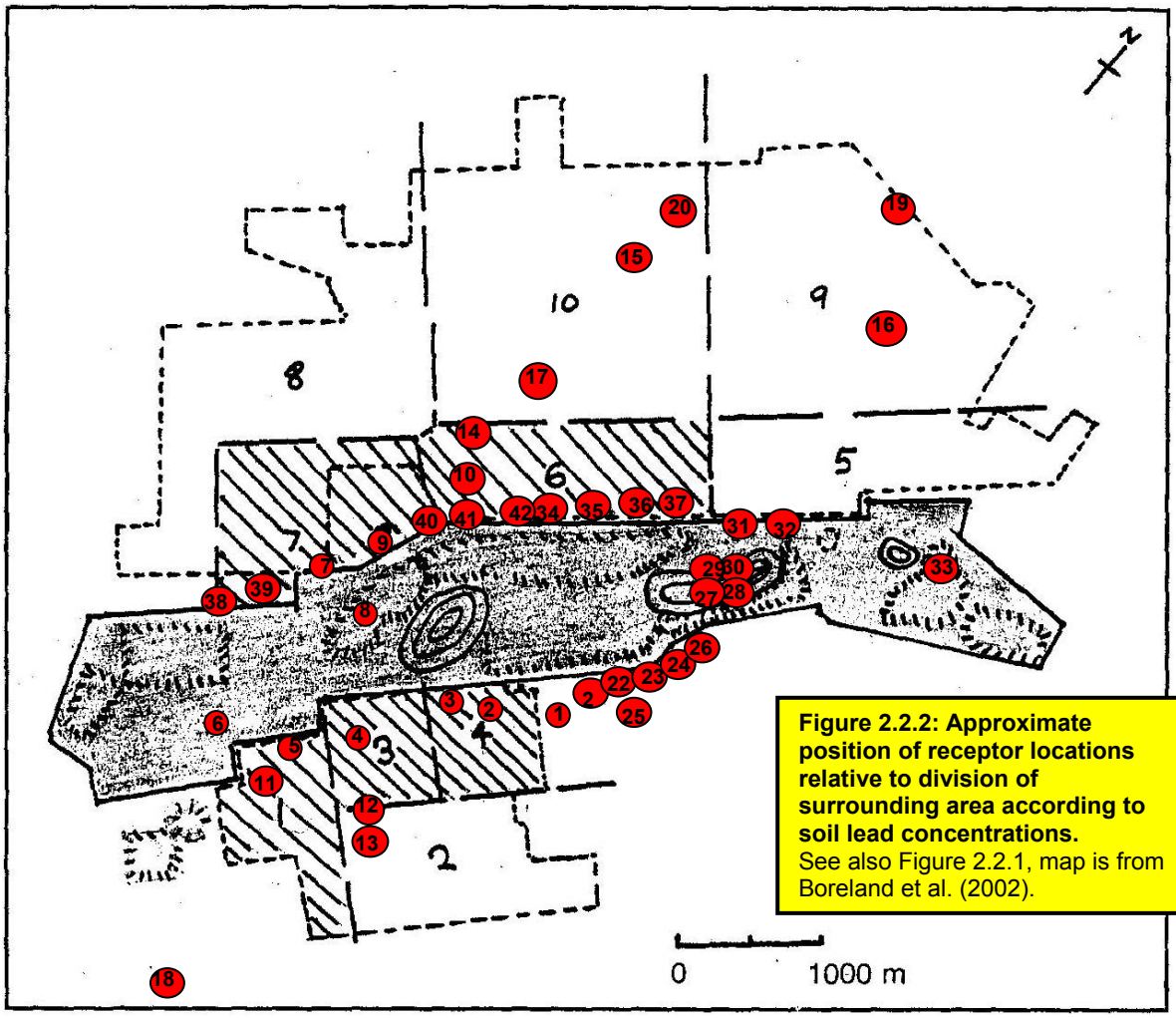
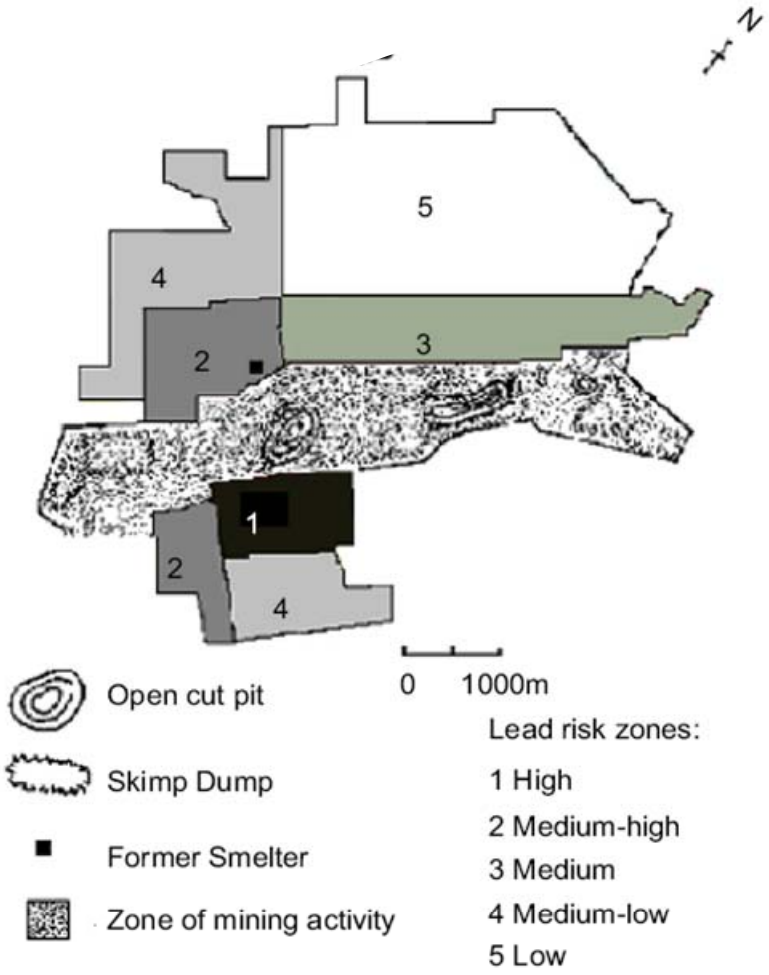


Figure 2.2.2: Approximate position of receptor locations relative to division of surrounding area according to soil lead concentrations.
See also Figure 2.2.1, map is from Boreland et al. (2002).

Figure 2.2.3: Lead risk zones in Broken Hill
Figure is from Boreland et al. (2009a) and data in table from Lyle et al. (2006) and Boreland (2010).



Risk zone (District) [c]	Lyle et al. (2006), (1992 data [a])		Boreland (2010), (2004-2008 data)
	Geometric mean soil lead (µg/g)	Geometric mean indoor dust (µg/m^2/30 days)	Geometric mean soil Pb (µg/g) [b]
1 (3+4)	1967	946	1350
2 (1+7)	794	717	540
3 (5+6)	621	490	580
4 (2, 7, 8)	365	216	260
5 (9+10)	262	201	220

[a] Soil lead concentration data is from the Broken Hill City Council 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). See also Table 2.2.1.

[b] Soil Pb levels for the 2004-2008 dataset (n=148 houses) were provided as geometric means and 95% confidence intervals for Broken Hill districts (Boreland 2010). For comparison with the Lyle et al. (2006) soil Pb 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. This 'average GM' was rounded to the nearest ten.

[c] See Table 2.2.1 for soil Pb concentrations in Broken Hill districts.

In a limited sampling regime, Gulson et al. (1994b) found the fine fraction of soil (-53+38 µm) generally had more lead than the bulk sample by a factor of 1 -2 times. In this study

- 35 analyses of 14 'old' soil[6] samples gave a range of lead concentrations of 110 – 4,490 ppm (µg/g),
- the measured range of 'new' soils (n = 4) brought in to replace old contaminated soil, was 10 – 470 ppm,
- the measured lead concentrations of sweepings of road side gutters were 80 – 22,000 ppm for bulk samples (n = 8), and
- lead levels in pavement gravels was 560 – 20,500 ppm for bulk samples (n=5).

Interestingly Gulson et al. (1994b) found that although lead in soil and house dust was dominated by ore body sources, the isotope fingerprinting of blood lead in children indicted a significant proportion of the latter lead was from different sources such as leaded petrol and paint.

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. The information in Boreland and Lyle (2006) suggests the soil data gathered by the Broken Hill City Council in 1992 and reported in Boreland et al. (2002) may represent soil lead levels prior to the remediation program.

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).

Thus 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

[6] Soils referred to as 'old' in Gulson et al. (1994b) corresponded to un-remediated soils in Broken Hill. 'New' soils included the soils that were brought in from west of Broken Hill in order to replace 'old' contaminated soils containing ~ 0.5 – 1% Pb, sandpit material, and cracker dust used as pavement material.

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, Included was remediation of public land (Lyle et al. 2006).

In conclusion, 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 this risk assessment, background soil lead concentrations have been assumed from data reported for soil sampling undertaken in 2004-2008, which is the most recent soil data available (Table in Figure 2.2.3). 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: risk zone 1, risk zones 2 and 3 have merged into one (denoted risk zone 2+3 in this assessment), and zones 4 and 5 have merged into one (denoted risk zone 4+5 in this assessment).

Table 2.2.2 summarises all the pertinent data from the 2004-2008 soil sampling (from Boreland 2010), together with the values selected as plausible representative background soil lead levels in this risk assessment.

Background concentrations of soil lead used in the TDI assessment (Chapter 4) required a specific number for intake calculations. Therefore, for conservatism, these values were roughly based on the upper 95% confidence limits at the respective districts:

- Risk zone 1: Background concentration of soil Pb assumed to be 2,000 µg/g.
- Risk zones 2+3: Background concentration of soil Pb assumed to be 1,000 µg/g.
- Risk zones 4+5: Background concentration of soil Pb assumed to be 500 µg/g.

Table 2.2.2: Most recent existing soil lead data (µg Pb/g soil) by district (D) and risk zone, and selected background soil Pb levels in this HHRA

Reference	Date of sampling	Areas sampled	Statistic	Risk Zone				
				1	2	3	4	5
Boreland 2010	2004-2008	Houses	Average	**1,833**	**893**	**983**	**417**	**433**
			Range	240-2,981	158-2,252	126-3,090	59-1,726	5-2,116
			GM	D3: **1,313** D4: **1,386**	D1: **478** D7: **597**	D5: **528** D6: **641**	D2: **341** D8: **183**	D9: **243** D10: **199**
			95% CI	D3: 941-1,831 D4: 1,007-1,908	D1: 214-1,069 D7: 340-1,048	D5: 287-971 D6: 324-1,269	D2: 172-676 D8: 114-292	D9: 178-332 D10: 108-369
			avg of GM [a]	**1350**	**540**	**580**	**260**	**220**
			n	35	18	21	22	52
Selected values used in risk assessment [b]								
		TDI analysis (approximate upper 95% CI) [b]		2,000	1,000		500	

[a] In the newest analysis of the data (personal communication with Frances Boreland 2010), soil Pb levels for the 2004-2008 dataset were given by district, as geometric means and 95% confidence intervals. If more than one district was located in a risk zone (as per Boreland et al. 2002), GM's for those districts were averaged to give an 'average GM' for that risk zone. The average GM was rounded to the nearest ten.

[b] The background soil Pb levels selected for intake equations in the TDI analysis (Chapter 4) were selected to coincide with the approximate upper 95% confidence limits for the respective risk zones.

2.3 Incremental receptor soil concentrations from mine site

Toxikos was provided with total annual deposition of lead and 13 other metals at each of the receptors depicted in Figure 1.2. Table 2.3.1 shows lead deposition at each modelled receptor for the cumulative scenario S3, data for other metals is in Appendix A2.

Also provided was the contribution from 'free area' and 'ore' sources at the mine site. The 'free areas' emission source represents dust emissions from the open areas within the 'surface rights' lease of the mine site that are not affected by activities associated with the mine proposal; the data in Table 2.3.1 assumes this area has 80% dust control. As discussed in Section 1.2, source apportionment due to 'ore' represents dust emissions from haul roads, run of mine (ROM) stockpile, crushing operations, and the ventilation shaft.

Figure 2.3.1 contains the contours for lead deposition. This shows that deposition close to the mine activities is approximately an equal combination from the mine activities *per se* and from the free areas 80% controlled for dust lift off. On the other hand for locations further away from the mine activities deposition is largely the result of dust lift off from the free areas.

In order that an appreciation can be gained of the potential impact from just the mine proposal, projected increases in soil concentrations have been calculated using annual lead deposition rates.

- for 'free areas' only (80% dust control) (Scenario 1; S1b),
- for the 'mine activity' only (Scenario 2; S2)
- and their combined 'cumulative' impact (Scenario 3; S3).

For practical purposes lead in soil is chemically stable (i.e. has an infinite environmental half life), the physical processes likely to remove deposited lead from soil at a given location are:

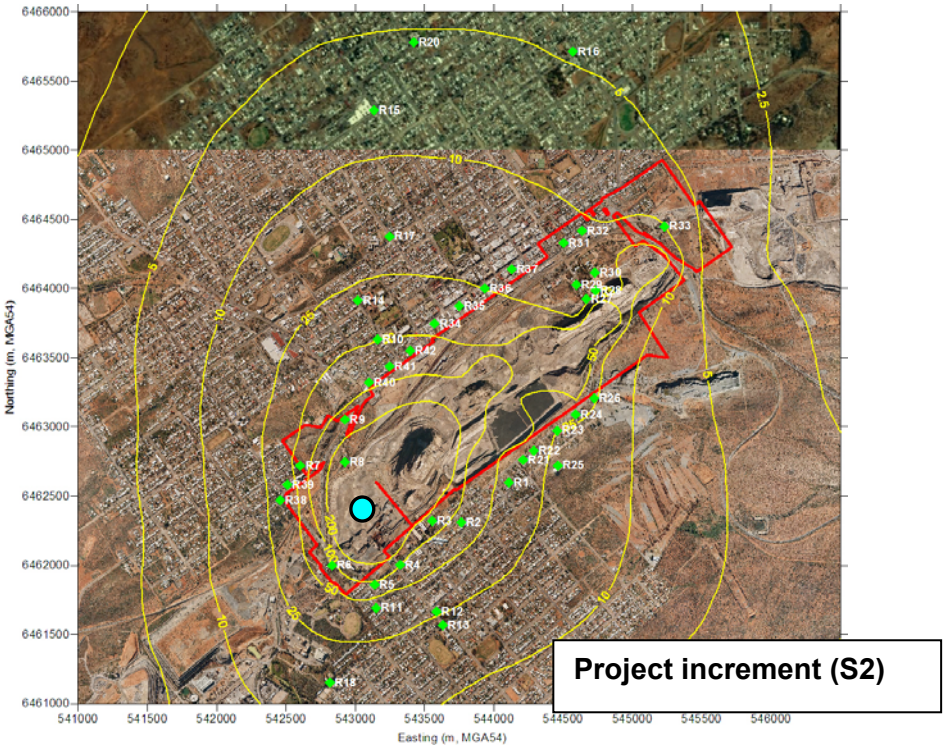
- wind erosion,
- transportation by rain washing surface soil lead laden particles away from receptor soil, and
- rain causing infiltration of lead to deeper soil so exposure is reduced (essentially cleansing the top soil layer).

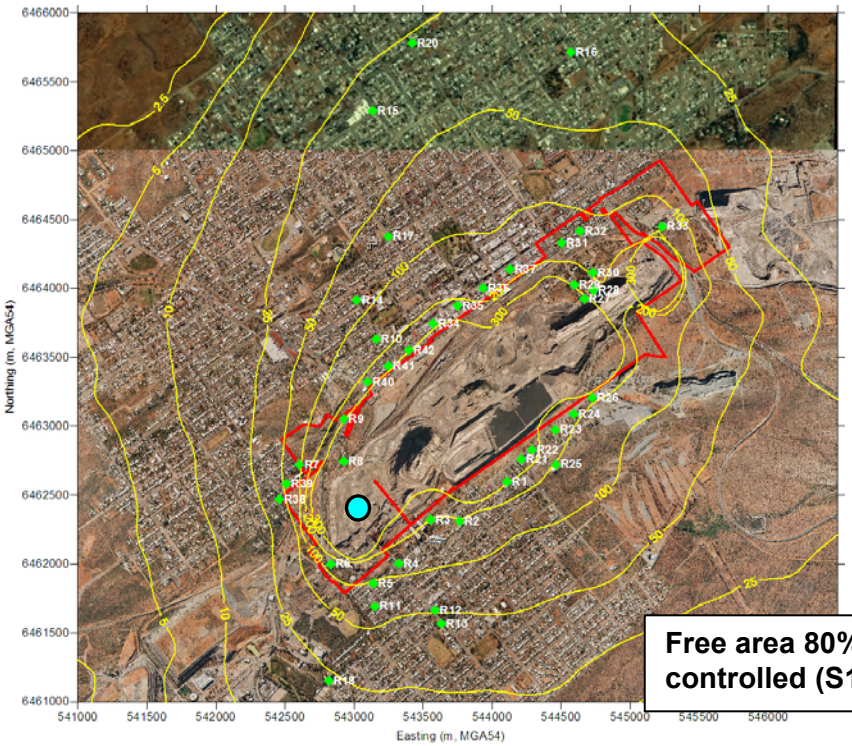
Data is not available to include these processes into the calculations of incremental increases of soil lead or other metals. Consequently it has been conservatively assumed for the HHRA that none of the deposited metals from the mine dust are removed from residential locations.

Table 2.3.1: Annual lead deposition at residential and other locations for cumulative scenario (S3).

Receptor	Total Pb deposition from TSP (g Pb/m^2/yr)	Source apportionment of total Pb deposition (% total Pb deposition)	
		Free Area	Ore
Residences			
1. Piper Street North	**0.3**	91	9
2. Piper Street Central	**0.3**	72	28
3. Eyre Street North	**0.4**	63	37
4. Eyre Street Central	**0.2**	65	35
5. Eyre Street South	**0.2**	65	35
6. South Road (1)	**0.2**	67	33
7. Carbon Lane	**0.1**	59	41
8. Old South Road	**1.1**	64	36
9. South Road (2)	**0.4**	70	30
10. Garnet & Blende Streets	**0.2**	83	17
21. Eyre Street North (1)	**0.4**	94	6
22. Eyre Street North (2)	**0.4**	94	6
23. Eyre Street North (3)	**0.3**	94	6
24. Eyre Street North (4)	**0.3**	96	4
25. Water tank, Lawton Street [a]	**0.2**	92	8
26. Quarry offices	**0.3**	97	3
27. Proprietary square (1)	**0.4**	98	2
28. Proprietary square (2)	**0.2**	97	3
29. Iodide Street (1)	**0.3**	97	3
30. Iodide Street (2)	**0.2**	97	3
31. Crystal Street (1)	**0.2**	96	4
32. Crystal Street (2)	**0.2**	96	4
33. Brown shaft dwelling	**0.2**	98	4
34. Crystal Street (3)	**0.3**	91	9
35. Crystal Street (4)	**0.3**	92	8
36. Crystal Street (5)	**0.2**	92	8
37. Crystal Street (6)	**0.2**	93	7
38. Gypsum Street (1)	**0.07**	57	43
39. Gypsum Street (2)	**0.1**	59	41
40. Silver City Hwy (1)	**0.3**	80	20
41. Silver City Hwy (2)	**0.3**	84	16
42. Silver City Hwy (3)	**0.4**	89	11
Other Locations			
11. Alma Bugdlie Pre-School	**0.1**	65	35
12. Playtime Pre-School	**0.05**	49	51
13. Alma Public School	**0.03**	36	64
14. Broken Hill High School	**0.07**	67	33
15. Broken Hill Hospital	**0.009**	30	70
16. North Broken Hill Public School	**0.005**	29	71
17. Broken Hill Public School	**0.03**	53	47
18. Rainbow Pre School	**0.03**	50	50
19. Willyama High School	**0.004**	29	71
20. Morgan Street Public School	**0.006**	30	70

[a] This receptor was included for purposes of air quality modelling, and was not included as a sensitive receptor.





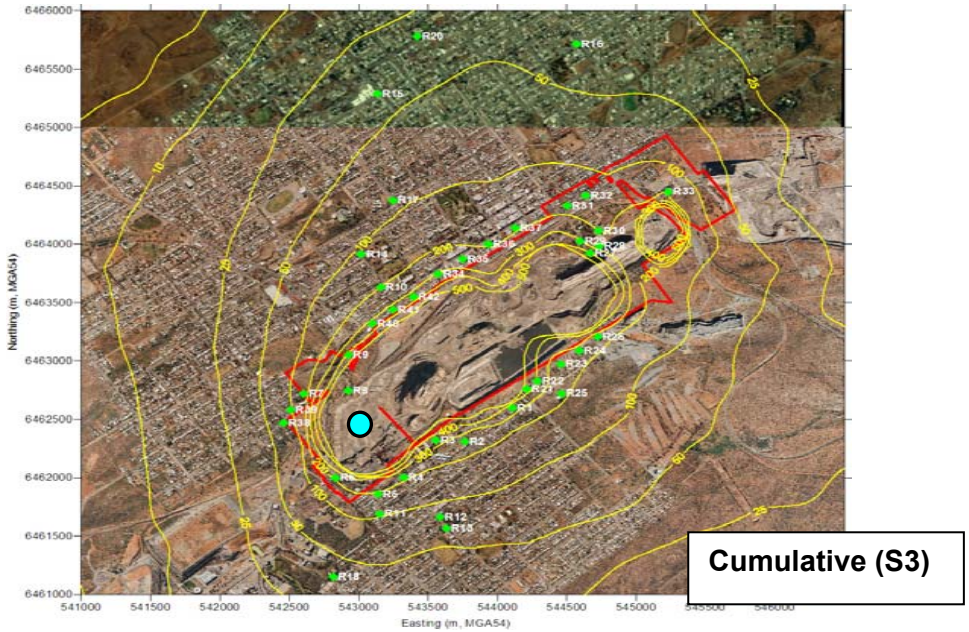


Figure 2.3.1: Contour plots of Pb deposition (mg/m^2/yr) due to dust from free areas (80% controlled, scenario S1b), mine activities (Scenario S2) and the combination of both (Scenario S3).

Note that near the boundary of the mine area near the processing activities, Pb deposition due to mine processes and dust lift off from the free areas (80% controlled) are approximately the same. Whereas further away from the boundary and mine activity (e.g. at R 14 or R17 and R26 or R33) Pb deposition is primarily from dust from free areas.

- Approximate position of mine processing activities.

Diagrams supplied by Environ Pty Ltd.

The concentration of lead and other metals in soil was calculated using Equation 2.1 (adapted from Equation 5-1 in US EPA 2005). The deposition rate ($Pb_{ann\ dep}$) for each metal was sourced from spread sheets provided by ENVIRON (ENVIRON 2010, see also Appendix A2.1), values for other parameters in Equation 1 are those recommended for untilled (non-agricultural) soil as outlined by US EPA (2005, pg. 5-21). The equation assumes the environmental half life of the metals at each receptor is infinite, and there is no physical loss from the soil due to wind erosion, percolation by rain or water runoff.

Based on a study that profiled dioxin measurements in soil, the US EPA (2005) recommend a soil mixing depth of 2 cm for untilled soils (US EPA 2005). This mixing depth has been adopted in estimating soil lead concentrations at receptor locations after 15 years operation of the Rasp mine. The influence of soil mixing depth for deposited lead is explored in the uncertainty analysis in Section 7.

$C_s = [Pb_{ann\ dep}\ (g\ Pb/m^2/yr) \times T\ (yr) \times 100] \div [\partial\ (g/cm^3) \times D\ (cm)]$ ……..**Equation 2.1**

Where:

C_s = Concentration of Pb in soil after a given number of years of mine operation (µg Pb/g soil).
$Pb_{ann\ dep}$ = Annual deposition of Pb from TSP onto residential soil from mine site (g $Pb/m^2/yr$). This value is receptor and scenario specific.
T = Time of mine operation (15 years).
100 = Units conversion factor (10^6 µg/g)/10^4 cm^2/m^2).
∂ = Soil bulk density, assumed to be 1.5 g/cm^3. This value is the recommended default by the US EPA (2005). US EPA guidance states that literature values for soil bulk density range from 0.93-1.84 g/cm^3, depending on soil type. It is not stated what soil type coincides with the value of 1.5 g/cm^3, and the original references were not available to Toxikos to verify the value selected. Therefore, the default value used by the US EPA (2005) has been adopted on face value in this risk assessment.
D = Soil mixing depth, assumed to be 2 cm (US EPA 2005).

Note that the lead deposition rate was provided as an annual value from TSP. To calculate the accumulation of metal in soil the annual deposition rate was simply multiplied by the number of years of exposure based on mine life (this assumes no loss of lead from soil).

The calculated soil concentrations (Table 2.3.2) representing 15 years of mine operation were used as inputs for the three scenarios evaluated for risk characterisation using the TDI analysis (Section 4) and by modelling incremental blood lead concentrations in children (Section 5).

Table 2.3.2: Calculated soil lead concentrations at selected receptors representing 15 years of mine operation for each Scenario evaluated in this HHRA, assuming zero loss of lead.

Selected Receptor	Calculated soil Pb concentrations (µg/g) after 15 years			
	Scenario 1a	Scenario 1b	Scenario 2	Scenario 3
Residences				
1. Piper Street North	702	140	25	166
2. Piper Street Central	427	85	55	140
3. Eyre Street North	566	113	112	225
4. Eyre Street Central	321	64	60	124
5. Eyre Street South	241	48	45	93
8. Old South Road	1646	329	223	552
9. South Road (2)	612	122	76	199
10. Garnet & Blende Streets	411	82	30	112
23. Eyre Street North (3)	601	120	15	135
32. Crystal Street (2)	346	69	7	76
36. Crystal Street (5)	463	93	15	106
38. Gypsum Street (1)	82	16	20	35
Other Locations				
11. Alma Bugdlie Pre-School	168	34	28	62
12. Playtime Pre-School	39	8	18	26
14. Broken Hill High School	89	18	18	36
17. Broken Hill Public School	20	4	10	15
18. Rainbow Pre School	19	4	9	13

A sensitivity analysis (Section 7.2) has been undertaken to examine the:

- influence of an annual removal of 10% to 35% of deposited lead (Figure 7.2.1) and
- assumed depth (0.5 – 10 cm) of surface soil into which the deposited lead is mixed (Figure 7.2.2).

The incremental accumulation of lead in residential soil over the life of the mine is shown in Figure 2.3.2, the calculated accumulation for all receptors after 15 years is summarised in Figure 2.3.3 and Table 2.3.2.

In Figure 2.3.2, showing the rate of accumulation over time, for each scenario (S1b, S2 and S3) the receptors were chosen on an approximate geometric scale of descending lead deposition rates:

- R8 - $Pb_{ann\ dep}$ 1.1 g/m^2/yr (89% from 'ore') (Residence)

- R3 - $Pb_{ann\ dep}$ 0.45 $g/m^2/yr$ (74% from 'ore') (Residence)
- R9 - $Pb_{ann\ dep}$ 0.41 $g/m^2/yr$ (74% from 'ore') (Residence)
- R11 - $Pb_{ann\ dep}$ 0.4 $g/m^2/yr$ (76% from 'ore') (Pre-school)
- R21 - $Pb_{ann\ dep}$ 0.12 $g/m^2/yr$ (55% from 'ore') (Residence)

From Figure 2.3.2 it is apparent that under the assumption of no loss of deposited lead to soil, the increase in soil lead concentration is linearly related to deposition rate and the period of mine operation. This is not the case if consideration is given to physical loss of lead (see Section 7). At receptor R8 (South Road) the soil lead concentrations approach the health investigation guideline level (HIL) of 300 µg Pb/g soil after approximately 9 years of mine operation ('cumulative' scenario'). At 10 and 15 years the soil lead level at this receptor is about 368 and 552 µg Pb/g soil respectively for Scenario 3.

At the receptor with the next highest dust deposition (R3, Eyre Street North) the soil lead levels in Scenario 3 are predicted to come close to, but not exceed the HIL after 15 years of mine operation; the incremental increase attributed to just the mine operations and the existing free areas (80% controlled) after 15 years is ~225 µg Pb/g soil (Scenario 3). The lead accumulation in soil at other receptors is well below the residential HIL – A value (Figure 2.3.3).

As expected, receptors close to operations of the proposed mine accumulate more lead over 15 years of mine operation than do locations further away (Figure 2.3.3). Thus, assuming no loss and 2 cm soil mixing depth, receptors

- R8, R3, R9 and R21 accrue 552 µg Pb/g, 225 µg Pb/g, 199 µg Pb/g, and 205 µg Pb/g soil respectively (for Scenario 3), but
- Other receptors only 2-183 µg Pb/g soil.

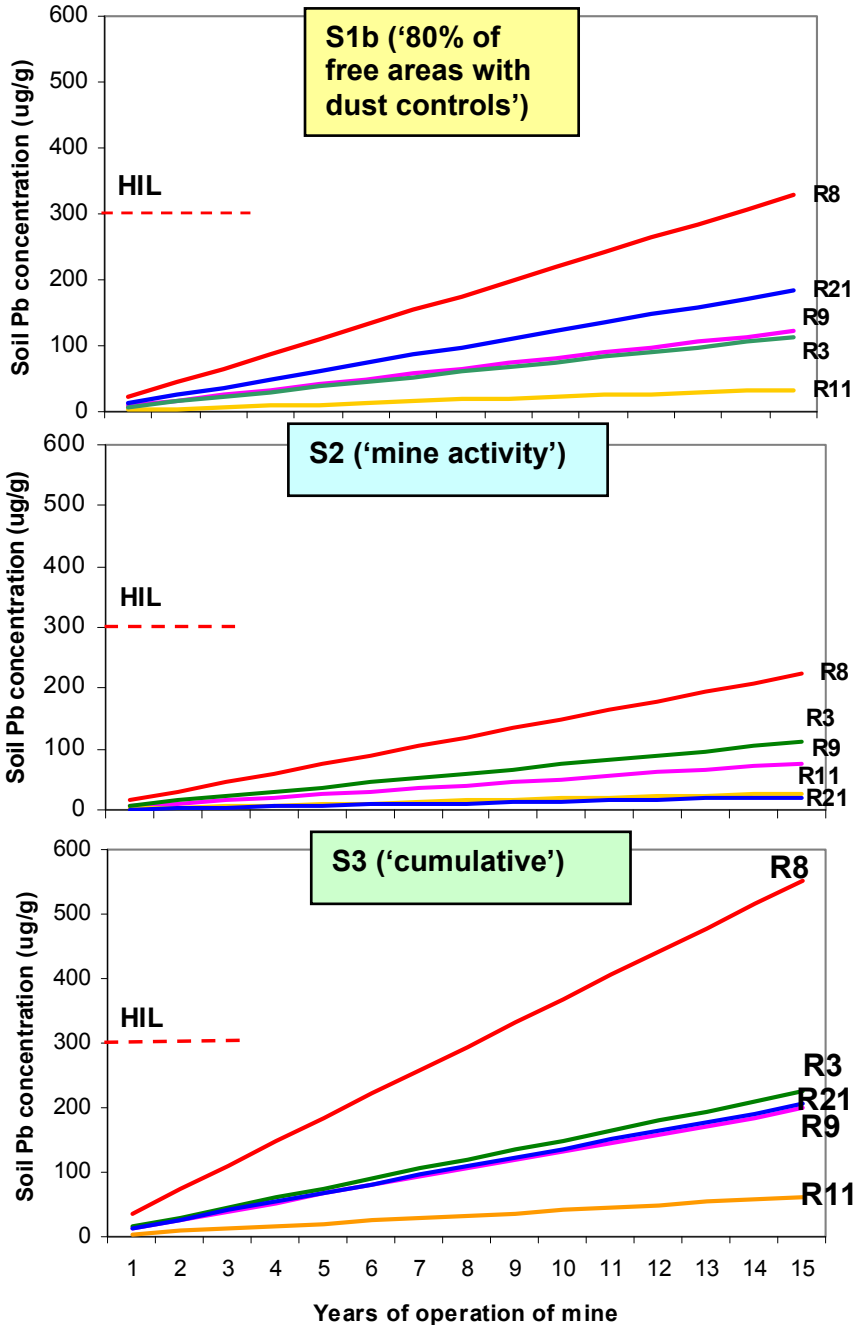


Figure 2.3.2: Predicted increases in soil Pb concentration at selected receptors (R3, 8, 9, 11 & 21). (Assumes no loss of deposited lead from soil and a soil mixing depth of 2 cm).

S1= Scenario 1b (i.e. '80% of free areas with dust controls'). Pb deposition rates (g Pb/m^2/yr) for these receptors is modelled to be 0.2 (R3), 0.7 (R8), 0.2 (R9), 0.07 (R11), and 0.4 (R21).

S2= Scenario 2 (i.e. 'mine activity'). Pb deposition rates (g Pb/m^2/yr) for these receptors is predicted to be 0.2 (R3), 0.4 (R8), 0.2 (R9), 0.06 (R11) and 0.04 (R21).

S3= Scenario 3 (i.e. 'cumulative'). Pb deposition rates (g Pb/m^2/yr) for these receptors is predicted to be 0.4 (R3), 1.1 (R8), 0.4 (R9), 0.1 (R11) and 0.4 (R21).

HIL = Health Investigation Level, 300 µg Pb/g (NEPM 1999).

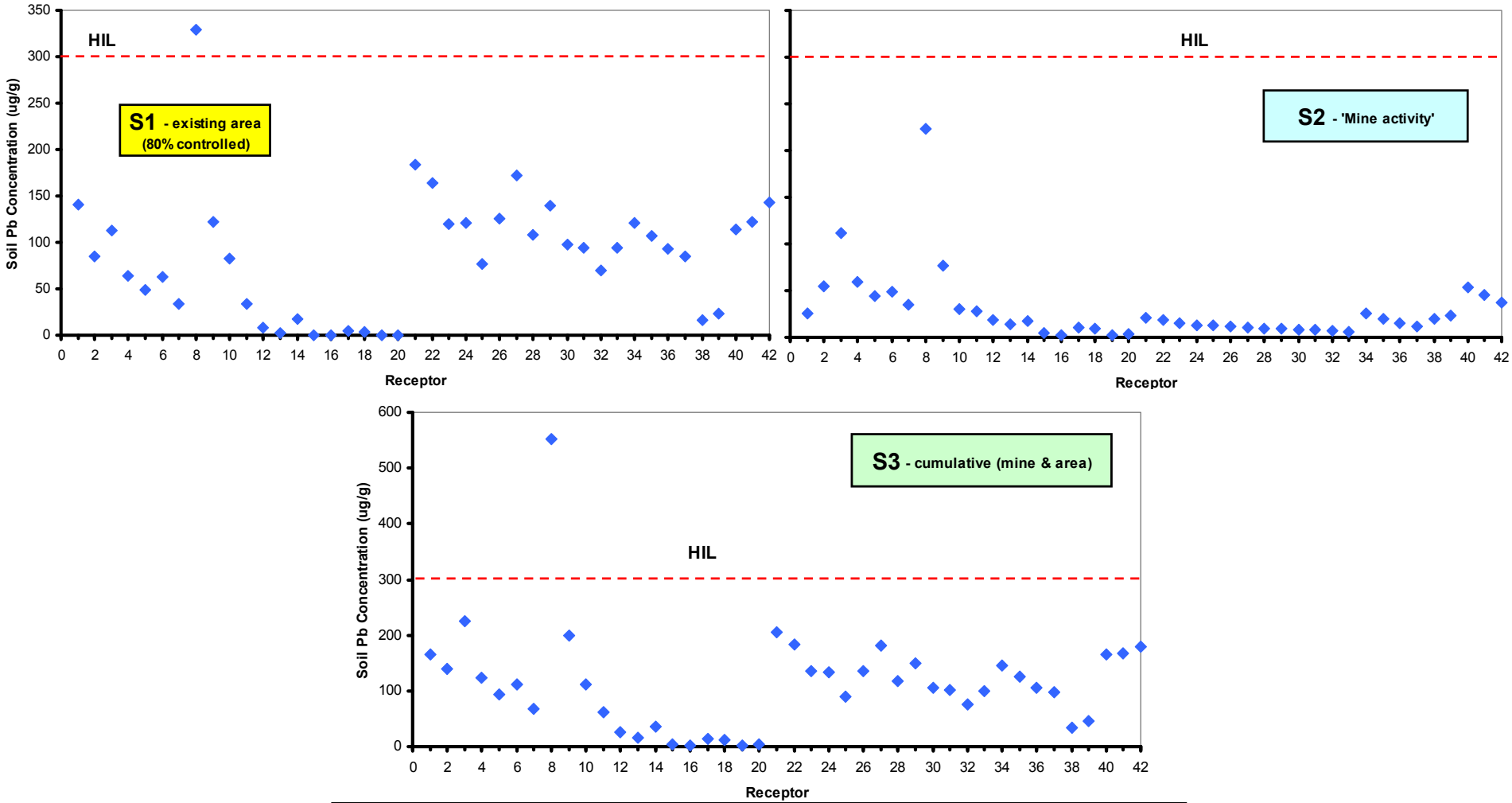


Figure 2.3.3: Receptor Soil Pb concentrations (assuming no loss) after 15 years of mine life and HIL for all receptors and scenarios.

Tables 2.3.1 and Appendix 3 respectively provide the source apportionment of lead deposition as percentage contribution from each of the major dust sources within the proposed mine site (for Scenario 3) and the total amount of lead in soil after different years of mine operation. In Figure 2.3.3 this information has been broken down into the incremental amount of lead in soil at the discrete receptors for Scenarios 1b (free areas, 80% controlled) and 2 (mine activity) after 15 years of operation.

Figure 2.3.4 importantly shows:

- Locations close the boundary of the mine lease accumulate the greatest amount of lead in soil (as also seen in Figure 2.3.3).
- The source of lead changes according to distance from the proposed mine:
  - Locations close to the mine are equally affected by lead from process activities and by dust from the free areas.
  - Further away from the mine the incremental soil lead increase is influenced little by dust from process activities but the lead in soil is strongly affected by dust from mine area sources and roads.

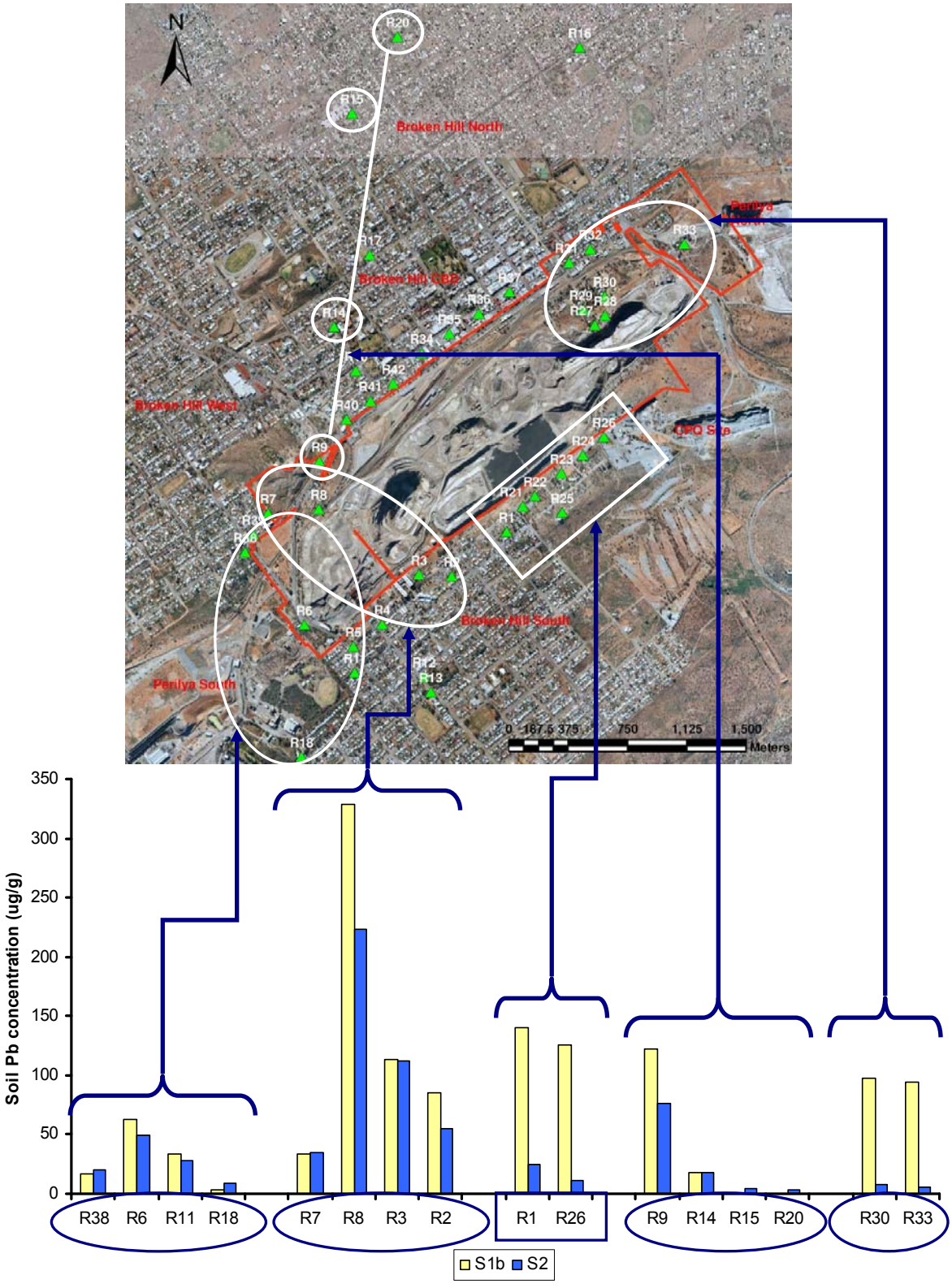


Figure 2.3.4: Source contribution to soil Pb from free areas (S1b, 80% control) and mine activity (S2) after 15 years relative to distance from the mine site.
Near the mine site free area dust and mine activity dust have approximately equal contributions to incremental off-site soil Pb, however further away Pb from free area dust has greater influence.

2.4 Soil concentrations used for exposure calculations

Soil concentrations of lead and other metals used in the exposure calculations for the different risk characterisation methods are summarised in Text Box 2.4.1, Table 2.3.2 (lead only at selected receptors) and Appendix 3. Table 2.3.2 shows soil lead concentrations after 15 years of mine operation for the receptors selected as being representative of locations around the proposed mine.

For the risk characterisation using TDI analysis for lead, incremental soil concentrations (Section 2.3) were added to an assumed existing background soil lead concentration described in Section 2.2.

Incremental increases in blood lead levels in children at specific receptor locations were modelled using the incremental soil lead concentrations (Section 5.4.1).

For comparison of blood lead levels in children exposed to lead from only the ‘existing 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, 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,.

Text Box 2.4.1: Soil concentrations used in exposure calculations

Risk characterisation via TDI considerations (Section 4):

- *For Pb (Section 4.3):*

Receptor specific incremental soil Pb (Section 2.3, Appendix A3) + assumed existing soil Pb for the risk zone (Section 2.2)

- *For other metals (Section 4.4):*

Receptor specific incremental soil metal concentration (Section 2.3, Appendix A3)

Risk characterisation via blood Pb modelling (Section 5.4):

- *For incremental blood Pb (Section 5.4.1):*

Receptor specific incremental soil Pb (Section 2.3, Appendix A3).

- *Comparison of existing free areas with cumulative scenario during mine operation (Section 5.4.2):*

Receptor specific incremental soil Pb (Section 2.3) + assumed variable existing soil Pb for the risk zone (Section 5.4.2).

2.5 Bioaccessibility

2.5.1 Bioaccessibility and Bioavailability concepts

Inorganic substances (i.e. metals) occur in soil as a complex mixture of solid phase compounds of varying particle size and morphology. These compounds may include discrete mineral phases, co-precipitated and sorbed species associated with soil minerals or organic matter, and dissolved species that may be complexed by a variety of organic and inorganic ligands (Juhasz et al. 2009; Ruby et al. 1999). A substance's dissolution properties (and, hence, its bioavailability) is therefore largely dependent on the nature of associations within/between these phases. The bioaccessible fraction of a given compound can vary considerably between different soils and chemicals.

Oral bioavailability (absolute bioavailability) is defined as the fraction of an orally administered dose of chemical that reaches the systemic circulation (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 consists of 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 markedly influences 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.

Thus for a chemical to be absorbed into the systemic circulation it must be in a form available for uptake. For absorption to take place a degree of desorption or dissociation from the medium in which it is present is required, the degree that this can occur is termed the substance's bioaccessibility from the medium (UK EA 2002). For soil, therefore, the bioaccessible fraction represents the amount of contaminant partitioned from the soil that is available for absorption.

There are a number of test protocols for bioaccessibility using *in vitro* digestion models that are intended to simulate conditions in various compartments of the gastrointestinal tract (saliva, stomach, small intestine). The RIVM (2009), UK Environment Agency (2002; 2005) and Van de Wiele et al. (2007) describe the protocols for many of these test systems.

For determining the bioaccessibility of metals from surface soil at the mine site and mine ore the method of Ruby et al. (1996) was used. This is a physiologically based extraction test (PBET), a two-stage digestion system simulating the leaching of a solid matrix in the human stomach and small intestine under feed and fasting conditions. Validation has been undertaken for lead and arsenic using oral bioavailability studies in rats, rabbits or monkeys. This test was chosen because a reputable laboratory in Australia (enTox at Queensland University) was set up to perform the test and apply it in their research programs. In addition 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.

2.5.2 Site dust for bioaccessibility analysis

During mine operations particulate matter (i.e. dust) at residential locations originates from various sources – roads on the site (paved and unpaved), the wider areas around the operations, ore handling, ore stockpile emissions, from the ventilation shaft, as well as the tailings material and concentrate. In the dispersion modelling these sources have been divided into "free areas" for the first two and "ore" for the rest. It is well recognized that different types of lead and different mineralogical forms of lead are absorbed differently into the body and is a major factor in determining blood lead concentrations (US EPA 2006, ATSDR 2007). Indeed the

bioavailability of lead from soil and minerals can be very site specific. In order 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, were sent to the University of Queensland for determination of bioaccessibility (EnTox 2009). These samples are respectively representative of dust soil/dust from waste rock left from various stages of mine life (i.e. 'free areas') and process (i.e. 'ore').

Dust from existing slag storage was not included as part of the surface dust collection as it has been used, with DECCW agreement, for dust suppression, so is not likely to be part of the dust leaving site [7].

Area dust was collected from surface soil as per the procedures for sampling surface/bulk dust loading (US EPA 1993). In brief five sub-samples of approximately 200 – 300g each were obtained from different locations within five designated areas (1 – 5) of the Rasp mine site by hand brushing the surface soil (approximately top 5mm soil) with a banister brush onto a plastic dustpan. The sub-samples were transferred to a plastic bag and mixed. Each bag was twisted and fastened with clear packing tape stuck around the bottom near the samples, then folded over and taped again. Samples were transported by road express to the laboratory for bioaccessibility analysis. To further make the bioaccessibility assessment specific for the Rasp mine HRA, the assessment was performed on the fines of a 100 µm sieving as this is the particle size fraction subject to potential airlift from the ground.

Bioaccessibility of lead in process and ROM dust was determined on fines from a 150 µm sieving of drill core samples (n = 4) after they had been broken up to small pieces with a hammer (roughly simulating the crushing process).

Figure 2.5.1 shows the approximate position for each dust sample prior to mixing within the designated areas of the Rasp mine site; Table 2.5.1 provides the specific coordinates of each sub-sample.

[7] Personal communication Gwen Wilson 16/11/09.

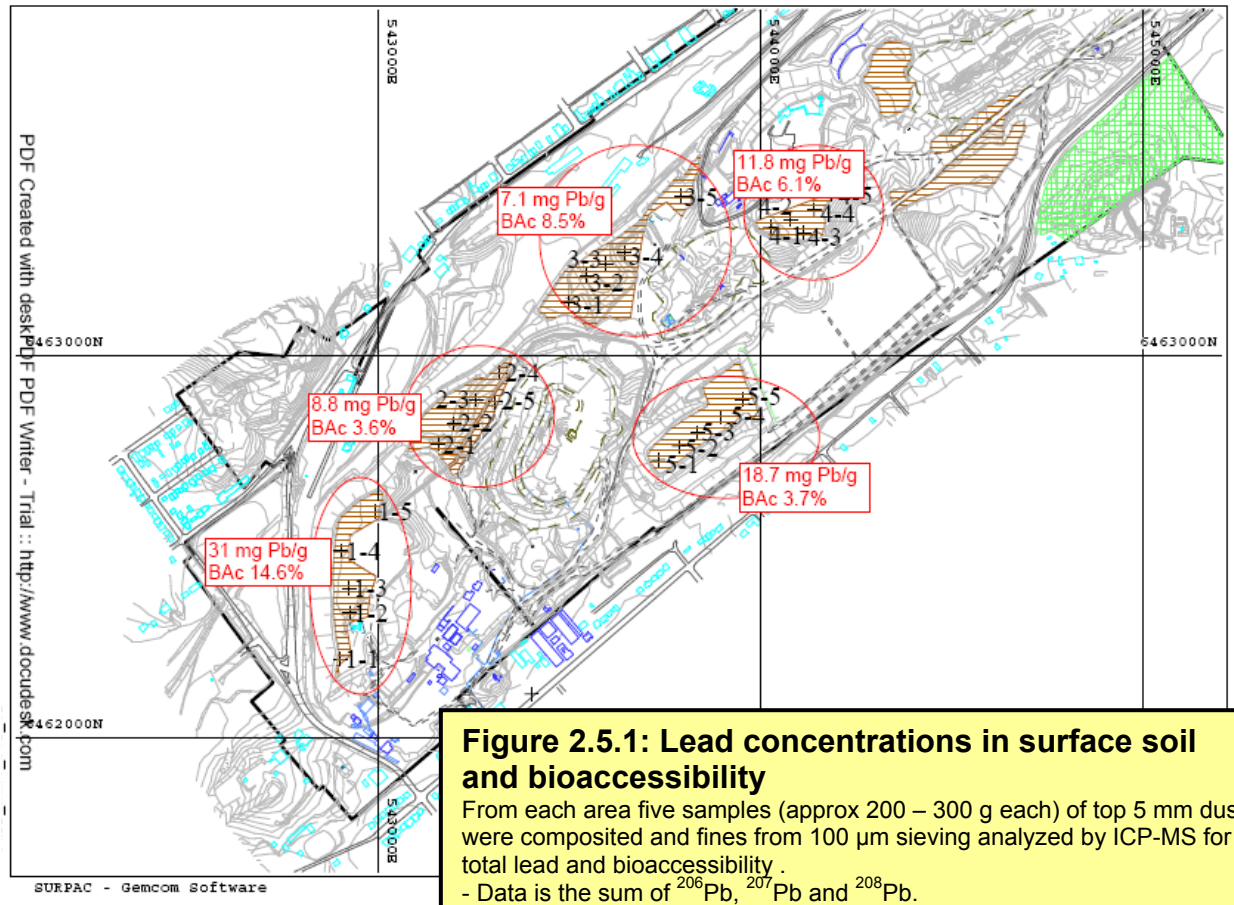


Figure 2.5.1: Lead concentrations in surface soil and bioaccessibility

From each area five samples (approx 200 – 300 g each) of top 5 mm dust were composited and fines from 100 µm sieving analyzed by ICP-MS for total lead and bioaccessibility .

- Data is the sum of ^{206}Pb, ^{207}Pb and ^{208}Pb.
- Bioacessibility (BAc) is shown as percentage (%); average BAc of all surface dust samples = 7.3% (See Table 2.5.2).

Table 2.5.1: Coordinates of sub-sample locations within the designated areas of the Rasp mine site [a]

Sampling point	Northing	Easting
1-1	6462206.394	542899.605
1-2	6462327.999	542923.926
1-3	6462392.045	542921.494
1-4	6462489.329	542900.416
1-5	6462591.621	542990.94
2-1	6462770.276	543155.491
2-2	6462822.954	543196.463
2-3	6462884.737	543253.694
2-4	6462955.625	543316.127
2-5	6462879.534	543303.77
3-1	6463139.978	543495.124
3-2	6463211.395	543543.037
3-3	6463240.323	543593.661
3-4	6463271.059	543642.478
3-5	6463418.413	543791.64
4-1	6463336.399	544023.881
4-2	6463356.859	544077.915
4-3	6463322.76	544112.538
4-4	6463383.613	544138.243
4-5	6463422.433	544184.932
5-1	6462727.7	543731.388
5-2	6462764.362	543784.878
5-3	6462799.221	543833.56
5-4	6462841.292	543894.864
5-5	6462885.167	543951.359

[a] See Figure 2.5.1 for map of locations and bioaccessibility results.

2.5.3 Bioaccessibility results - lead

The bioaccessibility tests were performed in duplicate for each sample with duplicates showing reasonable concordance with each other (Table 2.5.2).

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.

Table 2.5.2: Lead bioaccessibility from surface area dust and mine ore [a]

Sample		Total mean lead content [a] (mg/g)	Bioaccessibility (BAc (%) [b]			
			^{206}Pb	^{207}Pb	^{208}Pb	Average
			Surface dust			
Area 1	A	10.6	17.3	17.2	17.5	14.6
	B	9.9	12.0	11.9	12.2	
Area 2	A	3.1	3.6	3.6	3.5	3.6
	B	2.8	3.6	3.7	3.6	
Area 3	A	2.1	9.0	9.0	8.9	8.5
	B	2.6	8.0	8.0	8.0	
Area 4	A	4.2	6.1	6.0	5.9	6.1
	B	3.6	6.2	6.2	6.1	
Area 5	A	5.9	3.9	3.9	3.8	3.7
	B	6.6	3.6	3.7	3.5	
Overall Pb bioaccessibility from site area dust						**7.3**
			Mine ore			
WMDD 4063	A	60.8	1.3	1.5	1.4	1.26
	B	80.9	1.0	1.2	1.1	
WMDD 4075	A	60.3	1.4	1.6	1.5	1.55
	B	63.7	1.1	1.3	1.2	
WMDD 4079	A	62.7	0.8	0.9	0.8	0.88
	B	64.8	0.9	1.0	0.9	
WMDD 4080 [c]		73.4	2.1	2.1	2.1	2.1
Overall Pb bioaccessibility from mine ore						**1.4**

[a] The average of the three Pb isotopes is provided to give an indication of the total Pb content of the samples.

[b] Bioaccessibility of each lead isotope is calculated from the amount of the relative isotope in a strong acid extract of the sample.

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

Thus, receptor-specific BAc's were calculated. For lead in soil and dust, BAc's were converted to an absolute bioavailability by multiplying the receptor-specific BAc by 50%, which corresponds to the bioavailability of soluble lead in water and food (US EPA 1999, ATSDR

2007). The calculated receptor-specific bioavailabilities were then used as input values for the determining blood lead levels (Sections 5 and 6).

2.5.4 Bioaccessibility results –other metals

The physiologically based extraction test (PBET) described in the previous section was optimized for estimating the bioaccessibility of lead. The test has been formally validated for lead and As (Ruby et al. 1996, 1999), but not for the other metals addressed in this risk assessment. In optimizing the test for lead, considerable dilutions, particularly for the ore sample, were necessary to ensure the digestion for 'total' lead content of the sample was within the instrument response range[8]. This meant that for the ores, some elements present at much lower concentrations were diluted in the 'total' digestions to levels below that which could be quantified by ICP. Consequently bioaccessibility data for metals other than lead and As have associated with them some marked uncertainty an it was assumed that their bioaccessibility would be 100%. Table 2.5.3 gives the bioaccessibility results for arsenic.

[8] E-mail Correspondence with Jack Ng on 18th of February, 2010.

Table 2.5.3: Arsenic bioaccessibility from surface area dust and mine ore

Sample		Total metal content (µg/g)	Bioaccessibility (BAc (%)	
			^{75}As	Average
			Surface dust	
Area 1	A	75.4	3.5	3.2
	B	82.4	2.8	
Area 2	A	40.8	2.7	2.7
	B	33.4	2.7	
Area 3	A	56.5	3.2	3.2
	B	47.2	3.2	
Area 4	A	34.8	6.4	6.6
	B	34.0	6.7	
Area 5	A	125.6	2.3	2.1
	B	119.5	1.9	
Overall As bioaccessibility from site area dust				**4**
			Mine ore	
WMDD 4063	A	143.2	0.4	0.3
	B	324.3	0.1	
WMDD 4075	A	0	-	-
	B	0	-	
WMDD 4079	A	0	-	-
	B	0	-	
WMDD 4080		50.5	0.4	0.4
Overall As bioaccessibility from mine ore				**4 (0.4)** [a]

[a] Although ore samples give an average BAc for As of about 0.4%, its BAc was conservatively assumed to be 4% for both surface dust and ore samples, due to only two of the ore sample returning results.

Therefore, specific bioaccessibilities were applied for Pb and As based on the PBET test results. All other metals were conservatively assumed to be 100% bioaccessible in the TDI analysis.

For the As TDI analysis, source apportionment information by receptor was not available. Therefore, the calculated As BAc from the PBET test (4%) was applied to all receptors.

3. Hazard Identification

Toxicological hazard information on the major metals can be found in Appendix 1. The metal of most concern for this risk assessment is lead and a brief description of its hazards is below.

Lead is a naturally occurring element, making up about 0.0013% of the Earth's crust (UNEP 2006). There are three chemical forms of lead: metallic lead, inorganic lead compounds and organic lead compounds (containing carbon). Lead is usually obtained from sulphide ores, often in combination with other elements such as zinc, copper and silver. The main lead mineral is galena (PbS). Other common varieties include cerussite ($PbCO_3$), plattnerite (PbO_2) and angelsite ($PbSO_4$).Lead exists in three oxidation states: Pb(0) - the elemental form, Pb(II) and Pb(IV). Metallic lead, Pb (0), exists in nature, but its occurrence is rare (UNEP 2006, ATSDR 2007).

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 GI 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. ATSDR (2007) summarise a comparative study of dermal absorption of inorganic and organic salts of lead conducted in rats where approximately 100 mg of lead was applied in an occluded patch to the shaved backs of rats. Based on urinary lead measurements made prior to and for 12 days following exposure, lead compounds could be ranked according to the relative amounts absorbed (i.e., percent of dose recovered in urine): Pb naphthenate (0.17%), Pb nitrate (0.03%), Pb stearate (0.006%), Pb sulfate (0.006%), Pb oxide (0.005%), and metal Pb powder (0.002%).

Table 3.1 summarises the bioavailability factors selected for the present HRA.

Table 3.1: Bioavailability factors used in the present HRA [a]

Exposure route	Absorption factor	Basis
Oral	50%	Human studies investigating the absorption of water soluble Pb compounds indicate that 40-50% of ingested Pb 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 Pb absorbed was approximately 95% of the soluble deposited Pb 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 Pb in rats showing a dermal absorption for most inorganic Pb compounds (both relatively soluble and insoluble) to be less than 0.01% (Pb nitrate (0.03%), Pb stearate (0.006%), Pb sulfate (0.006%), Pb oxide (0.005%), and metal Pb powder (0.002%). US EPA 2006, ATSDR 2007.

[a] The above bioavailabilities were modified for bioaccessibility as per Section 2.5.

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 lead from bone stores to blood (US EPA 2006 p 4-18).

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 3.2 provides a summary of the blood lead levels associated with a range of effects.

Lead has been shown to have effects on haemoglobin synthesis and anaemia has been observed in children at lead blood levels above 40µg/dl. Lead is also known to cause kidney damage. Some of the effects are reversible, whereas chronic exposure to high lead levels may result in continued decreased kidney function and possible renal failure. Renal effects have been seen among the general population when more sensitive indicators of function were measured (WHO 1995, US EPA 2006, ATSDR 2007).

The reproductive effects of lead in the male are limited to sperm morphology and count. In the female, some adverse pregnancy outcomes have been attributed to lead. Lead does not appear to have deleterious effects on skin, muscle or the immune system (WHO 1995, US EPA 2006, 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).

Table 3.2: Blood lead response [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 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 recently 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.

Interactions with other metals:

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

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 lead blood concentrations and time-integrated lead blood 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.

4. Risk Characterization – TDI

4.1 Introduction

In this section, lead intake from all complete exposure pathways is calculated and compared to the tolerable daily intake (TDI) set by WHO/JECFA (1986, 2000). This is a standard risk assessment procedure (enHealth 2004) and has been applied in other risk assessment of environmental lead exposure in Australia (e.g. Golders 2009).

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 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.

Exposure calculations for life stage daily intake of lead included:

- Predicted receptor soil concentrations after 15 years of mine operation assuming no loss of deposited lead from the residential soils (see Sections 2.3-2.4).
- Assumed existing background soil concentrations (Section 2.2)
- Background dietary intake (Section 4.2.5)
- Intake from ingestion of reticulated water (Section 4.2.5)
- Incremental air concentrations of lead predicted by dispersion modelling (ENVIRON 2010).
- Assumed lead concentration in background dust (Section 4.2.5, ENVIRON 2010).

Parameter values for the above lead intakes are summarised in Section 4.2.7.

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

I_{oral} = Soil ingestion.
I_{derm_s} = Dermal absorption of metal from soil.
I_{inhal} = Inhalation of airborne dust delivered from mine site.

Therefore residential intake = $\underbrace{I_{oral} + I_{derm\_s}}_{\text{Receptor Site Soil}} + \underbrace{I_{inhal}}_{\text{Mine dust}}$

The equations for the different pathways considered are described in Section 4.2.

4.2 Exposure Calculations

4.2.1 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 using Equation 4.1 below.

Equation 4.1

I_{oral} (µg/kg/day)

$$= \frac{C_s \text{ (µg/g)} \times S_{oral} \text{ (mg/day)} \times 10^{-3} \times ET \text{ (hr/day)} \times 1/24 \text{ (day/hr)} \times EF \text{ (day/yr)} \times ED_x \text{ (yr)} \times BAc_{soil} \times BA_{oral}}{BW_{as} \text{ (kg)} \times AT_x \text{ (days)}}$$

Where:

I_{oral}	=	Life stage intake of metals from soil via ingestion (µg/kg bw/day).
C_s	=	Concentration of compound in soil (µg/g). Estimated from modelled soil deposition data (Sections 2.3 - 2.4).
S_{oral}	=	Age-specific rate of soil ingestion per day (mg/day) (Section 4.2.7).
10^{-3}	=	Conversion factor from mg to g (1 mg = 10^{-3} g).
ET	=	Exposure time (hr/day). 24 hours per day for residential exposure.
1/24	=	Conversion factor from days to hours (1 day = 24 hours).
EF	=	Exposure frequency (days/yr). Residential exposure assumes exposure throughout the whole year (365 days/year).
ED_x	=	Age-interval exposure duration (yr). Number of years in relevant age dependent exposure span [9].
BAc_{soil}	=	Bioaccessibility of metal in soil. Unitless. (Section 2.5).
BA_{oral}	=	Bioavailability of ingested metal. Assumed to be the same as the bioavailability in the study from which the TDI was derived. For Pb this was assumed to be 100%. Unitless.
BW_{as}	=	Age-specific body weight in kg.
AT_x	=	Averaging time (days). This is the age-specific exposure period.

4.2.2 Dermal absorption

Dermal absorption of inorganic lead is much lower than by ingestion or inhalation (ATSDR 2007, US EPA 2006). Few studies have provided quantitative estimates of dermal absorption of inorganic lead in humans. For example, Moore et al. (1980) found absorption of ^{203}Pb-labeled lead acetate in cosmetics following skin application to 8 male volunteers for 12 hours to be $\leq$0.3%, based on whole-body, urine and blood ^{203}Pb measurements. The applied preparations had concentrations of 0.12 mg Pb in 0.1 mL or 0.18 mg Pb in 0.1 g of a cream. The absorption was predicted to be 0.06% in normal use of such preparations.

[9] Neonate exposure is considered to be very low for the first 0.5 years of life. Hence for the ages 0-3 years the exposure interval is 2.5 years (US EPA 1994a, pg. 1-19).

Animal studies have also revealed dermal absorption of inorganic lead is low. From rat studies described by ATSDR (2007), where approximately 100 mg of lead was applied in a patch to the shaved backs of rats, urinary measurements made prior to and for 12 days following exposure revealed that about 0.006% of Pb sulphate, 0.03% of Pb nitrate, 0.005% of Pb oxide, and 0.002% of metal Pb powder was absorbed through the skin. Percent of dose recovered in urine was calculated by ATSDR (2007). No other tissues were measured.

Because lead from the Rasp mine is predominantly in the form of weathered galena (anglesite, or lead sulphate), and has low bioaccessibility (Section 2.5) the absorption of mining lead through the skin is expected to be negligible. In these considerations the absolute bioavailability of 'soluble' lead was taken to be 0.01% [10]

Equation 4.2

I_{derm_s} (µg/kg/day) =

$[C_s$ (µg/g) x SA_d (mg/cm^2) x SA_{skin} (cm^2) x 10^{-3} x ET (hr/day) x 1/24 (day/hr) x EF (day/yr) x ED_x (yr) x BAc_{soil} x ABA_{derm})] ÷ [BW_{as} (kg) x AT_x (days)]

Where:

I_{derm_s}	=	Intake of metals from soil via dermal absorption (µg/kg bw/day)
C_s	=	Concentration of compound in soil (µg/g). This is calculated from modeled soil deposition data (refer to Sections 2.3-2.4).
SA_d	=	Soil adherence (mg/cm^2).
SA_{skin}	=	Age-specific surface area of skin (cm^2).
10^{-3}	=	Conversion factor from mg to g (One mg = 10^{-3} g).
ET	=	Exposure time (hr/day). This is assumed to be 24 hours per day, since it is residential exposure.
1/24	=	Conversion factor from days to hours (1 day = 24 hours).
EF	=	Exposure frequency (days/yr). Residential exposure assumes exposure throughout the whole year (365 days/year).
ED_x	=	Age-interval exposure duration (yr). This refers to the number of years the resident is exposed. The resident is assumed to have lived at the receptor since birth.
BAc_{soil}	=	Bioaccessibility of metal in soil. Unitless.
ABA_{derm}	=	Absolute bioavailability of dermally absorbed metal. Unitless. This is assumed to be 0.01% for all inorganics [2].
BW_{as}	=	Age-specific body weight in kg.
AT_x	=	Averaging time (days). This refers to the age-specific exposure.

[10] The dermal soil bioavailability factor for inorganic substances of 0.01% is based on the rationale (commonly used to set existing Australian soil health investigation levels for inorganic substances) that inorganic compounds have negligible absorption through intact skin. This is consistent with default bioavailability of 0% for inorganics from soil used by the UK Environment Agency (UK EA 2009, p. 113) and the Dutch National Institute for Public Health and Environment (RIVM 2007, p. 19). For practical purposes, the value of 0.01% is considered negligible.

4.2.3 Inhalation of airborne dust delivered from mine site

Estimated daily intake via inhalation of airborne dust was calculated using Equation 4.3 below.

Equation 4.3

I_{inhal} (µg/kg/day) =

$[C_A$ (µg/m^3) x IR_{as} (m^3/day) x ET (hr/day) x 1/24 (day/hr) x EF (day/yr) x ED_x (yr) x BAc_{air} x $ABA_{inhal}] \div [BW_{as}$ (kg) x AT_x (days)]

Where:

I_{inhal}	=	Intake of metal from air delivered from the mine site via inhalation (µg/kg bw/day).
C_A	=	Modelled concentration of compound in PM_{10} fraction in air (µg/m^3).
IR_{as}	=	Age-specific inhalation rate (m^3/day).
ET	=	Exposure time (hr/day). This is assumed to be 24 hours per day, since it is residential exposure.
1/24	=	Conversion factor from days to hours (1 day = 24 hours).
EF	=	Exposure frequency (days/yr). Residential exposure assumes exposure throughout the whole year (365 days/year).
ED_x	=	Age-interval exposure duration (yr). This refers to the number of years the resident is exposed. The resident is assumed to have lived at the receptor since birth.
BAc_{air}	=	Bioaccessibility of metal in air. Unitless. Taken to be BAc of metal in PM_{10} modeled data.
ABA_{inhal}	=	Absolute bioavailability of inhaled metal. Unitless. Taken to be 100%.
BW_{as}	=	Age-specific body weight in kg
AT_x	=	Averaging time (days). This refers to the age-specific exposure.

4.2.4 Total age-specific daily intake

Total intakes were calculated for each pathway for four age groups:

- 0.5 up to 3 years.
- 3 up to 13 years.
- 13 up to 18 years.
- 18 up to 70 years.

Total intakes involved consideration of background intakes, as per Equation 4.4.

Equation 4.4

I_{total_as} (µg/kg/day) = $I_{ingest\_s} + I_{derm\_s} + I_{inhal} + I_{Bkgd}$

Where:

I_{total_as}	=	Total age-specific intake. The sum of metal intake via soil ingestion, dermal absorption, and inhalation.
I_{ingest_s}	=	Intake of metals from soil via ingestion (µg/kg bw/day).
I_{derm_s}	=	Intake of metals from soil via dermal absorption (µg/kg bw/day).
I_{inhal}	=	Intake of metal from air delivered from the mine site via inhalation (µg/kg bw/day).
I_{Bkgd_as}	=	Age-specific background intake of metal (Section 4.2.5).

4.2.5 Background intake

Background intake of lead from all sources was incorporated into the intake calculations for this metal, however only very limited information was available regarding background intakes for the other metals (Table 4.2.1). Hazard quotients for the metals other than lead have been generated, both including and excluding the available information on background intakes (Section 4.4). The hazard indices, which sum all hazard quotients for all the metals, were calculated excluding background information except for lead. 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.

Age-specific background intake of Pb (I_{Bkgd_as}) is further defined with Equation 4.5 where background intake consists of intake from soil, air, food, water and consumer goods.

Equation 4.5

$$I_{Bkgd\_as}\ (\mu g/kg/d) = I_{soil\_bkgd} + I_{air\_bkgd} + I_{food\_bkgd} + I_{water} + I_{consumergoods}$$

Where:

I_{Bkgd_as} = Age-specific background intake of metal

I_{soil_bkg} = This is equivalent to the approximate intake of Pb from existing soil. Upper 95% CI soil Pb concentrations were sourced from Boreland (2010) and vary by risk zone (Section 2.2). Age-specific background soil intakes were calculated using Equation 4.1.

I_{air_bkgd} = This is equivalent to the approximate intake of Pb in background air. The background air PM_{10} Pb concentration was taken to be 0.2 $\mu g/m^3$, as outlined in the air quality modelling report (ENVIRON 2010a). Age-specific background air intakes of Pb were calculated using Equation 4.2.

I_{food_bkgd} = 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 Pb from diet was assumed to be at or about the top of the range for the age group; 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.[11]

I_{water} = It is assumed that tank water is not used for drinking water purposes, as recommended by the local authorities (NSW Gov 2009). The maximum Pb 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 outlined in Table 4.2.1.

[11] For 0.5-3 year olds = intakes for infants and toddlers range from 0.01-1.2 µg/kg/d (assume 1 µg/kg/d).
For 3-13 year olds = intakes for toddlers and children aged 12 years range from 0.01- 0.93 µg/kg/d (assume 0.9 µg/kg/d).
For 13-18 years olds & adults = intakes for adults and 12 year-olds range from 0.01-0.43 µg/kg/d (assume 0.4 µg/kg/d).

$I_{consumergoods}$ = The intake of Pb 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.

4.2.6 Lifetime time-weighted average intake (TWADI)

Total lifetime average daily intake was calculated from Equation 4.6.

Equation 4.6

$$I_{lifetime}\ (\mu g/kg/day) = \frac{(I_{total(0.5\text{-}3)} \times AT_x) + (I_{total(3\text{-}13)} \times AT_x) + (I_{total(13\text{-}18)} \times AT_x) + (I_{total(18\text{-}70)} \times AT_x)}{AT_{lifetime}\ (days)}$$

Where:
$I_{lifetime}$ = Total daily intake of metal over a lifetime (µg/kg/day).
$I_{total(0.5\text{-}3)}$ = Total intake of metal for a child aged 0.5-3 years (µg/kg/day).
$I_{total(3\text{-}13)}$ = Total intake of metal for a child aged 3-13 years (µg/kg/day).
$I_{total(13\text{-}18)}$ = Total intake of metal for an adolescent/adult aged 13-18 years (µg/kg/day).
$I_{total(18\text{-}70)}$ = Total intake of metal for an adult aged 18-70 years (µg/kg/day).
AT_x = Averaging time (days). This refers to the age-specific exposure.
$AT_{lifetime}$ = Averaging time for lifetime exposure (70 years x 365 days/year = 25,550)

The total lifetime intake was then compared to the tolerable daily intake of the metal to generate a hazard quotient, as per Equation 4.7.

Equation 4.7

$$HQ = \frac{I_{lifetime}\ (\mu g/kg/day)}{TDI\ (\mu g/kg/day)}$$

Where:
HQ = Hazard quotient for a metal at each receptor. Unitless.
$I_{lifetime}$ = Average lifetime daily exposure of metal for 70 year lifespan (µg/kg/day).
TDI = Tolerable daily intake over an individual's lifetime (µg/kg/day). Taken from reputable agency/organization (Appendix 1).

4.2.7 Exposure Assumptions

Exposure calculations (also applied in the IEUBK modelling) rely on a number of assumptions for exposure in the absence of site-specific data. Where possible this risk assessment incorporates exposure factors from contemporary Australian studies, where this is not appropriate data from recently published US Exposure Factors Handbooks (US EPA 2008, 2009a) has been applied with reasoning why it has been chosen.

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).

A summary of the exposure parameters used in the equations above for each life stage is given in Table 4.2.1.

Table 4.2.1: Summary of exposure parameters

Parameter		Life Stage				Comment
		Infant 0.5-3 yr	Child 3-13 yr	Adolescent 13-18 yr	Adult 18-70 yr	
RESIDENTIAL SOIL INGESTION						
Soil concentration (µg/g)	C_s	-	-	-	-	Varies by receptor and metal and is calculated from modelled soil deposition data (Section 2.3, Appendix 3).
Soil ingestion (mg/d)	S_{oral}	100	100	50	50	Default values (enHealth 2004). For adults, it is the upper end of experimental average intake [a].
Conversion factor (g/mg)	10^{-3}	10^{-3}	10^{-3}	10^{-3}	10^{-3}	Conversion factor from mg to g.
Exposure Time (hr/day)	ET	24	24	24	24	For a residential exposure scenario, it is assumed exposure is continuous.
Conversion factor: 1/24 (day/hours)	1/24	1/24	1/24	1/24	1/24	Conversion factor
Exposure Frequency (day/yr)	EF	365	365	365	365	Assumed to be continuous.
Age-interval exposure Duration (yr)	ED_x	2.5 [f]	10	5	52	This refers to the duration of exposure of the age range.
Age interval body weight (kg)	BW_{as}	15	35	55.5	75	Corresponds to the recommended body weight for the average age in the age range [b].
Age-specific averaging time (days)	AT_x	912.5	3,650	1,825	18,980	Equivalent to averaging time for each age group.
Averaging time [exposure duration (70 years) x days (365) per year]	$AT_{lifetime}$	25,550	25,550	25,550	25,550	Standard calculation for lifetime intake.

Bioaccessibility of metal in soil	BAc_{soil}	-	-	-	-	Varies with receptor and metal. Unitless. This is derived from site-specific BAc data and the relative proportion of dust at the receptor that comes from free areas or mine activity.
Absolute bioavailability of ingested metal	BA_{oral}	-	-	-	-	Varies with metal, based on TDI
RESIDENTIAL DERMAL EXPOSURE TO SOIL						
Soil concentration (µg/g)	C_s	-	-	-	-	Varies by receptor and metal and is calculated from modelled soil deposition data (Section 4.3, Appendix 3).
Soil adherence (mg/cm^2)	SA_d	0.5	0.5	0.5	0.5	Average soil adherence for children and adults based on several studies [c]
Surface area of skin exposed to soil (cm^2)	SA_{skin}	2,318	3,650	5,374	6,760	Body surface area of exposed skin [h].
Conversion factor (g/mg)	10^{-3}	10^{-3}	10^{-3}	10^{-3}	10^{-3}	Conversion factor from mg to g.
Exposure Time (hr/day)	ET	24	24	24	24	For a residential exposure scenario, it is assumed exposure is continuous.
Conversion factor: 1/24 (day/hours)	1/24	1/24	1/24	1/24	1/24	Conversion factor.
Exposure Frequency (day/yr)	EF	365	365	365	365	Assumed to be continuous.
Age-interval exposure Duration (yr)	ED_x	2.5 [f]	10	5	52	This refers to the duration of exposure of the age range.
Age interval body weight (kg)	BW_x	15	35	55.5	75	Corresponds to the recommended body weight for the average age in the age range [c].
Age-specific averaging time (days)	AT_x	912.5	3,650	1,825	18,980	Equivalent to averaging time for each age group.
Averaging time [exposure duration (70 years) x days (365) per year]	$AT_{lifetime}$	25,550	25,550	25,550	25,550	Standard calculation for lifetime intake.

Bioaccessibility of metal in soil	BAc_{soil}	-	-	-	-	Varies with receptor and metal.. Unitless. This is derived from site-specific BAc data.
Absolute bioavailability of dermally absorbed metal.	ABA_{derm}	0.0001	0.0001	0.0001	0.0001	Unitless. This is assumed to be 0.01% for all inorganics [d].
INHALATION OF METAL FROM MINE SITE AIRBORNE DUST						
Concentration of compound in air (µg/m^3)	C_A	-	-	-	-	Modelled concentration of compound in PM_{10} fraction in air (ENVIRON 2010). Varies by receptor and by metal.
Inhalation Rate (m^3/d)	IR_{as}	9.5	12.4	15.1	16	Inhalation rate for average age of age range. Derived from US EPA (2008 and 2009b)[e]
Exposure Time (hr/day)	ET	24	24	24	24	For a residential exposure scenario, it is assumed that exposure is continuous.
Conversion factor: 1/24 (day/hours)	1/24	1/24	1/24	1/24	1/24	Conversion factor
Exposure Frequency (day/yr)	EF	365	365	365	365	Assumed to be continuous.
Age-interval exposure Duration (yr)	ED_x	2.5 [f]	10	5	52	This refers to the duration of exposure of the age range.
Age interval body weight (kg)	BW_x	15	35	55.5	75	Corresponds to the recommended body weight for the average age in the age range [b].
Age-specific averaging time (days)	AT_x	912.5	3,650	1,825	18,980	Equivalent to averaging time for each age group.
Averaging time [exposure duration (70 years) x days (365) per year]	$AT_{lifetime}$	25,550	25,550	25,550	25,550	Standard calculation for lifetime intake.
Bioaccessibility metal in air	BAc_{air}	-	-	-	-	Varies with receptor and metal. Unitless. This is derived from site-specific BAc data and receptor specifc source apportionment of metal in PM_{10}.
Absolute bioavailability of inhaled metal.	ABA_{inhal}	1	1	1	1	Unitless. Taken to be 1[g]

BACKGROUND INTAKE (For Pb)						
Background intake of Pb from soil ingestion (µg/kg/d)	I_{soil_bkgd}	-	-	-	-	Varies with receptor and scenario. This was estimated with Equation 4.1 and all exposure assumptions for the soil ingestion pathway, using the average concentrations of soil Pb by five risk zones as given by Boreland (2010). See Section 2.2.
Background intake of Pb in air by inhalation (µg/kg/d)	I_{air_bkgd}	-	-	-	-	Varies with receptor and scenario. This was estimated with Equation 4.3 and all exposure assumptions for the inhalation pathway using a background air Pb concentration of 0.2 $\mu g/m^3$ in PM_{10} from ENVIRON (2010).
Background dietary intake of Pb (µg/kg/d)	I_{food_bkgd}	1	0.9	0.4	0.4	Intakes at or about the upper end of the dietary intake range reported by FSANZ (2003) for infants, toddlers, 12-year old children and adults.
Background Pb intake from drinking water (µg/kg/d)	I_{water}	0.03	0.02	0.02	0.01	Calculated using a maximum concentration of 0.5 µg Pb/L from the reticulated water supply (Countrywater 2009), assumed body weight and 95th percentile drinking water ingestion rates [i]
BACKGROUND DIETARY INTAKE (for metals other than Pb) [j]						
As (µg/kg/d)	I_{food_bkgd}	1.4	1	0.8	0.8	Intakes calculated using the upper end of the dietary intake range reported by FSANZ (2003) for infants, toddlers, 12-year old children and adults.
Sb (µg/kg/d)		0.25	0.2	0.09	0.08	
Cd (µg/kg/d)		0.7	0.6	0.3	0.2	
Cu (µg/kg/d)		65	40	21	16	
Hg (µg/kg/d)		0.25	0.2	0.1	0.09	
Zn (µg/kg/d)		630	370	250	170	

[a] The soil ingestion values are intended to represent a combination of soil and outdoor dust that has settled or been tracked indoors without distinguishing between the two locations. Note that most studies on soil ingestion have been conducted with children aged 1-7 years. The age group that generally ingests the highest amount of soil is 1-5 year old children due to intimate contact with soil during play. The mean and median intakes from studies are approximately 50 mg soil/day (Davis et al. 1990, Davis and Mirick 2006, Calabrese et al. 1997, and Van Wijnen et al. 1990). Given the uncertainties in the primary data, an upper end soil ingestion intake value of 100 mg/day was used. This value is consistent with 95th percentile estimates for soil intakes in recent studies (Stanek and Calabrese 2000, van Holderbecke et al. 2007) as well as recommendations by authorities in the UK, USA, and the Netherlands (UK EA 2009, US EPA 2008, US EPA 2009a, van Holderbecke et al. 2007). The average and median values for soil ingestion by adults from experimental studies ranged between 10-53 and 1-30 mg/day, respectively (Davis and Mirick 2006, Calabrese et al. 1990, and Stanek et al. 1997). Therefore, the upper end of the

experimental average intake (50 mg/day) was selected for adults; this is also consistent with recommendations from overseas (RIVM 2007, UK EA 2009, US EPA 1997, 2009a).

[b] The body weights in this table correspond to the average body weight of Australian children and adults by age range (ABS 1995).

[c] An average value of 0.5 mg of soil/cm^2 for soil adherence was chosen based on the analysis of various studies (Hawley 1985, Finley et al. 1994, Kissel et al. 1996, Choate et al. 2006, Ferguson et al. 2009). Same as the default in enHealth (2004).

[d] The soil absolute bioavailability factor by the dermal route for inorganic substances of 0.01% is based on the rationale (commonly used to set existing Australian soil health investigation levels for inorganic substances) that inorganic compounds have negligible absorption through intact skin. This is consistent with default bioavailability of 0% for inorganics from soil used by the UK Environment Agency (UK EA 2009, p. 113) and the Dutch National Institute for Public Health and Environment (RIVM 2007, p. 19). For practical purposes, the value of 0.01% is considered negligible.

[e] Inhalation rates for the first three age groups correspond to mean recommended inhalation rates from the US EPA child exposure factors handbook (2008) for the first three age groups (1-<2 year old, 7-<8 year-old, and 16-<18 year old). The adult mean recommended inhalation rate stems from US EPA (2009b).

[f] Note that exposure was calculated as an average daily intake over a lifetime from all exposure routes, starting with the age range of 0.5-2 years. Neonate exposure was not considered as it is expected exposure to receptor site soil will be very low for the first 0.5 years of life.

[g] The inhalational bioavailability is assumed to equal the measured bioaccessibility, as lung absorption is assumed to be 100%.

[h] Wong et al. (2000) reported on two surveys that gathered information from 211 individuals up to the age of 17 years on activity patterns related to dermal contact with soil. During the surveys, the clothing worn during “gardening and yard work” and “outdoor play activities” was recorded. Thus Wong et al. (2000) were able to calculate the area of skin potentially exposed to soil during outdoor activities. This data was included in the US EPA exposure factors handbook for children (2008, Table 7-11). For children <5 years, the percentage of total skin area exposed was 38% and for 5-17 year olds it was 33.8%. For adults information was not provided, but it would be reasonable to assume a similar value as for adolescents. The surface area of exposed skin was calculated by applying these percentages to total skin surface areas for the average age group interval from US EPA (2008 and 2009a).

[i] Calculated with the following equation: [(0.5 µg Pb/L x DW intake (L/day)] / Body weight. Drinking water intakes were assumed to be 1, 1.4, and 2 L/day for 0.5-3, 3-13, and 13-70 year olds, respectively. These values correspond to the average 95th percentile drinking water intakes by each age group sourced from US EPA (2008) (all except the adult drinking water intake). The adults’ drinking water intake of 2 L/day is a reasonable estimate of long term drinking water intake and is consistent with the NHMRC default for the development of drinking water guidelines and the 90th percentile intake for adults determined in overseas studies (1.9-2.4 L/day) (US EPA 1997, 2009a, Ershow and Cantor 1989).

[j] Background intakes were only available for ingestion from diet for a selected number of metals other than Pb. No background intakes were available for Ag, Ba, Be, Cr, Fe, Mn, and Ni.

4.3 Lead Hazard Quotients

Table 4.3.1 summarises total (background plus incremental) 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 (i.e. diet, assumed existing soil lead concentrations, water and air). Of this, intake from diet and existing soil lead each contribute about 50% (Table 4.3.2).

Intake from the mine lease area (existing free areas 80% dust controlled and mine activity related, i.e. Scenario 3) is a minor contributor to overall intake, less than 10% of the TDI for all age groups (Table 4.3.1).

Mean intakes as a percentage of the TDI decreases with increasing age (Table 4.3.1). Calculated lead intake by the infant/toddler is the highest of all the age groups (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.

Table 4.3.1: Total lead intake (background + 'cumulative' incremental intake from mine lease, i.e. Scenario 3) (µg Pb/kg bw/d) [a].

Assumed existing soil Pb concentration (ug/g) and corresponding risk zone [b]		Receptors located within RZ		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 bkgd	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 bkgd	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 bkgd	96	98	98	99
			% total intake from 'cumulative' exposure	4	2	2	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 bkgd	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 bkgd	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] These 'existing' soil concentrations are approximately the upper 95th percentile confidence limit for the respective Broken Hill district amalgamated into risk zones as per Section 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.

Table 4.3.2 summarises the background lead intakes by risk zone and age group. It is evident that the majority of the background intake of lead stems from dietary intake, followed by ingestion of soil.

Similarly, the pie charts in Figure 4.3.1, which depict the incremental intake by pathway for an infant/toddler and an adult for two example receptors, Receptors 3 and 8, show ingestion accounts for around >90% of total intake in the majority of cases.

Figure 4.3.2 shows incremental lead intake for Scenarios 1b ('free areas 80% dust control'), Scenario 2 ('mine activity') and Scenario 3 ('cumulative'), and background intakes (i.e. intake from the assumed existing soil concentrations of lead, Section 2.2, and high end dietary intake) for the highest risk zone, Risk Zone 1) and for the most sensitive receptor (a child) and the TDI for lead at Receptors 3 and 8. It can be seen the incremental intake is low for the mine scenarios and that the assumed background intakes dominate the overall intake.

Figure 4.3.3 shows the risk zones of Broken Hill and the associated receptors' estimated average lead intake as a percentage of the TDI for Scenario 3.

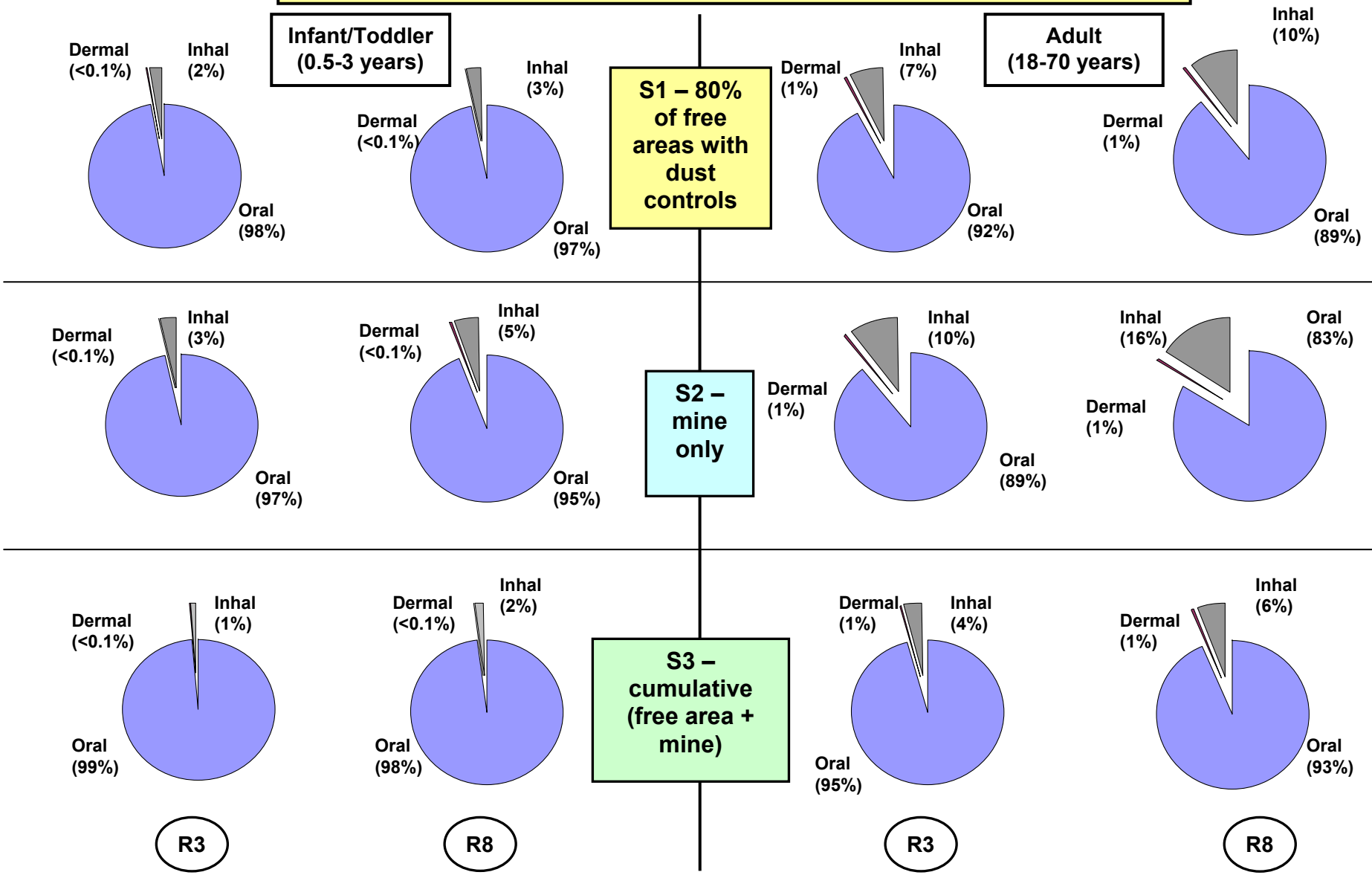
Table 4.3.2: Background lead intake (µg Pb/kg bw/d) [a]

Assumed soil Pb concentration (ug/g) and corresponding risk zone		Receptors located within this RZ		Age (yrs)			
				0.5-3	3-13	13-18	18-70
2000	RZ 1	R1-R4 R8 [b]	**Bkgd intake**	2.0	1.3	0.6	0.5
			% from soil	48	31	24	19
			% from diet	50	67	72	78
			mean bkgd intake as % TDI	**58**	**38**	**16**	**15**
1000	RZ 2	R10, R14 R38-R39	**Mean bkgd intake**	1.5	1.1	0.5	0.5
			% from soil	32	18	13	11
			% from diet	66	79	82	87
			mean bkgd intake as % TDI	**44**	**32**	**14**	**13**
1000	RZ 3	R5-R7 R9, R11 R21-R37 [b] R40-R42	**Mean bkgd intake**	1.5	1.1	0.5	0.5
			% from soil	32	18	13	11
			% from diet	66	79	72	87
			mean bkgd intake as % TDI	**44**	**32**	**14**	**13**
500	RZ 4	R12-R13 R18 [b]	**Mean bkgd intake**	1.3	1.0	0.5	0.4
			% from soil	19	10	7	6
			% from diet	78	87	88	91
			mean bkgd intake as % TDI	**37**	**29**	**13**	**12**
500	RZ 5	R15-R17 R19-R20	**Mean bkgd intake**	1.3	1.0	0.5	0.4
			% from soil	19	10	7	6
			% from diet	78	87	88	91
			mean bkgd intake as % TDI	**37**	**29**	**13**	**12**

[a] Note background exposures are the same for all scenarios and receptor within a particular risk zone but differ by age group due to the differences in physiological and behaviour parameters. Risk zones differ only by the assumed existing (i.e. background) soil concentrations.

[b] 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 4.3.1: Contribution of intake pathway to incremental intake



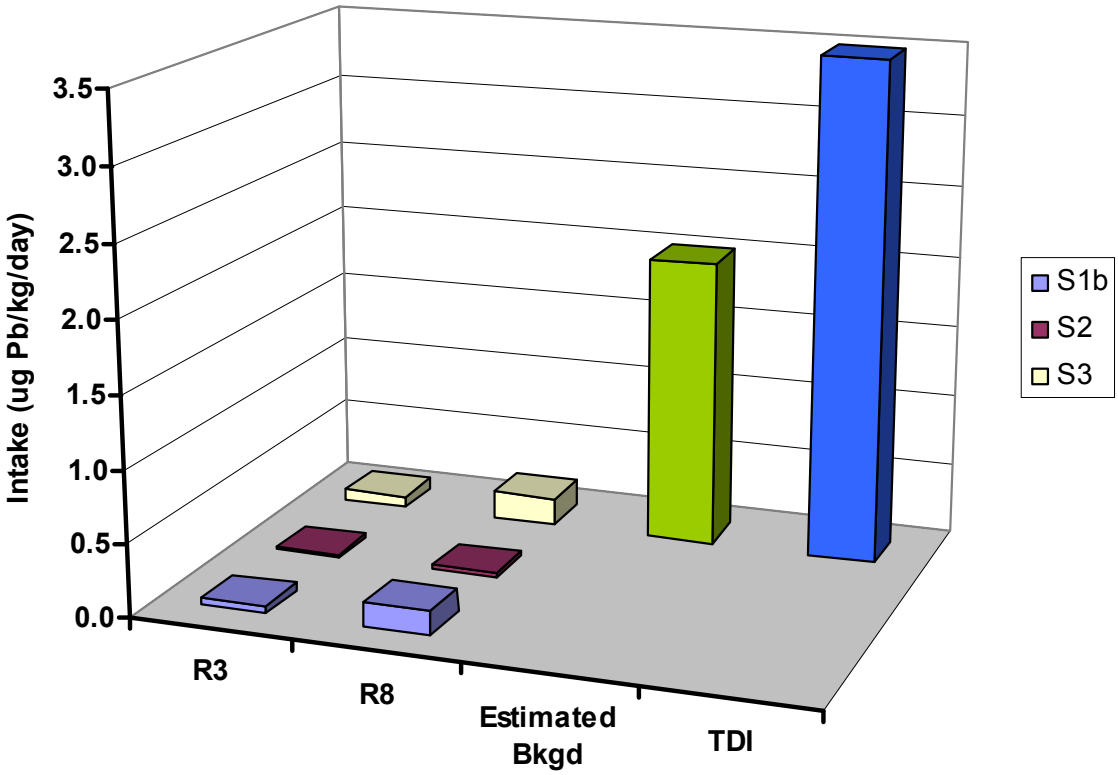


Figure 4.3.2: Incremental Pb intakes (µg/kg bw/d) and assumed background for receptors 3 and 8 in the highest risk zone (RZ1) and most sensitive receptor (child) for Scenarios 1b, 2 and 3.

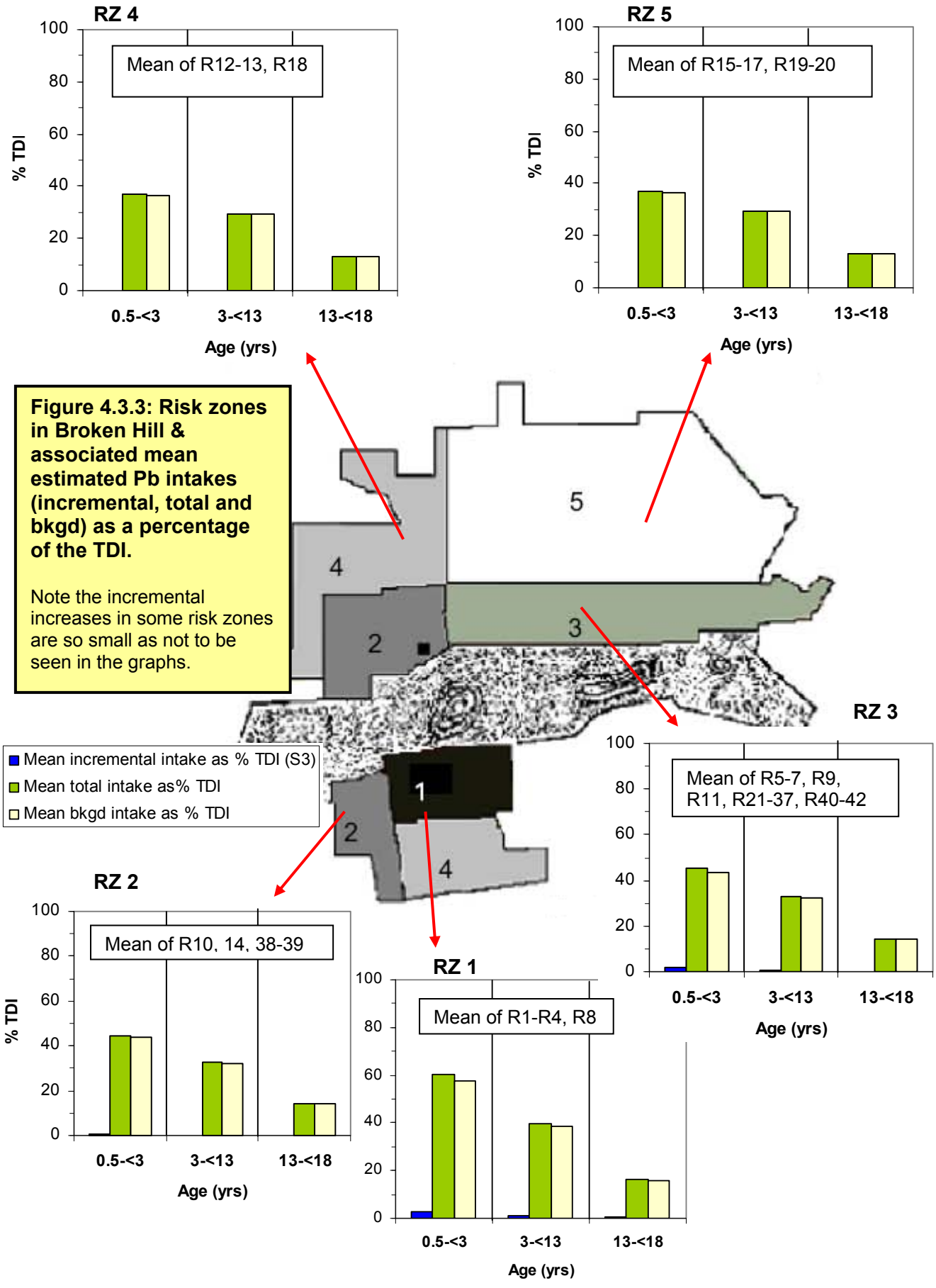


Figure 4.3.3: Risk zones in Broken Hill & associated mean estimated Pb intakes (incremental, total and bkgd) as a percentage of the TDI.

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

4.4 Other Metal Hazard Quotients

The TDI analysis was carried out with thirteen additional metals (other than lead) for which the air dispersion modelling provided data for metal concentration in PM_{10} and deposition to soil.

Because existing soil information and background dietary intakes were not readily available for the majority of the metals (Section 4.2.7, Table 4.2.1) this part of the HRA has taken the form of a screening evaluation in order to determine if the other metals should be regarded as potential contaminants of concern and therefore subject to a more detailed risk assessment; specifically whether the primary scientific literature should be interrogated for information on dietary intakes.

Hazard quotients were calculated for Scenarios S1b, S2 and S3 (i.e. exposure to dust from free areas 80% controlled, dust associated with mine activities only and the combination of the two) (Table 4.4.1). This was done by comparing the incremental intakes (calculated as the sum of intakes from Equations 4.1 – 4.3) with the respective TDI for each metal (the TDI's and a brief description of their derivation is in Appendix 1, Table A1.1.5).

In Table 4.4.1 the hazard quotients for the most sensitive life stage (0.5 – 3 year old, see Table 4.3.1) at Receptor 8 are presented (other life stages hazard quotients are in Appendix 4, Section A4.2.6). It can be seen the hazard quotients for all the metals resulting from receptor exposure to mine site derived dust (i.e. S3, free area dust 80% controlled plus that from mine activity) are very low. The highest hazard quotients (other than lead) are for Mn, Cr and Fe, these being 0.008, 0.007, and 0.007, respectively. That is, the estimated intake is just 0.7-0.8% of the respective TDI for these metals.

Since the incremental hazard quotients of the thirteen individual metals are all less than 0.01 (less than 1% of the TDI) and the incremental hazard index (the sum of hazard quotients) for metals other than lead is only 0.05, it is concluded the incremental increase in exposure to metals other than lead is trivial and is unlikely to materially change current background exposures to these metals. Consequently metals other than lead are not regarded as contaminants of concern and do not require further detailed investigation.

The above considers 'other metals' for the most sensitive life stage. In Section 4.5 the influence of the other thirteen metals are evaluated in conjunction with lead for an assumed lifetime exposure.

Table 4.4.1: Hazard quotients for individual incremental intakes (µg/kg/d) of metals other than lead for a child when compared to TDI at Receptor 8.

Metal	Scenario		
	S1b	S2	S3
As	0.003	0.003	0.003
Ag	0.00002	0.00002	0.00002
Ba	0.0003	0.0003	0.0003
Be	0.003	0.003	0.003
Cd	0.006	0.006	0.006
Cr	0.007	0.002	0.007
Cu	0.001	0.0003	0.001
Fe	0.005	0.002	0.007
Hg	0.004	0.003	0.004
Mn	0.008	0.0001	0.008
Ni	0.001	0.0005	0.001
Sb	0.002	0.001	0.003
Zn	0.002	0.004	0.006
Incremental Hazard Index			**0.05**

4.5 Hazard Indices

The hazard index, 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 quotient is less than unity it indicates that under the exposure scenario being considered there is little likelihood of interactive health effects between the substances. This process does not take into account toxicological mechanisms or kinetics.

However, in strict biological terms an interaction, additivity or synergy, would only be expected if chemicals were affecting the same tissue types in an equivalent manner. However for the purposes of the present risk assessment all the metals included in the hazard index are, by default, conservatively assumed to act in an additive manner. Therefore it is important to consider the level of conservatism underlying this assumption. Table 4.5.1 summarises the basis for the toxicity reference values (yellow shaded cells) used in the present risk assessment together with other tissue effects for each chemical of interest (denoted by a tick mark). More detailed information on the health effects of individual chemicals is provided within the toxicity profile. However the exposures or doses that may be associated with tissue effects other than

that upon which the TDI was established are not included as this was beyond the resources of the HHRA.

From Table 4.5.1 it is apparent that Pb, As, Cd and Hg may all affect the kidney, this organ therefore represents the most likely tissue to reflect any long term exposure interactions amongst the metals. Exposure to Pb and As are both associated with hypertension. An assessment of the mode of action of the individual metals on the kidney was not undertaken hence it is uncertain whether such interaction would occur at exposure levels relevant for this risk assessment. Pb and Hg can cause central nervous system toxicity manifested as behavioural changes. Pb and Cd are able to cause changes to bone structure.

The US Department of Health (ATSDR 2004, 2006) have reviewed the health effects and toxicity of different mixtures of metal compounds (i.e. Pb and As; Pb and Hg; Pb, As & Cd) to investigate whether their interactions are less than, equal to or greater than additive. Most of the available information is for binary mixtures (ATSDR 2004, 2006). The reviews did not identify clear evidence of other than additive potential for combinations of either Pb, As and Cd nor between Pb and Hg (ATSDR 2004, ATSDR 2006).

The TDI for each metal has been set against the most sensitive toxicological end point and is intended to be a life time daily intake that does not cause adverse effects. If the hazard index, calculated using estimated average daily lifetime intake against the TDI, is less than unity it signifies there is no significant interaction between the metals that gives rise to health concerns. Under these circumstances it is very unlikely there will be interaction for other health effects that will cause concern.

Hazard indices were calculated by summing the incremental hazard quotients for all 14 metals evaluated in the risk assessment. The hazard quotients for the metals did not include background intake.

Table 4.5.1: Summary [a] of potential target organs of concern

Systemic Effects	Pb	Zn	As	Cd	Hg
Neurological	✓ (CNS - behavioural)		✓ (peripheral neuropathy)		✓ [d] (CNS – behavioural)
Haematological	✓ (heme synthesis)	✓ (copper deficiency)[b]	✓ (vascular disease)		
Cardiovascular	✓ (hypertension)		✓ (hypertension)		
Renal	✓ (function)		✓	✓	✓
Testicular	✓ (sperm prod.)				
Dermal lesions			✓		
Respiratory [c]		✓ (metal fume fever)	✓	✓	
Cancer			✓ (skin, urinary bladder, and lung)	✓ (lung)	
Hepatic					
Immune function	✓ (function)				
Bone	✓			✓	
Developmental	✓ (reduced birth weight, neurological)				✓

[a] References used to determine health effects are summarised within the toxicity profile for each compound. ATSDR (2004), ATSDR

[b.] requires high oral doses, refer to zinc toxicity profile (Appendix 1).

[c.] following inhalation exposure

[d] Inhalation toxicity reference values for elemental Hg are based on CNS effects (tremors).

Figure 4.5.1 shows the combined incremental metal hazard indices (average lifetime exposure calculated as in Section 4.2.6) at selected receptors for Scenarios 1b, 2, and 3 (i.e. for receptor

exposures arising from dust from the free areas (80% controlled), dust from mine activities, and the sum of both).

The resulting incremental HI's are all markedly less than unity.

The majority of the hazard index is attributable to lead, as shown by Figure 4.5.2.

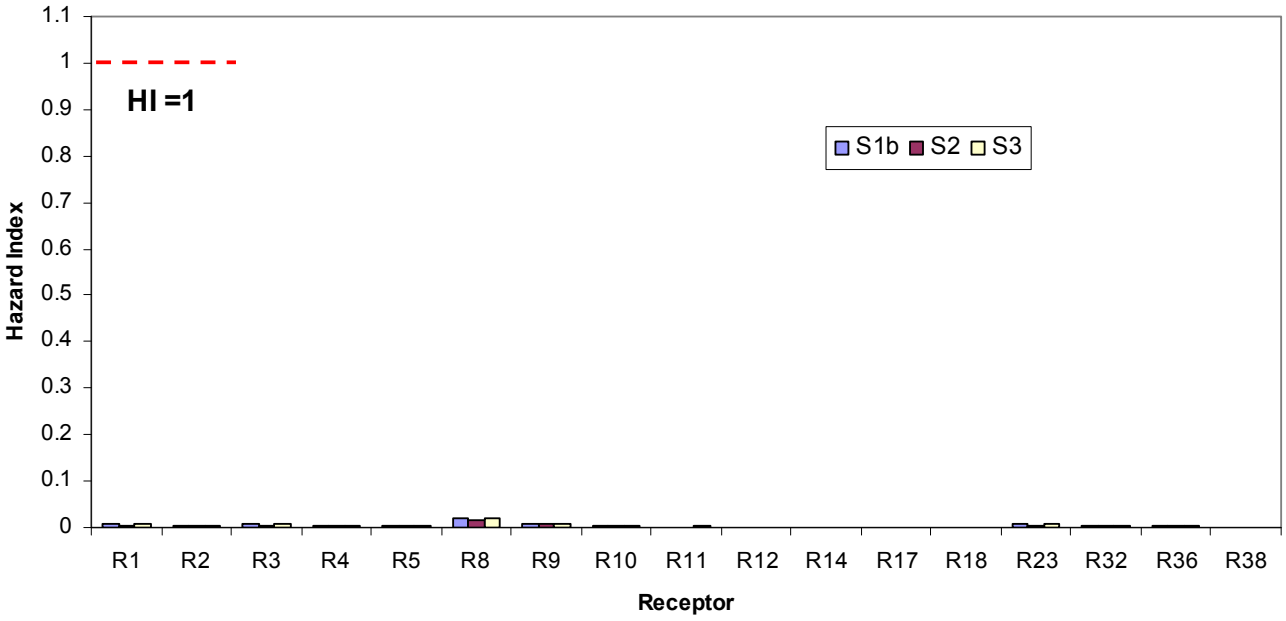


Figure 4.5.1: Combined incremental metal hazard indices at selected receptors.
These incremental hazard indices are calculated for an assumed lifetime exposure. Background concentrations of metals were not included in the overall hazard index. Pb contributes about 40% to the incremental HI (Figure 4.5.2).

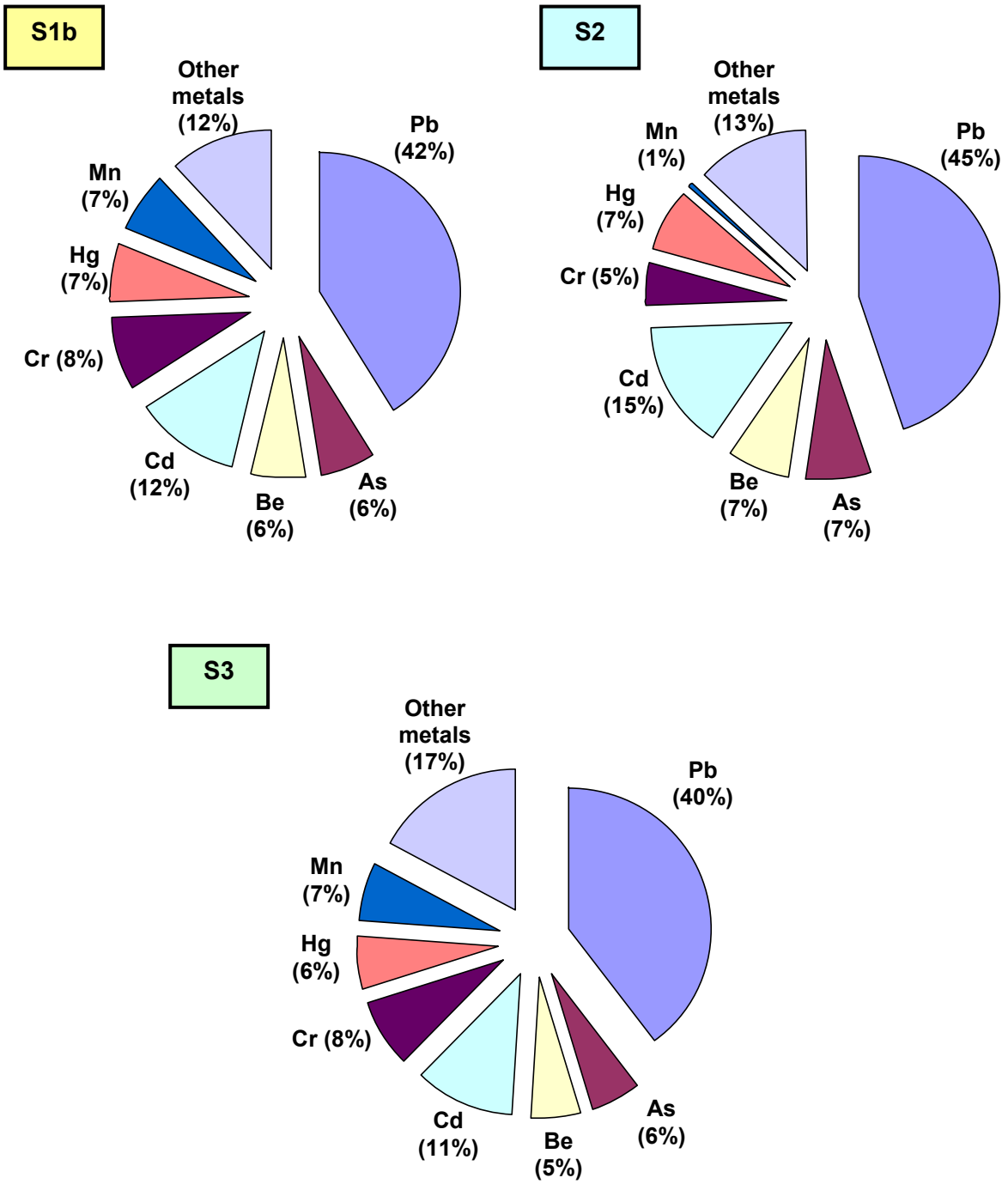


Figure 4.5.2: Average percentage contribution of Pb and other metals to the incremental hazard index for the three scenarios for the selected receptors in Figure 4.5.1.

Note this incremental hazard index is the average of the HIs for the selected receptors calculated for an assumed lifetime exposure. The HI is very small, for example at the most affected receptor (R8) the HI = 0.02 for Scenario 3 (Appendix 4, Section A4.2.5).

4.6 Discussion and conclusions

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 influenced 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 at 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 (0.5 – 3 years), 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 (i.e. background soil lead concentrations) 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 50% 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 HRA the incremental hazard quotients were summed to give a cumulative exposure hazard index. This was only 0.1 for 0.5 – 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 from dust emissions from the Rasp mine site,
- that since conservative high end 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 mine are unlikely to result in health effects for the surrounding community.

5. Risk characterization – predicted blood lead

5.1 Introduction

5.1.1 Background

As described in Section 3 the dose - response of lead is described in terms of the effects that occur at given blood lead concentrations. Indeed throughout the world the health effects of lead, both for the public and workers in lead related industries are managed by maintaining blood lead concentrations below critical levels. In Australia the NHMRC (2009) recommend all Australians should have a blood lead level below 10 µg/dL. The NHMRC note that based on research evidence on the effects of low-level exposure to lead, it is not possible to make a definitive statement on what constitutes a 'safe level' or 'level of concern' for blood lead concentrations. This arises because the tools of psychologists and psychiatrists used to investigate subtle impacts on intellectual performance and development are not precise and the outcomes are influenced by such things as genetics, socio-economic status and early life experience/environment.

From 1993 to 2005, the NHMRC has recommended an overall goal of all Australians having a blood lead concentration below 10 µg/dL, together with a series of graduated responses depending on the proportion of children in a community who exceed blood lead concentrations of 15 and 25 µg/dL. As noted above the latest recommendation from the NHMRC is that *all* Australians should have blood lead levels below 10 µg/dL.

Thus, given the established dose-response for lead based on blood concentrations and the management goal of the NHMRC for public health in relation to lead, prediction of blood lead concentrations associated with a given level of environmental lead exposure is an important method in being able to interpret potential community impacts from the proposed Rasp mine. Furthermore it is important that such predictions are made for the most susceptible sub-population, i.e. children.

A number of lead PBPK models have been developed that are able to predict blood lead concentrations, e.g. O'Flaherty 1993, 1995 and Leggett 1993, IEUBK. However only the

Integrated Uptake Exposure Biokinetic (IUEBK) model developed by the US EPA is publically available in a form amenable for use. Consequently this is the predictive model used in this HRA; apart from being readily available and validated, the IEUBK model has the advantage of being regularly maintained and updated, having an extensive user manual, being 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).

A description of the IUEBK PBPK model for lead is given in Section 5.2. Figure 5.1.1 provides an overview of the methodology followed to generate predictive blood lead modelling in relation to lead exposure arising from the proposed Rasp mine. Briefly, 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), an Excel spread sheet made available by the US EPA as a companion to IEUBK. Detailed model descriptions, input parameters, and outputs are respectively provided in Section 5.2, Appendix 5 and Appendix 6.

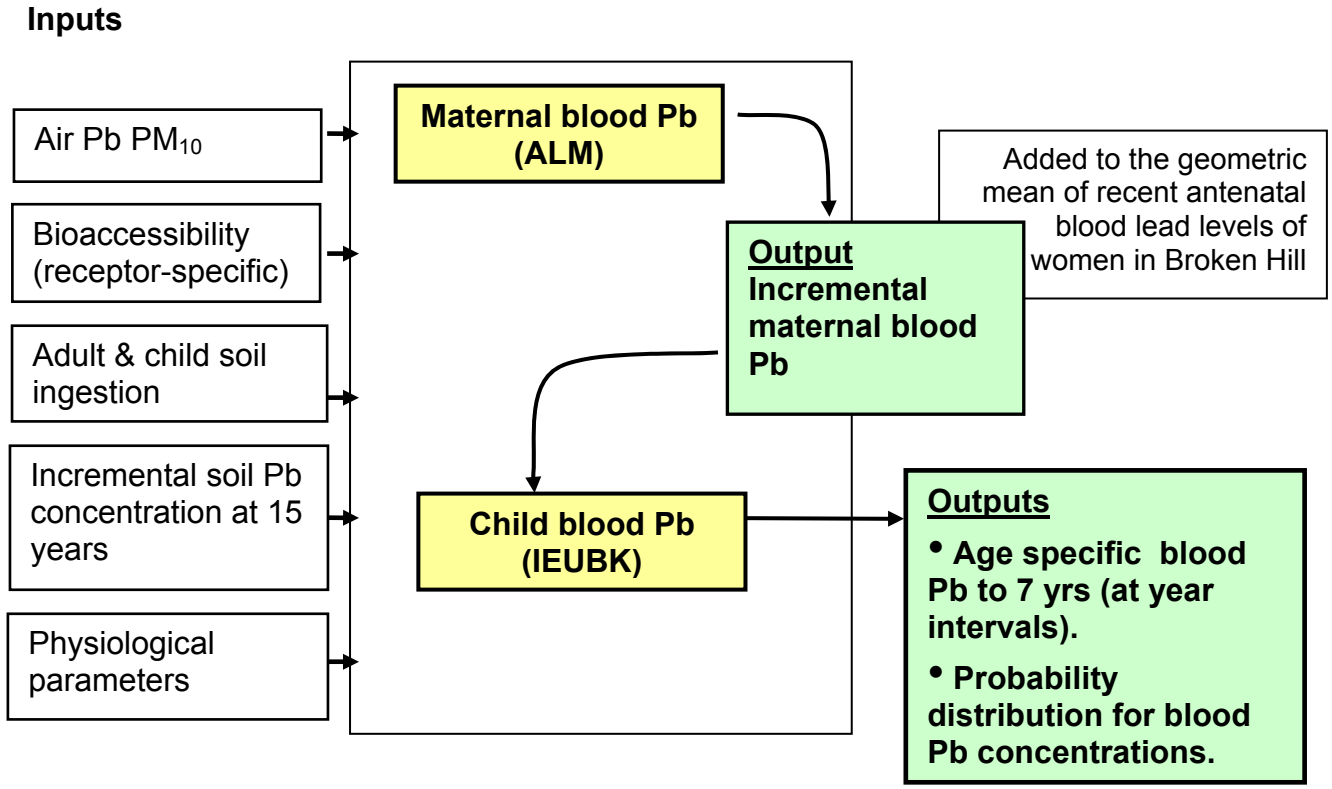


Figure 5.1.1: Overview of Methodology for Blood Pb predictions

5.1.2 Background blood lead levels

The Greater Western Areas Health Service (GWAHS 2009) have published information on blood lead levels for people in Broken Hill, this included 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, via personal correspondence, with additional summary statistics which included the minimum, maximum, 95th percentile, and standard deviation of the 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/L (Figure 5.1.2).

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 5.1.3). 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.

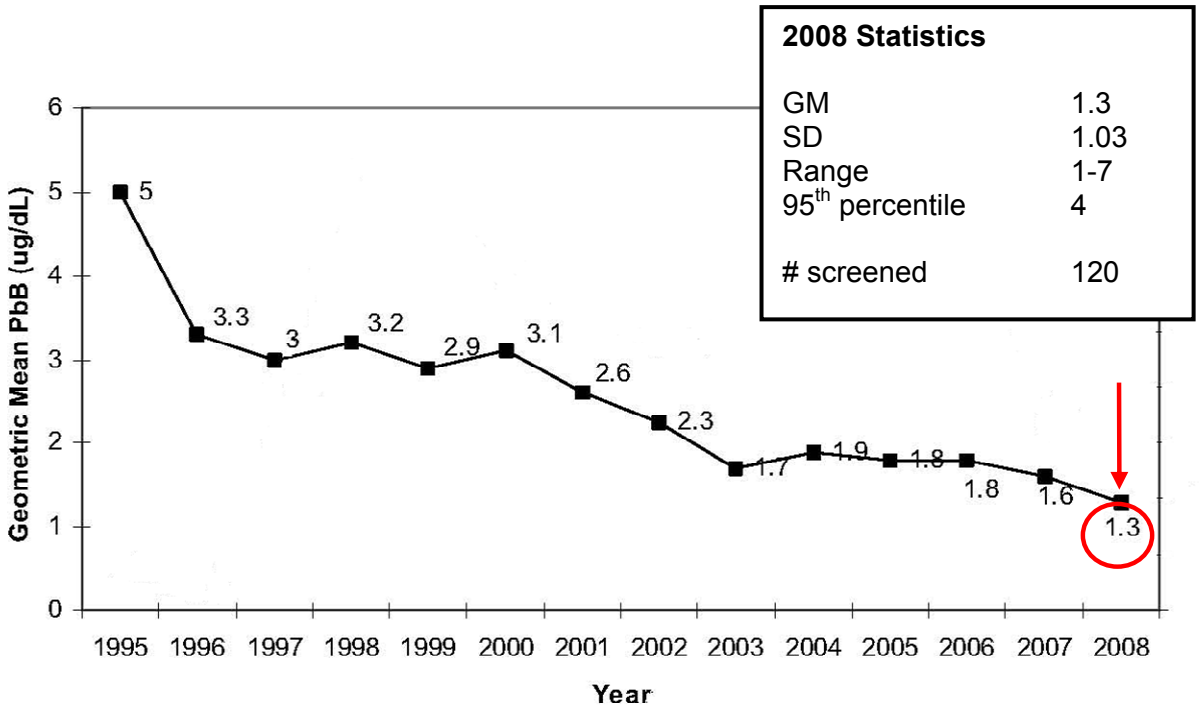


Figure 5.1.2: Geometric mean blood Pb concentrations (µg/dL) of antenatal women in Broken Hill measured from 1995-2008 (GWAHS 2009 and Lesjak 2010).

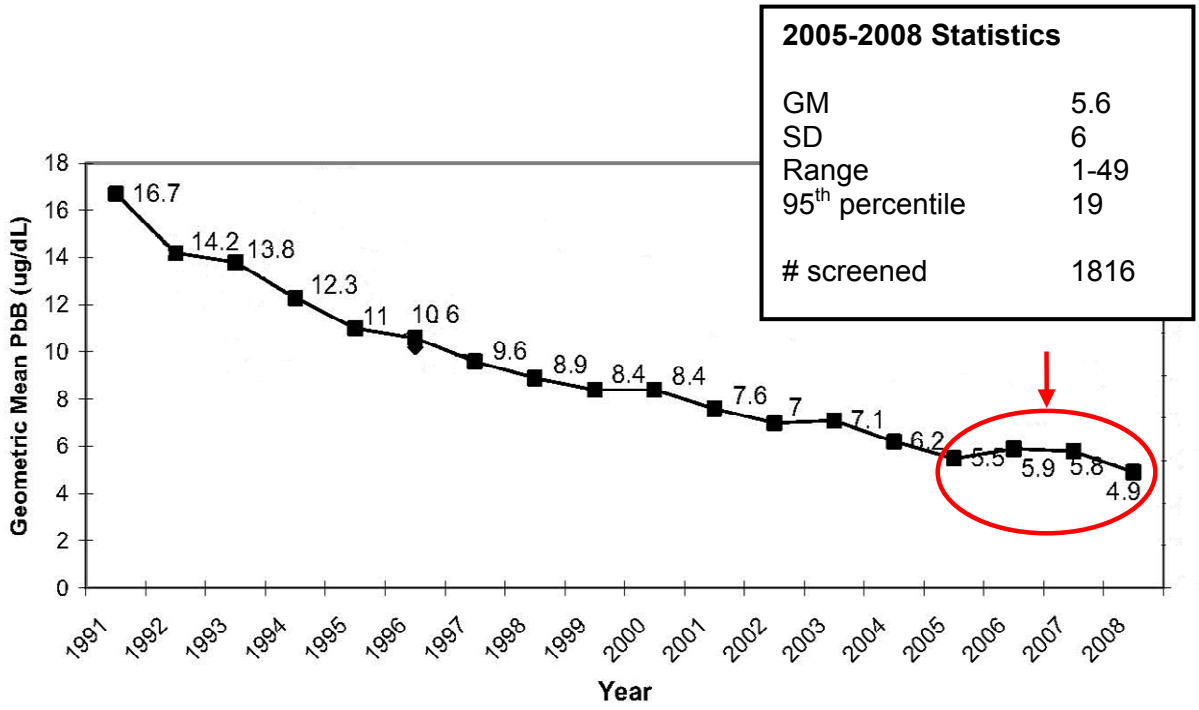


Figure 5.1.3: Geometric mean blood Pb concentrations (µg/dL) of children aged 1-4 years in Broken Hill measured from 1995-2008 (GWAHS 2009 and Lesjak 2010).

5.2 Lead PBPK Model Description

5.2.1 IEUBK model

5.2.1.1 Introduction

The Integrated Exposure Uptake Biokinetic (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 6 months to 7 years old.

The model was first created by the US EPA in 1994 as an 'MS dos' version, this was converted to Windows in 2002, and has since been updated a few times. The latest Windows version of the model available for download on the US EPA is the IEUBKwin v 1.1 build 9, 32-bit version, which became available in June 2009. The US EPA website has the original detailed guidance document for the 1994 'dos' version for download (US EPA 1994a), as well as a reference manual describing the changes made in the model upon conversion from 'dos' to Windows operating systems (US EPA 2002), and an updated user's guide for the IEUBK model (US EPA 2007a). An additional document also exists giving an overview of the changes that were made

from the previous Windows version of the IEUBK model (version 1 build 264) to the current version (version 1.1) (US EPA 2009c).

The four main components of the IEUBK model are:

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

The IEUBKwin 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[12]. 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 GSD value is a measure of the relative variability in blood lead concentrations of a child of a specified age, or children from a hypothetical population whose lead exposures are known (US EPA 2007a). The GSD in IEUBKwin covers biological and behavioral differences, measurement variability from repeat sampling, variability as a result of sample locations, as well as analytical variability. The recommended default is 1.6, and was derived from empirical studies with young children where both blood and environmental lead concentrations were measured (US EPA 2007a).

The IEUBKwin model can be used for a variety of applications:

A: One location

A1: one living unit, one child;

A2: one living unit, more than one child;

[12] The IEUBKwin model calculates the blood lead of children aged 6-84 months from integrated exposure to lead. This means that for a 5 year-old child, for example, the model assumes the child has been exposed to the concentrations of environmental lead since birth. This is evident by the examples provided by the US EPA (1994a, Chapter 5) for the applicability of the model. In these examples, the model can be used to calculate the blood lead concentration for a child or children where environmental lead concentrations change over time. This has also been confirmed through personal communication with the IEUBK electronic helpdesk (Follansbee 2009).

A3: more than one living unit, more than one child, homogenous media concentrations;
B: Multiple locations, one neighborhood, homogenous media concentrations;
C: Multiple locations, one neighborhood, heterogeneous media concentrations;
D: Multiple locations, more than one neighborhood, heterogeneous media concentrations.

A single run of the model is enough for categories A and B, but for categories C and D, a classification or disaggregation of the neighborhood into distinct exposure groups is required. Neighborhood-scale or community-scale risk estimation requires aggregating the risk estimates for individuals or subgroups (US EPA 2007a).

The IEUBK model is not intended to be used in a worst-case scenario, but rather provides a best estimate of the geometric mean blood lead for the exposures entered into the model; while these are intended to reflect the actual environmental exposure determined by measurement, worst case or high end exposure estimates can be used as is the case in the application of the model for this HRA. The model does not inherently apply any uncertainty or modifying factors in making risk estimates. If there is some uncertainty about model parameters, these can be evaluated using sensitivity analyses (US EPA 1994a). This has been undertaken in Section 7.3 for the ratio of indoor dust lead to outdoor soil lead, the effects on blood lead associated with changing soil mixing depth and incorporating a loss factor for lead in soil.

The biological structure of the IEUBK model is shown pictorially in Figure 5.2.1.

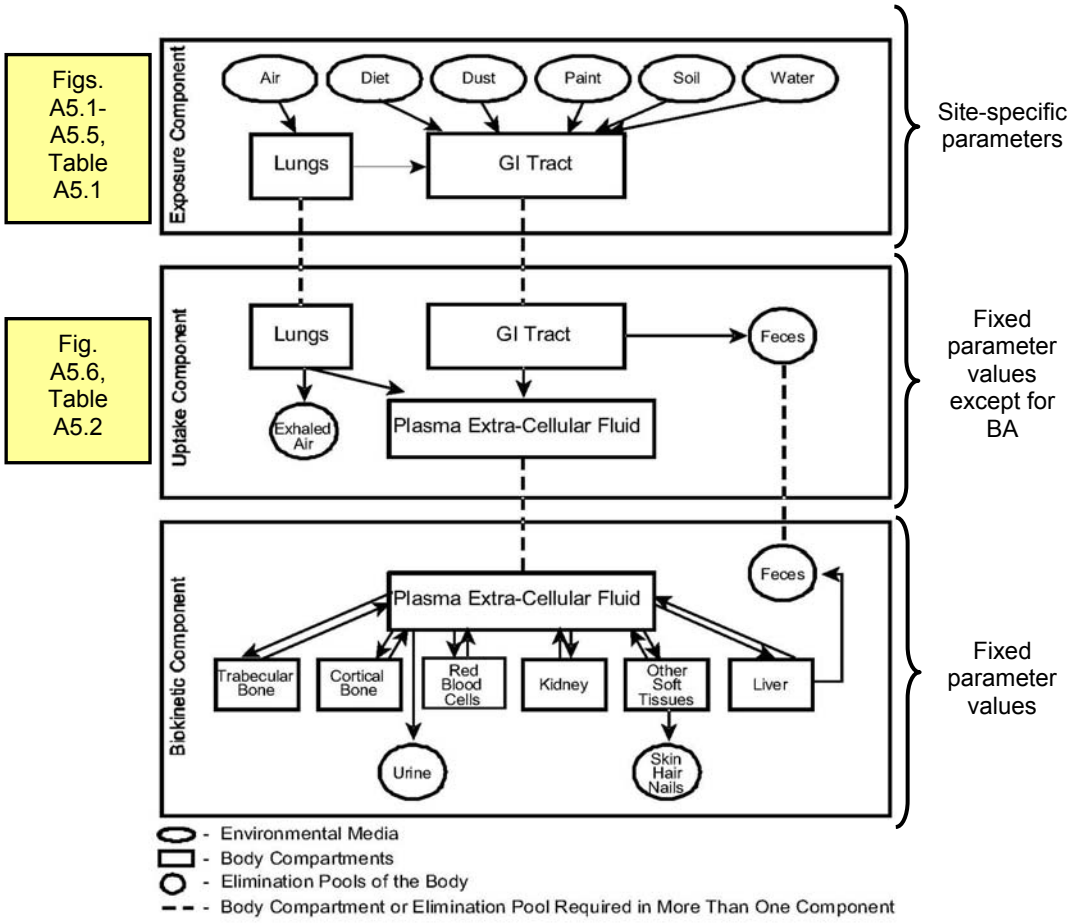


Figure 5.2.1: Biological structure of IEUBKwin model (US EPA 2007a, pg. 4)

5.2.1.2. Default parameters

The model inserts default values whenever site-specific information is not used. The default values (e.g. dietary lead concentrations and consumption values) are broad-based estimates of the expected lead environment of a child; they are not necessarily appropriate for every site. They were reviewed and modified if site/scenario or Australian specific information was available (Appendix 5 and Section 5.3). The model uses standard age-weighted exposure parameters for consumption of food, drinking water, soil and dust, and inhalation of air, together with site-specific concentrations of lead found in these media in order to estimate exposure to a child. The model simulations assume concentrations of lead in environmental media are at steady state for at least a year. Blood lead levels are provided in yearly age increments, the level for each year being integrated with previous yearly exposures.

The model contains more than 100 input parameters that are initially set to default values (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.

For example, ingestion parameters are based on surveys of drinking water and tap water consumption (Ershow and Cantor 1989), estimates of dietary intake (Pennington 1983; Gartrell 1986), and on soil and dust ingestion studies for children in the US (Binder et al. 1986; Calabrese et al. 1989, 1991; Davis et al. 1990; van Wijnen et al. 1990).

The default values for the absorption component (bioavailability values) are based on *in vivo* data in infant and juvenile baboons, as well as human infants whose intake of lead has been observed (Mallon 1983; Sherlock and Quinn 1986). The model accounts for situations where the absorption may be non-saturable or saturable. In the non-saturable circumstance, absorption is a constant fraction of total ingested lead, whereas the saturable mode follows the Michaelis-Menten kinetics for saturable absorption proposed by Aungst and Fung (1981). The algorithm was also developed based on data from lead balance and feeding studies in human infants and children (Alexander et al. 1974; Alexander 1974; Ryu et al. 1983; Ziegler et al. 1978).

The compartmental structure is based on distribution of lead in adults, but was verified and extended using data from studies in infant and juvenile baboons (Mallon 1983) (aged 5-26 months, 2.5-6 kg). Ratios of lead concentrations in tissues of human children from autopsies (Barry 1981) were used to adjust the baboon's biokinetics distribution parameters (Harley and Kneip 1985).

In total, there are five data input windows for each of the common sources of lead exposure, an additional input window for consideration of alternative sources of lead and a window to specify bioavailabity for each of those sources. The parameters can be changed in each window to reflect site-specific or exposure scenario conditions (US EPA 2007a). The windows are:

1. Air Data
2. Dietary Data
3. Drinking Water Data
4. Soil/Dust Data

5. Maternal Data[13]
6. Alternative Source Data
7. Gastro intestinal values/Bioavailability

If an alternate source of lead is considered, the total percent accessible for absorption needs to be changed in the GI/bioavailability input window. An alternate source, for example, would be intake of lead-based paint (in addition to fraction of lead-based paint in household dust, which is considered in the soil/dust portion of the model). US EPA (2007a) states that building an exposure scenario using alternate source data should be done with care. Because no site-specific data for alternate sources of lead in Broken Hill was available, IEUBK modeling was performed with the assumption that alternate intake of lead equals zero. Table 5.2.1 gives a list of the modifiable parameters in IEUBKwin v1.1, with the default values applied in the model, and a brief description of where the default value has come from. Table 5.2.2 gives a list of those non-modifiable parameters in the IEUBKwin v1.1. model, for which a distinct value exists. The parameters that were modified, together with the rationale for modification are described in Section 5.3.

[13] Note that the IEUBK model calculates blood lead and tissue lead burdens for ages from 0-84 months in yearly increments. However, the blood lead concentrations in children less than 6 months of age will be affected by pre-natal lead exposure and are not likely to show much influence from exposure to soil, dust and paint (US EPA 1994a). Therefore, the results of the model simulations in the text file are not reported for children younger than 6 months of age. However, the user can specify whether or not he wants to display the blood lead results for children younger than 6 months in the graphical distribution curves.

Table 5.2.1: Modifiable default parameters for IEUBKwin v1.1 (US EPA 2007a, 1994a, 1994b)

(Section 5.3 describes the parameters modified by Toxikos to reflect site specificity for the Rasp mine. Appendix 5 (Table A5.1) contains all parameters used in the IEUBK predictions for blood lead).

Parameter		Default Value	Units	Origin/ Description	Reference/ Source
Indoor air Pb concentration (% of outdoor)		30	%	Based on homes near Pb point sources.	Reported in US EPA 1989a, estimated by Cohen & Cohen 1980
AIR (by year)					
Air Pb concentration (outdoor)		0.1	µg/m^3	Default based on lower end of range 0.1-0.3 µg Pb/m^3 reported in US cities without Pb point sources.	US EPA 1989a
Ventilation rate			m^3/day	These estimates are based on body size in combination with smoothed data.	US EPA 1989a
Age	0-1 year (0-11 mo)	2			
	1-2 years (12-23 mo)	3			
	2-3 years (24-35 mo)	5			
	3-4 years (36-47 mo)	5			
	4-5 years (48-59 mo)	5			
	5-6 years (60-71 mo)	7			
	6-7 years (72-84 mo)	7			
Time spent outdoors			h/day	Roughly the middle of the ranges of values established by the US EPA for time spent outdoors.	US EPA 1989a
Age	0-1 year (0-11 mo)	1			
	1-2 years (12-23 mo)	2			
	2-3 years (24-35 mo)	3			
	3-7 years (36-84 mo)	4			
Lung absorption		32	%	US EPA established 25-45% absorption for young children living in non-point source areas & 42% for those living near point sources. Intermediate value of 32% was chosen.	US EPA 1989a
DATA ENTRY FOR DIET (by year)					
Dietary Pb intake			µgPb/day	FDA food monitoring data collected in 1995-2003	US FDA 2006
Age	0-1 year (0-11 mo)	2.26			
	1-2 years (12-23 mo)	1.96			
	2-3 years (24-35 mo)	2.13			
	3-4 years (36-47 mo)	2.04			
	4-5 years (48-59 mo)	1.95			
	5-6 years (60-71 mo)	2.05			
	6-7 years (72-84 mo)	2.22			
DATA ENTRY FOR ALTERNATE DIET SOURCE (by food class)					
Concentration		0	µgPb/day		
	home-grown fruits				
	home-grown vegetables				
	recreationally-caught fish				
	hunted game animals				
Percent of food class		0	%		
	home-grown fruits				

Parameter		Default Value	Units	Origin/ Description	Reference/ Source
	home-grown vegetables	0			
	recreationally-caught fish				
	hunted game animals				
DATA ENTRY FOR DRINKING WATER					
Drinking water Pb concentration		4	µg/L	Based on analysis of data from the American Water Works Service Company	Marcus 1989
Drinking water ingestion rate			L/day	From US EFH, from a survey of drinking water consumption in US children.	US EPA 1989c; original data from Ershow & Cantor 1989
Age	0-1 year (0-11 mo)	0.20			
	1-2 years (12-23 mo)	0.50			
	2-3 years (24-35 mo)	0.52			
	3-4 years (36-47 mo)	0.53			
	4-5 years (48-59 mo)	0.55			
	5-6 years (60-71 mo)	0.58			
	6-7 years (72-84 mo)	0.59			
DATA ENTRY FOR ALTERNATE DRINKING WATER SOURCES					
Pb Concentration			µg/L	First-draw water & flushed: Marcus 1989. Flushed: Fountain: Default assumption is that fountain has Pb-lined reservoir, but consumption is not always first draw. Value from 5-25µg/L was selected.	Marcus 1989
	first-draw water	4			
	flushed water	1			
	fountain water	10			
Percentage of total intake			%	First-draw: In absence of data, conservative value corresponding to consumption largely after four hours stagnation time was used. Fountain: Default value based on 4-6 trips to water fountain at 40-50 mL per trip.	
	first-draw water	50			
	flushed water	100 – first draw & fountain			
	fountain water	15			
DATA ENTRY FOR SOIL/DUST (constant over time)					
Concentration (starting values to be modified using appropriate site data)			µg/g	Air quality criteria document for lead	US EPA 1986
	soil	200			
	dust	200			
Soil/dust ingestion weighting factor (% soil) or percentage of total soil & dust ingestion that is soil		45	%	Best judgment of properly weighted ratio.	US EPA 1994a

Parameter		Default Value	Units	Origin/ Description	Reference/ Source
DATA ENTRY FOR SOIL/DUST INGESTION (by year)					
Soil/Dust ingestion			g/day	Reported in OAQPS report. Estimated for children 12-48 mo, by several authors. Sedman (1987) extrapolated these estimates for those children, ages 0-84 mo.	US EPA 1989a
Age	0-1 year (0-11 mo)	0.085			
	1-2 years (12-23 mo)	0.135			
	2-3 years (24-35 mo)	0.135			
	3-4 years (36-47 mo)	0.135			
	4-5 years (48-59 mo)	0.100			
	5-6 years (60-71 mo)	0.090			
	6-7 years (72-84 mo)	0.085			
DATA ENTRY FOR SOIL/DUST MULTIPLE SOURCE ANALYSIS (constant over time)					
Fraction of indoor dust Pb attributable to soil (MSD)		0.7	unitless	Represents real sites where soil is a major contributor to household dust (1983 East Helena study)	US EPA 1989a
Ratio of indoor dust Pb concentration to outdoor air Pb concentration		100	µg Pb/g dust per µg/m^3 air	Analyses of 1983 East Helena study suggest about 267 µg/g increment of Pb in dust for each µg/m^3 Pb in air. A much smaller factor is assumed for non-smelter community exposure.	US EPA 1989
DATA ENTRY FOR SOIL/DUST MULTIPLE SOURCE ANALYSIS WITH ALTERNATIVE HOUSEHOLD DUST LEAD SOURCES (constant over time)					
Concentration (starting values to be modified using appropriate site data)			µg/g	Daycare, school & second home dust concentrations are assumed to be the same as default residence dust concentrations. Others are from the air quality criteria document for lead.	US EPA 1986
	household dust (calculated value)	150			
	secondary occupational dust	1,200			
	school dust	200			
	daycare centre dust	200			
	second home	200			
	interior lead-based paint	1,200			
Percentage			%		
	household dust (calculated value)	100 minus all other			
	secondary occupational dust	0			
	school dust	0			
	daycare centre dust	0			
	second home	0			
	interior lead-based paint	0			

BIOAVAILABILITY DATA ENTRY FOR ALL GUT ABSORPTION PATHWAYS					
Total Pb absorption (at low intake)			%	The default in the model assumes there is no alternate source of Pb ingestion, which is why alternate source Pb absorption is assumed to be zero.	US EPA 1989a
	diet	50			
	drinking water	50			
	soil	30			
	dust	30			
	alternate source	0			
Fraction of total net absorption at low intake rate that is attributable to non saturable (passive) processes		0.2	unitless	This is assumed to be partitioned into nonsaturable (6%) and saturable (24%) components. Based on *in vitro* everted rat intestine data, reanalyses, or infant baboon data, as well as infant duplicate diet study.	Aungst & Fung 1981; Marcus 1994; Mallon 1983; Sherlock & Quinn 1986
DATA ENTRY FOR ALTERNATE SOURCES (by year)					
Total Pb intake			µg/day		
Age	0-1 year (0-11 mo)	0			
	1-2 years (12-23 mo)	0			
	2-3 years (24-35 mo)	0			
	3-4 years (36-47 mo)	0			
Age	4-5 years (48-59 mo)	0			
	5-6 years (60-71 mo)	0			
	6-7 years (72-84 mo)	0			
DATA ENTRY MENU FOR MATERNAL-TO-NEWBORN LEAD EXPOSURE					
Mothers blood lead concentration at childbirth		1	µg/dL		
DATA ENTRY MENU FOR PLOTTING AND RISK ESTIMATION					
Geometric standard deviation (GSD) for blood Pb		1.6	unitless		
Blood Pb level of concern, cutoff		10	µg/dL		
COMPUTATION OPTIONS					
Iteration time step for numerical integration		4	h	This is a technical matter, which does not normally have to be changed from the default. The time step is used in the biokinetics component of the model in combination with compartmental transfer times to calculate bodily tissue lead distribution.	

Table 5.2.2: Non-modifiable default parameters for IEUBKwin (1994b)[a]

Parameter	Default Value	Units	Origin/ Description	Reference/ Source
BIOKINETIC COMPONENT				
Max Pb concentration capacity of red blood cells	1200	µg/dL	Based on Marcus (1983) reanalysis of infant baboon data from Mallon (1983).	
Hematocrit at birth	0.45	Decimal percent		Silve et al. 1987; Spector 1956; Altman & Dittmer 1973
Ratio of Pb mass in blood to Pb mass in plasma-ECF	100	unitless	Based on lower end of 50-500 range for red cell/plasma Pb concentration ratio recommended.	Diamond & O'Flaherty 1992
Ratio of endogenous fecal Pb elimination rate to urinary Pb elimination rate	0.75	unitless	Assume child ratio is larger than adult ratio; values derived from reanalysis of data from Ziegler et al. (1978) and Rabinowitz & Wetherill (1973).	Ziegler et al. 1978; Rabinowitz & Wetherill 1973.
Ratio of elimination rate via soft tissues to endogenous fecal Pb elimination rate	0.75	unitless	Within range of values derived from reanalysis of data from Ziegler et al. (1978) and Rabinowitz & Wetherill (1973).	Ziegler et al. 1978; Rabinowitz & Wetherill 1973.
Pb transfer time from blood to bone (24 months – other are calculated)	1	day	24 mo: Two-yr old child data based on information in Heard & Chamberlain (1982) for adults, and O'Flaherty (1993). 0-84 mo: Calculations done from data for 24 mo & assumed proportional body surface area to other ages.	Heard & Chamberlain 1982; O'Flaherty 1993
Pb transfer time from blood to kidney (24 months – other are calculated)	10	days		Heard & Chamberlain 1982; O'Flaherty 1993

Parameter	Default Value	Units	Origin/ Description	Reference/ Source
Pb transfer time from blood to liver (24 months – other are calculated)	10	days		Heard & Chamberlain 1982; O'Flaherty 1993
Pb transfer time from blood to other soft tissue (24 months – other are calculated)	10	days		Heard & Chamberlain 1982; O'Flaherty 1993
Pb transfer time from blood to urine (24 months – other are calculated)	20	days		Various (consult US EPA 1994b, pg. B-16)
Pb transfer time from plasma-ECF to red blood cells	0.1	days	Value assigned as plausible nominal value reflecting best professional judgement.	

[a] Note the parameters in this table reflect only those non-modifiable parameters for which a distinct value exists. In addition to these parameters, a large number of non-modifiable equations exist that result in other parameters essential to the uptake and biokinetic modules of the IEUBKwin model.

5.2.1.3 Calculations

As mentioned previously, the IEUBK model calculates blood lead concentrations in yearly increments in children aged 0-84 months (7 yrs) based on user-specified inputs or default values for exposure and uptake parameters.

The user-specified exposure parameters are applied in a series of equations to calculate media-specific intake rates by age (refer to Appendix 5, Figures A1.1-A1.5 for exposure equations and Table A5.1 for descriptions and values of exposure parameters as used in the site modeling). These media-specific intake rates are then transformed into media-specific uptake rates through consideration of lead absorption (or bioavailability), as well as attribution to passive and active uptake (refer to Appendix 5, Figure A5.6 for uptake equations, and Table A5.2 for descriptions and values of uptake parameters used in the site modeling). Media-specific uptake rates are then added together to create a total uptake rate, which is then used as an input for a series of differential equations in the biokinetic component of the model [14].

5.2.1.4 Outputs

A user may enter values up to six significant figures. As with any modeling exercise the true precision of any calculated output can be highly influenced by the least precise input value (US EPA 2007a). In the IEUBK model, outputs are reported up to three significant figures to the right

[14] For details of the biokinetic equations and parameters, refer to US EPA (1994b, 2009d)

of the decimal point, except for blood lead concentrations, which are reported to one digit to the right of the decimal point, at least in the text output files. The graphical output files report blood lead levels up to three decimal places and this output has been used to allow prediction of blood lead concentrations less than 0.1 µg/dL since the alternative would report these as zero.

IEUBKwin calculates four primary outputs:

- geometric mean blood lead levels, by age (text file)
- percent of children with blood lead levels in excess of a user-specified level of concern (typically 10 µg/dL) (distribution curve)
- average media-specific daily lead uptake rates (text file)
- media-specific remediation goals

As previously stated, IEUBKwin provides estimates of a plausible distribution of blood lead levels that are centered on the calculated geometric mean blood lead from the entered exposure data. This is the geometric mean blood lead level of a hypothetical population of children which has an assumed geometric standard deviation derived from population surveys of blood lead levels in US children. From this distribution, IEUBKwin calculates the probability that a child's blood lead concentration will exceed a user-selected blood lead concentration level of concern (generally 10 µg/dL) according to the following equation (US EPA 2007a):

$$Z = (\ln(10) - \ln(GM)/\ln(GSD)$$

Where:

z	= standard normal deviate (distance between blood lead of 10 µg/dL and the GM blood lead concentration).
10	= 10 µg/dL cutoff value
GM	= geometric mean
GSD	= geometric standard deviation

The default GSD is 1.6.

After calculating the 'z' score, the IEUBKwin model simulates a standard normal distribution using the calculated GM and GSD. A difference approximation method is used to calculate the probability of exceedence.

Figure 5.2.2 shows an example of the type of graph produced by the IEUBKwin model. It graphs the probability distribution (%) of blood lead concentrations µg/dL) for a 1 – 2 year old child located at Receptor 8 for Scenarios 1a and 3, this is the receptor location most impacted by dust emissions from the proposed Rasp mine. Scenario 1a is lead exposure from existing free areas, and Scenario 3 is lead exposure from dust derived from free areas with 80% dust control

plus the dust exposure from mine activities. The graphs show a predicted incremental blood level of 3.0 and 0.75 µg/dL for Scenario 1a and 3, respectively, resulting from this exposure with 0.5% and 0% of a hypothetical population of 2 yr old children at that location exceeding the NHMRC recommended goal of 10 µg/dL.

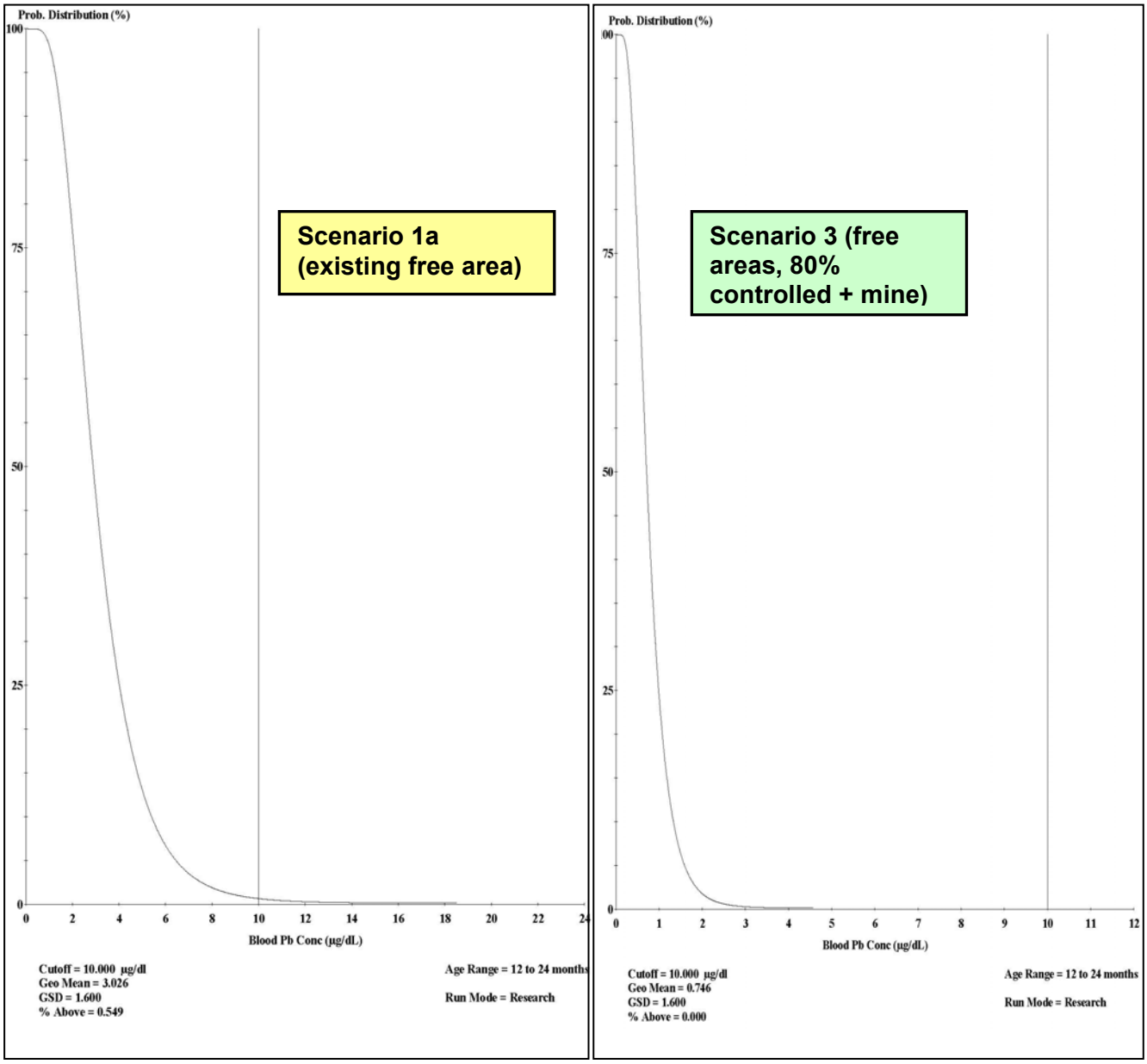


Figure 5.2.2: Probability Distribution (%) for a 1-2 year old child's blood Pb concentration (µg/dL) at Receptor 8, Scenarios 1a and Scenario 3.
Note Receptor 8 is the most affected receptor location within the air dispersion modeling domain and is within the boundary of the mine lease. Also there is a marked difference between the existing situation after 15 years (S1a) and that if the mine proceeds (S3). The predicted increase in blood concentrations is 3 µg/dL for the former and 0.75 for the latter (a 25% benefit).

5.2.1.5 Model limitations

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. Changes in exposure are "smoothed over one year" (US EPA 1994a). The IEUBK model allows changes in air, food, dust and soil lead exposure input parameters only at one-year intervals, and does not allow annual changes in drinking water lead.

The model therefore assumes that for any year environmental lead concentrations are at steady state during that year. In this HRA 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.

The blood lead concentrations in children less than 6 months of age will be affected by pre-natal lead exposure and are not likely to show much influence from exposure to soil, dust and paint (US EPA 1994a). Therefore, the results of the model simulations are not reported for children younger than 6 months of age in the text files.

5.2.1.6 Model Validation

In designing the IEUBK model, the US EPA underwent several validation exercises, described in the review document for the National Ambient Air Quality Standards for Lead (US EPA 1989a). The most detailed analysis involved using data generated around the smelter in East Helena, Montana. For this dataset, two types of validation efforts were undertaken. The first included using the best historical data available for the model parameters such as air, soil, and household dust lead exposures; in the second effort, predicted levels of air (using dispersion modeling that accounted for emissions and background contributions), soil and dust (using generalized relationships from empirical analyses of a range of point source data) were used to estimate blood lead levels. Results of the two different East Helena validations showed good concordance between observed and predicted average blood lead levels in children living near the smelter (the differences observed were not statistically significant). Other validation

exercises by the US EPA used the model for predicting blood lead levels in Omaha, Nebraska (1971-1977) and Silver Valley, Idaho (1974-1975). The model also performed well in these validations in predicting blood lead levels below 25-35 µg/L. At higher exposure levels, the model underestimated actual exposures.

Zaragoza and Hogan (1998) independently tested the computer code within the IEUBK model, and concluded that the model correctly calculates the equations specified in the model theory documentation. They identified several issues with the model documentation that may affect the ability of an independent reviewer to understand the workings of the model but not the model's reliability.

Specific validation using site data was also conducted by Hogan et al. (1998). They used residence-specific environmental lead measurements from three epidemiologic datasets (collected in Palmerton, Pennsylvania; Madison County, Illinois; Jasper County, Missouri; and Galena, Kansas [15]) as inputs for the IEUBK model to predict blood lead measurements and compared the predictions with blood lead levels measured in children living in those residences. The authors found IEUBK-predicted blood lead levels agreed with observed blood lead levels within 1µg/dL, and IEUBK-predicted risk of blood lead greater than 10 µg/dL agreed with observed population exceedences within 4%. The authors concluded that the agreement was reasonably close. They stated that the only limitation to the model they were able to identify was that blood lead level predictions >30 µg/dL were less reliable, based on limited supporting data. This may have been due to measurement error (e.g. dust samples may not have been proportionally collected according to the children's typical activities, thus the lead measurement of the composite may not represent actual exposure).

Carroll and Galindo (1998) also reported on this so-called "measurement error." They did not conduct specific validation work for the IEUBK model but found that measurement error in exposures have the potential to falsely invalidate a model, which is functioning correctly. They stated that applying error-prone lead exposure data to the IEUBK model will unsurprisingly result in poor blood lead predictions, especially of extremes. Therefore, the authors conclude that the IEUBK model cannot be validated by applying it to error-prone lead exposure data.

[15] This multisite study was designed by the ATSDR and the US EPA and conducted in 1991 in order to evaluate populations of all ages near these Superfund sites for possible health effects related to chronic, low-level Pb and Cd exposure associated with nearby, but no longer active, smelters. The authors combined the Jasper County and Galena datasets into one, as they had been designed with a common comparison group. This was done in order to maximize sample sizes for comparisons within subsets of each dataset.

Various overseas agencies have applied the IEUBK model in their risk assessments (ATSDR 2006, 2008, 2009; Ontario MoE 2002; SARA Group 2008, commissioned by the Ontario MoE). US EPA has also applied the IEUBK model in setting air quality standards for lead, as well as to establish cleanup criteria at numerous sites of environmental contamination (US EPA 1994e).

5.2.2 Adult Lead Model (ALM)

The Adult Lead Methodology (ALM) is a model developed by the US EPA in 1996, originally published as a report and now available as an excel spreadsheet It 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 employees a simplified representation of lead biokinetics to predict quasi-steady state blood lead concentrations among women with relatively steady exposure patterns (US EPA 1996). The algorithm embedded into the excel spreadsheet ALM is be depicted with the following equation:

$$PbB_{adult,central} = PbB_{adult,0} + \frac{PbS \times BKSF \times IR_s \times AF_s \times EF_s}{AT} \quad \text{.....Equation 5.1}$$

Where:

$PbB_{adult,central}$	=	Central estimate of blood Pb concentration (µg/dL) in women of child-bearing age that have site exposure to soil Pb concentration, PbS.
$PbB_{adult,0}$	=	Typical blood Pb concentration (µg/dL) in women of child-bearing age in the absence of exposure to the site being assessed.
PbS	=	Soil Pb concentration (µg/g).
BKSF	=	Biokinetic slope factor relating (quasi-steady state) increase in typical blood Pb concentration to average daily Pb uptake (µg/dL blood Pb increase per µg/d Pb uptake).
IR_s	=	Intake rate of soil, including both outdoor soil and indoor soil-derived dust (g/d).
AF_s	=	Absolute gastrointestinal absorption fraction for ingested Pb in soil and Pb in dust derived from soil (unitless).
EF_s	=	Exposure frequency for contact with assessed soils and/or dust derived in part from these soils; may be taken as days per year for continuing long term exposure.
AT	=	Averaging time; the total period during which soil contact may occur; 365 days/year for continuing long term exposures.

The US EPA has set a number of recommended default values in the ALM, which are illustrated in Table 5.2.3.

Toxikos has made use of the ALM model for calculating 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 (refer to Section 5.4). The input parameters for the ALM were modified to reflect site-specific conditions. The modifications of the input parameters and descriptions of their derivation can also be found in Table 5.2.3.

Table 5.2.3: Default input parameters for the ALM model and those applied by Toxikos.

Parameter	Default Value (US EPA 1996)	Toxikos Used Value	Description & Comments
$PbB_{adult,0}$	1.0 µg/dL	1.3 µg/dL	This represents the 'background' geometric mean (GM) blood Pb concentration in women of child-bearing age, in the absence of exposures being assessed. The value used by Toxikos reflects the latest published data for GM blood Pb levels (from 2008) in antenatal women in Broken Hill (GWAHS 2009).
BKSF	0.4 µg/dL per µg/d	Not modified	This parameter relates the blood Pb concentration to Pb uptake. The recommended value is based in data reported by Pocock et al. (1983, cited in US EPA 1996) on the relationship between tap water Pb concentrations and blood Pb concentrations for a sample of adult males, and on estimates of the bioavailability of Pb in tap water. There is some uncertainty associated with applying this value to populations of pregnant women, since it is extrapolated from adult men to women of child bearing age. In the absence of site-specific information, the default value was applied.
IR_s	0.05 g/day	Not modified	This default value is considered a plausible point estimate of the central tendency for daily soil intake from all sources, and is supported by recommendations in the current and updated draft US EPA exposure factors handbooks (US EPA 1997, 2009a)

AF_s	0.12	Modified to reflect receptor-specific values of BAc	This is the fraction of Pb in soil ingested daily that is absorbed from the gastrointestinal tract. The default value is calculated by multiplying the absorption factor for soluble Pb of 0.2 and a relative bioavailability of 0.6 (soil/soluble). The absorption factor for soluble Pb of 0.2 represents a weight of evidence determination on experimental estimates of the BA of ingested Pb in adult humans, with consideration of three major sources of variability (food intake, Pb intake and Pb form and particle size). This factor was not modified. However, the BAc of Pb has been measured for samples in Broken Hill by means of the PBET test. Receptor-specific BAc (or relative BA) values were calculated and used in lieu of the default value of 0.6 to calculate the absorption fraction (ie. 0.2 x BAC = AF_s)
EF_s	219 days/year	365 days/year	The default exposure frequency is the central tendency occupational exposure frequency recommended by the US EPA superfund guidance. However, because the risk assessment considers residential exposure, not occupational, this was modified to a continuous exposure throughout the year.
ET	365 days/year	Not modified	This is consistent with the averaging time for continuing long term exposures.

5.3 IEUBK Parameters modified by Toxikos

5.3.1 Time spent outdoors

The IEUBK default values for time spent outdoors (timeout) are 1, 2, 3, and 4 hours/day for 0-1, 1-2, 2-3, and 3-7 year olds, respectively. These default values were derived from a literature review of the US population (Pope 1985, cited in US EPA 1994b).

These values have been adjusted to reflect the most recent available data for an Australian population. Brinkman et al. (1999) conducted a longitudinal study for activity patterns in Port Pirie, South Australia, and estimated time spent indoors, outdoors and away from home for children aged 7-31 months during a child's 'waking hours' (14 hours, 7:30 a.m. – 9:30 p.m.). Table 5.3.1 shows the average percent time spent outside for a child's 'waking hours' (% of 14 hours) for each age group estimated by Brinkman et al. (1999), as well as the corresponding hrs/day (hrs/24 hrs) spent outdoors.

Table 5.3.1: Average percentage time (out of 14 'waking hours') spent outdoors by child age

Age group (months)	Average time spent outdoors (%)	Corresponding hrs/day spent outdoors
7-9	4	0.6
10-12	3	0.4
13-15	7	1
16-18	10	1.4
19-21	11	1.5
22-24	12	1.7
25-27	15	2.1
28-31	13	1.8

To adopt the information from the Brinkman et al. (1999) study to the IEUBK inputs, the number of hours per day spent outdoors was averaged for each representative age group in IEUBK to give the following:

- 0.5- <1 year old : 0.5 hours/day
- 1- <2 year old : 1.4 hours/day
- 2- <3 year old : 2 hours/day
- 3- <7 year old : assume same as 2-3 year old.

5.3.2 Ventilation rate

The IEUBK defaults for ventilation rates (ventrate) by age are 2, 3, 5, and 7 m^3/day for 0-1, 1-2, 2-5, and 5-7 year olds, respectively. The estimates are based on body size in combination with smoothed data from Phalen et al. (1985, cited in US EPA 1994b).

These values have been adjusted to correspond to the most recent available data for daily inhalation rates recommended for use in risk assessments in the US EPA child exposure factors handbook (US EPA 2008, Table 16-1). The IEUBK has not yet been updated for these exposure parameters.

The inhalation rates recommended for use by the US EPA (2008) are the following:

- 0- <1 year old : 5.4 m^3/d
- 1- <2 year old : 8.0 m^3/d
- 2- <3 year old : 9.5 m^3/d

- 3- <4 year old : 10.9 m^3/d
- 4- <5 year old : 10.9 m^3/d
- 5- <6 year old : 10.9 m^3/d
- 6- <7 year old : 12.4 m^3/d

5.3.3 Soil and dust ingestion

The IEUBK default values for combined soil and dust ingestion are 0.085, 0.135, 0.1, 0.09, and 0.085 g/day for 0-1, 1-4, 4-5, 5-6, and 6-7 year olds, respectively. These values correspond to values reported for 1-2 year old children by several authors such as Binder et al. (1986, cited in US EPA 1994b) and Clausing et al. (1987, cited in US EPA 1994b). Sedman (1987, cited in US EPA 1994b) extrapolated these estimates to those for children aged 0-7 years.

These values have been adjusted to correspond to the most recent available data for soil and dust ingestion rates for non-soil-pica and non-geophagy behaviour recommended for use in risk assessments in the US EPA child exposure factors handbook (US EPA 2008, Table 5-1).
The soil and dust ingestion rates recommended for use by the US EPA (2008) are the following:

- 0 - <1 year old : 0.06 g/d
- 1 - 7 year old : 0.1 g/day (this is the same as the default recommended by enHealth 2004)

5.3.4 Ratio of indoor dust lead to soil lead concentration

Because measurement data for indoor dust levels at residences in Broken Hill were not available, indoor dust lead levels were estimated based on outdoor soil lead levels.

In the absence of site-specific indoor dust lead data, IEUBK normally derives the lead concentration in indoor dust through use of the “multiple source analysis” function[16]. The multiple source analysis function calculates indoor dust levels based on the sum of the fraction of outdoor soil and outdoor air lead that contribute to indoor dust lead. Equation 5.2, which is

[16] The function is called “multiple source analysis” because, other than calculating Pb levels in indoor dust based on outdoor soil and air Pb concentrations, it also takes into consideration the fractional contribution of other sources, which include school, occupation and secondary home. The fractional contribution from other sources was switched off for this risk assessment.

reproduced below from Equation Du1b (Figure A5.5 in Appendix 5) shows how IEUBK calculates indoor dust levels of lead.

indoordustconc(t) = [soildust_ratio x Soilconc(t)] + [dustair_ratio x outairconcentration(t)]

……Equation 5.2

Where :

indoordustconc(t)	= the concentration of Pb in indoor dust (µg/g) by age group
soildust_ratio	= the ratio of indoor dust Pb concentration to soil Pb concentration. This value is set to 0.7.
Soilconc(t)	= the concentration of Pb in soil (µg/g) by age group
dustair_ratio	= the ratio of indoor dust Pb concentration to air Pb concentration. This value is not a percentage but rather a mass of indoor dust per mass of outdoor air. This value is set to 100 µg/g dust per µg/m^3 of air [17].
outairconcentration(t)	= outdoor air Pb concentration (µg/m^3). This concentration varies with each receptor.

The IEUBK default value for the ratio of indoor dust lead to soil lead concentration (soildust_ratio) is 0.7 g Pb soil/g Pb dust. A short sheet on the basis of applying this variable and the assumptions associated with it was published by the US EPA (1998). There are a number of assumptions associated with the default value of 0.7:

- Soil lead is the major source of indoor dust lead at the site in question
- There is no enrichment or reduction of lead in the soil fraction that is transported to indoor dust.
- The areas where soil samples are collected coincide with the major areas for soil derived indoor dust.

The default value of 0.7 reflects an analysis of several residential site measurements of outdoor soil and corresponding indoor dust, where the following ratios of indoor dust to soil were calculated:

- East Helena, MT (smelter community) : 0.81-0.89 to 1
- Midvale, UT (smelter community) : 0.68-0.72 to 1
- Butte, MT (mining community) : 0.26 to 1
- Kellogg, ID (smelter community) : 0.09 to 1

[17] The value is cited in the parameter list for IEUBK (US EPA 1994b, US EPA 2009d) as having come from analyses of the 1983 East Helena study (in US EPA 1989a, B-8), which suggest about 267 µg/g increment of Pb in dust for each µg/m^3 Pb in air. A much smaller factor of 100 was assumed by the US EPA for non-smelter community exposure. However, upon consultation of the original document, the derivation of this value is not very clear. Table A-3 in the review of the criteria document for Pb (US EPA 1989a, pg. A-9) suggests that an average of 764 µg/g of indoor dust was found per µg/m^3 of outdoor air near East Helena, a smelter community. Table B-2 (pg. B-8), where the 267 µg/g value came from, does not clearly indicate the meaning of this value. The original East Helena dataset was not publicly available.

A literature search revealed other ratios for indoor dust lead to soil lead to generally be higher than unity (Gulson et al. 1995b & 1996, Rasmussen 2003, NRC 2005). This is most likely due to preferential tracking indoors of soil particles with higher lead concentrations than bulk soil samples, resulting in a particle fractionation-enrichment between outdoor soils and indoor dust. NRC (2005), for example, analysed lead data from soils, entryway mats, and vacuum cleaner dusts at 34 houses along the Coeur d'Alene River basin (in the US), and found lead to generally be a factor of two higher in mat dust and about a factor of 1.2 higher in vacuum cleaner dust when compared to soil.

For this reason, the default factor of 0.7 was modified to reflect the most recent site-specific data for Broken Hill. The only data located for Broken Hill, which included paired residential measurements for both soil lead and indoor dust lead (in the form of vacuum cleaner dust) was from Gulson et al. (1995b). Gulson et al. (1995b) reported measured soil and vacuum cleaner dust lead concentrations from bulk samples (<2 mm) and the fine size fraction (-53+38 µm) for a number of houses in Broken Hill, from which lead isotope ratios indicate that the majority of lead in these houses is derived from an orebody source. Paired data existed for four houses (numbers 526, 541, 556 and 557); each soil lead measurement was conducted in replicate. Thus there were a total of eight soil lead and four vacuum cleaner dust lead measurements for the four houses. This allowed for the calculation of the ratio of average indoor dust lead (taken to be vacuum cleaner dust lead) to average outdoor soil lead. Because one of the calculated ratios (bulk sample, house 556) was more than two standard deviations away from the mean, it was termed an outlier and excluded from the analysis. Therefore, the following ratios (of the mean indoor dust lead to mean outdoor soil lead concentrations) were determined:

- Bulk samples (n=7) : 1.2 :1
- Fine fraction (n=8) : 1.5 :1

Table 5.3.2: Indoor dust to soil lead ratio analysis from Gulson et al. (1995b)

House No. (replicate)	Dust Fraction	Vacuum dust Pb (ppm)	Soil Pb (ppm)
526 (1)	Bulk	4090	2660
526 (2)		4090	1700
541 (1)		615	850
541 (2)		615	1350
~~556 (1)~~ [a]		~~4490~~ [a]	~~500~~ [a]
556 (2)		4490	3260
557 (1)		590	2330
557 (2)		590	650
Average		**2150**	**1830**
Ratio of averages		**1.2 : 1**	
526 (1)	-53 + 38 µm	8300	3815
526 (2)		8300	3165
541 (1)		3750	2415
541 (2)		3750	3225
556 (1)		12100	4540
556 (2)		12100	8500
557 (1)		2600	4475
557 (2)		2600	4735
Average		**6690**	**4360**
Ratio of averages		**1.5 : 1**	

[a] This sample was excluded from the analysis because it was determined to be an outlier. The resulting ratio of indoor dust: outdoor soil of 9 to 1 is more than two standard deviations away from the mean ratio of all other samples.

A ratio of 1.5 to 1 (indoor dust to soil lead) was therefore adopted in the IEUBK analysis instead of the default factor of 0.7 to 1.

5.3.5 Total gastrointestinal absorption of dust and soil lead

The IEUBK default value for total gut absorption of soil (ABSS) and dust (ABSD) lead is 0.3. This value is based on a bioaccessibility of lead in soil of 60% and a bioavailability of soluble lead in water and food of 50% (i.e. 60% x 50% = 30%) (US EPA 1999, 1994a, 2007b).

This default parameter has been modified to reflect site-specific information based on bioaccessibility (BAc) data determined from a PBET test on surface and core soil samples from Broken Hill (refer to Section 2.5). BAc of lead in surface soil samples from the free areas was determined to be 7.3%; that of ore samples was 1.4%.

Hence after determining the relative contribution of lead in dust from free areas (BAc 7.3%) and from mine activities (BAc 1.4%) to the incremental soil increase at specific receptors the bioavailability input for IEUBK modeling for each receptor was calculated as below.

{[Proportion from free area x BAc (free area)] + [Proportion from mine activity x BAc (mine activity)]} x BA (50%).

5.3.6 Net percentage lung absorption of air lead

The IEUBK default value for the net percentage absorption of air lead is 32%. It is based on an estimation of deposition efficiencies of airborne lead particles in young children by the US EPA (1989a), which resulted in a calculated respiratory deposition/absorption of 25-45% for young children living near non-point source areas while 42% was calculated for those living near point sources. The US EPA chose an intermediate value of 32% as the default. The percentage is said to reflect respiratory deposition and absorption.

The site-specific BAc data of lead from the PBET test was conservatively assumed to represent the proportion of lead in the airborne dust that would be available to be absorbed by the lung. The latter was assumed to be 100% absorbed so the bioavailability input for IEUBK became the bioaccessibility of the lead from the respective dust source.

5.4 Model Results

Blood lead modelling for children was carried out in IEUBK for selected receptors which were selected after consideration of proximity to mine activity, the risk zones delineated by Boreland (2010) (Section 2.2), and predicted total dust deposition (low, medium, high) from the mine lease site (i.e. Scenario 3).

Table 5.4.1 below is reproduced from Section 2.3, showing cumulative predicted dust deposition rates at each of the modelled receptors, together with source apportionment at each receptor. The receptors selected for blood lead modelling in children are highlighted in yellow.

Table 5.4.1: Annual lead deposition at residential and other locations (Scenario 3)

Receptor	Total Pb deposition (g Pb/m^2/yr)	Source apportionment of total Pb deposition (% total Pb deposition)	
		Area	Ore
Residences			
1. Piper Street North	**0.3**	91	9
2. Piper Street Central	**0.3**	72	28
3. Eyre Street North	**0.4**	63	37
4. Eyre Street Central	**0.2**	65	35
5. Eyre Street South	**0.2**	65	35
6. South Road (1)	**0.2**	67	33
7. Carbon Lane	**0.1**	59	41
8. Old South Road	**1.1**	64	36
9. South Road (2)	**0.4**	70	30
10. Garnet & Blende Streets	**0.2**	83	17
21. Eyre Street North (1)	**0.4**	94	6
22. Eyre Street North (2)	**0.4**	94	6
23. Eyre Street North (3)	**0.3**	94	6
24. Eyre Street North (4)	**0.3**	96	4
25. Water tank, Lawton Street [a]	**0.2**	92	8
26. Quarry offices	**0.3**	97	3
27. Proprietary square (1)	**0.4**	98	2
28. Proprietary square (2)	**0.2**	97	3
29. Iodide Street (1)	**0.3**	97	3
30. Iodide Street (2)	**0.2**	97	3
31. Crystal Street (1)	**0.2**	96	4
32. Crystal Street (2)	**0.2**	96	4
33. Brown shaft dwelling	**0.2**	98	4
34. Crystal Street (3)	**0.3**	91	9
35. Crystal Street (4)	**0.3**	92	8
36. Crystal Street (5)	**0.2**	92	8
37. Crystal Street (6)	**0.2**	93	7
38. Gypsum Street (1)	**0.07**	57	43
39. Gypsum Street (2)	**0.1**	59	41
40. Silver City Hwy (1)	**0.3**	80	20
41. Silver City Hwy (2)	**0.3**	84	16
42. Silver City Hwy (3)	**0.4**	89	11
Other Locations			
11. Alma Bugdlie Pre-School	**0.1**	65	35
12. Playtime Pre-School	**0.05**	49	51
13. Alma Public School	**0.03**	36	64
14. Broken Hill High School	**0.07**	67	33
15. Broken Hill Hospital	**0.009**	30	70
16. North Broken Hill Public School	**0.005**	29	71
17. Broken Hill Public School	**0.03**	53	47
18. Rainbow Pre School	**0.03**	50	50
19. Willyama High School	**0.004**	29	71
20. Morgan Street Public School	**0.006**	30	70

[a] This receptor was included for purposes of air quality modelling, and was not included as a sensitive receptor.

5.4.1 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 highlighted in Table 5.4.1.

Tables 5.4.2 - 5.4.4 respectively show the resulting 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. The information in Table 5.4.4 (Scenario 3, cumulative exposures from free areas 80% controlled plus mine operations) show 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 4.3.1 showing lead intake relative to 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.

Tables 5.4.2 – 5.4.4 also demonstrate that after 15 years during which the mine lease is left in its existing condition (Scenario 1a) (i.e. 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 5.4.1 for the different age groups, and in Figure 5.4.2 for a two 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 (Figure 5.4.4).

The impact of free area dust control relative to the ‘risk zones’ of Broken Hill is explored further in Section 5.

Table 5.4.2: Predicted blood lead levels (µg/dL) in children for Scenario 1a ('existing free areas', no dust controls)

Receptor	Child Age						
	0.5-1	1-2	2-3	3-4	4-5	5-6	6-7
R1	1.08	1.34	1.24	1.16	1.1	1.03	0.95
R2	0.66	0.82	0.76	0.72	0.68	0.63	0.58
R3	0.87	1.07	0.99	0.93	0.88	0.82	0.76
R4	0.5	0.62	0.57	0.54	0.51	0.48	0.44
R5	0.38	0.47	0.43	0.41	0.39	0.36	0.33
R8	2.46	3.03	2.82	2.66	2.53	2.38	2.2
R9	0.95	1.17	1.09	1.02	0.97	0.91	0.84
R10	0.64	0.79	0.74	0.69	0.65	0.61	0.56
R11	0.26	0.33	0.3	0.29	0.27	0.25	0.23
R12	0.06	0.08	0.07	0.07	0.07	0.06	0.06
R14	0.14	0.18	0.16	0.16	0.15	0.14	0.13
R17	0.04	0.05	0.04	0.04	0.04	0.04	0.03
R18	0.03	0.04	0.04	0.03	0.03	0.03	0.03
R23	0.93	1.15	1.06	1	0.95	0.88	0.82
R32	0.54	0.67	0.62	0.58	0.55	0.52	0.48
R36	0.72	0.89	0.83	0.78	0.74	0.69	0.63
R38	0.13	0.16	0.15	0.14	0.14	0.13	0.12

Table 5.4.3: Predicted blood lead levels (µg/dL) in children aged 0.5-7 years for Scenario 1b ('free areas' with 80% dust controls).

Receptor	Child Age						
	0.5-1	1-2	2-3	3-4	4-5	5-6	6-7
R1	0.22	0.27	0.25	0.24	0.23	0.21	0.19
R2	0.14	0.17	0.16	0.15	0.14	0.13	0.12
R3	0.18	0.22	0.2	0.19	0.18	0.17	0.16
R4	0.1	0.13	0.12	0.11	0.1	0.1	0.09
R5	0.08	0.1	0.09	0.08	0.08	0.07	0.07
R8	0.52	0.64	0.59	0.55	0.52	0.49	0.45
R9	0.19	0.24	0.22	0.21	0.2	0.18	0.17
R10	0.13	0.16	0.15	0.14	0.13	0.12	0.11
R11	0.05	0.07	0.06	0.06	0.05	0.05	0.05
R12	0.01	0.02	0.02	0.01	0.01	0.01	0.01
R14	0.03	0.04	0.03	0.03	0.03	0.03	0.03
R17	0.007	0.009	0.008	0.008	0.007	0.007	0.006
R18	0.006	0.008	0.007	0.007	0.006	0.006	0.006
R23	0.19	0.23	0.22	0.2	0.19	0.18	0.17
R32	0.11	0.14	0.13	0.12	0.11	0.1	0.1
R36	0.15	0.18	0.17	0.16	0.15	0.14	0.13
R38	0.03	0.03	0.03	0.03	0.03	0.03	0.02

Table 5.4.4: Predicted blood lead levels (µg/dL) in children aged 0.5-7 years for Scenario 3

Receptor	Child Age						
	0.5-1	1-2	2-3	3-4	4-5	5-6	6-7
R1	0.24	0.3	0.27	0.26	0.24	0.23	0.21
R2	0.17	0.21	0.19	0.18	0.17	0.16	0.15
R3	0.25	0.31	0.28	0.27	0.25	0.24	0.22
R4	0.14	0.17	0.16	0.15	0.14	0.13	0.12
R5	0.1	0.13	0.12	0.11	0.11	0.1	0.09
R8	0.61	0.75	0.69	0.65	0.61	0.57	0.53
R9	0.24	0.29	0.27	0.25	0.24	0.22	0.21
R10	0.15	0.19	0.17	0.16	0.16	0.14	0.13
R11	0.07	0.09	0.08	0.07	0.07	0.07	0.06
R12	0.02	0.03	0.03	0.03	0.02	0.02	0.02
R14	0.04	0.05	0.05	0.04	0.04	0.04	0.04
R17	0.02	0.02	0.02	0.02	0.02	0.01	0.01
R18	0.01	0.02	0.01	0.01	0.01	0.01	0.01
R23	0.2	0.25	0.23	0.22	0.2	0.19	0.17
R32	0.11	0.14	0.13	0.12	0.12	0.11	0.1
R36	0.15	0.19	0.17	0.16	0.16	0.14	0.13

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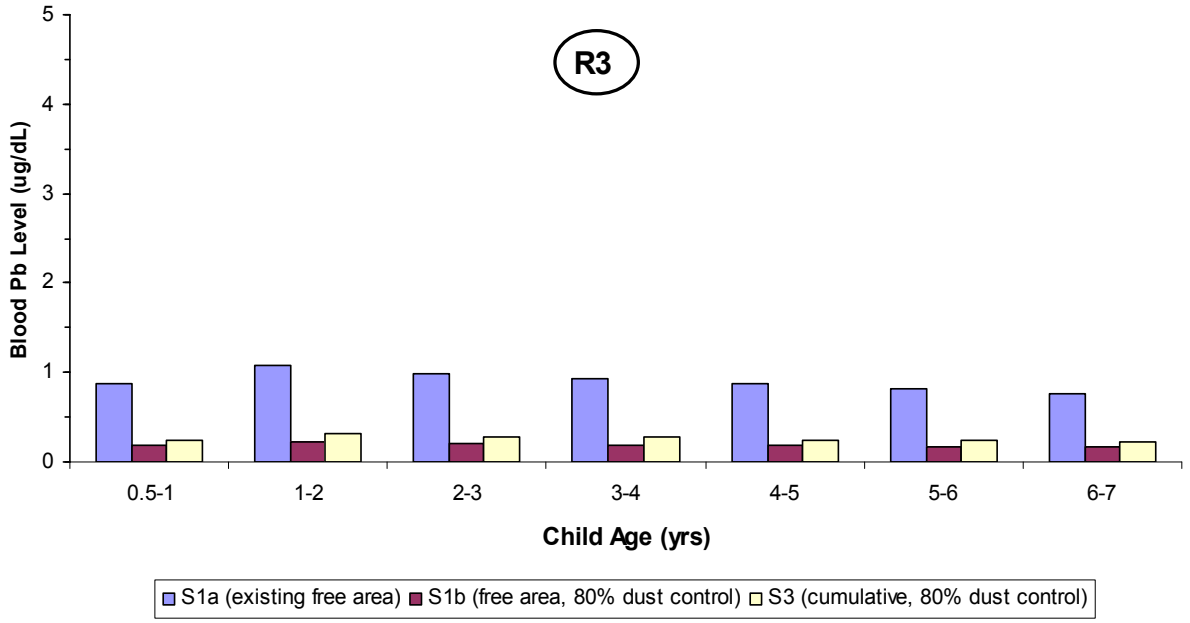


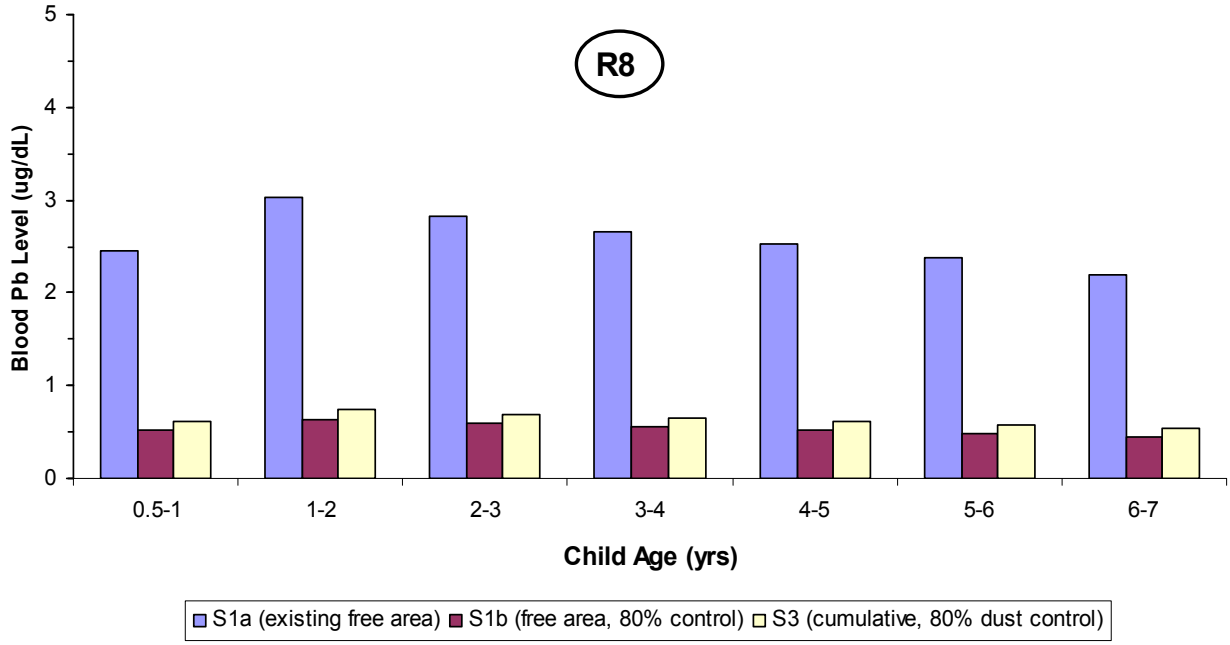


Figure 5.4.1: Predicted incremental blood Pb levels by age group and scenario at Receptors 3 and 8 after 15 years.
The NHMRC (2009) target for blood lead is <10 µg/dL

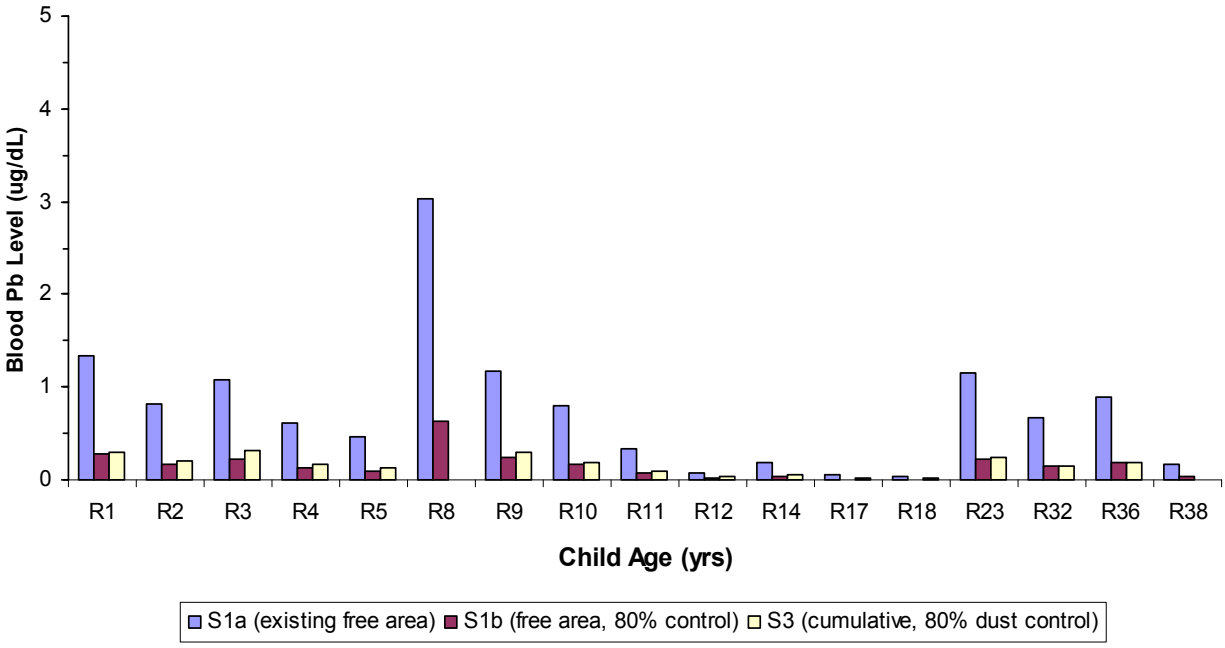
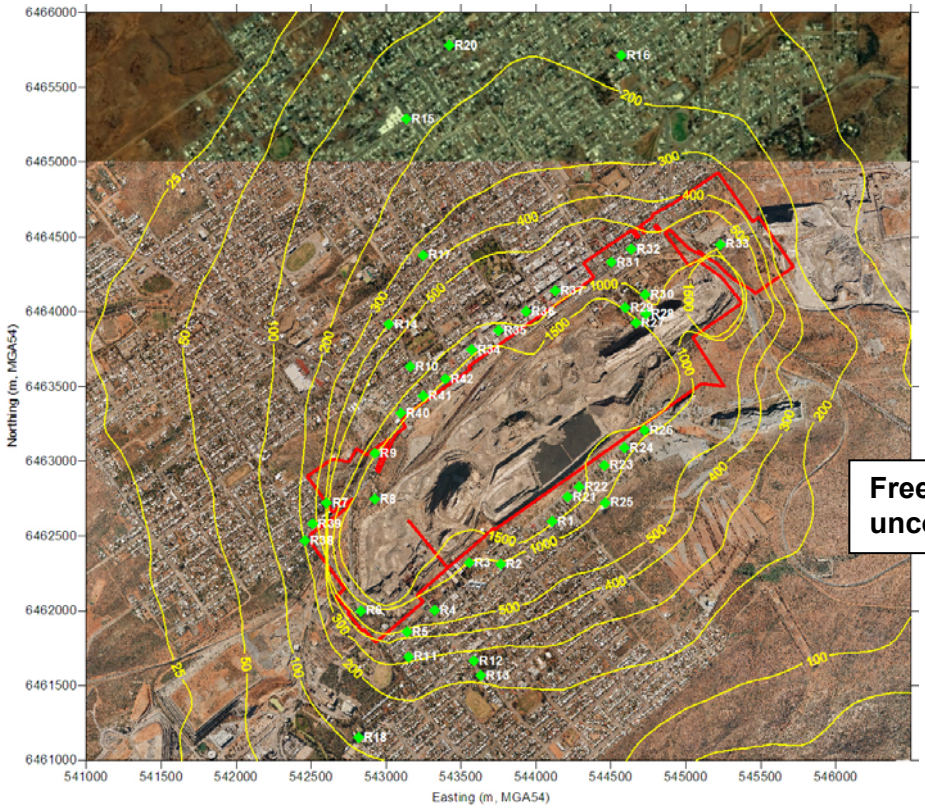


Figure 5.4.2: Predicted blood Pb levels for a 1-2 year old child at selected receptors.

The NHMRC (2009) target for blood lead is <10 µg/dL



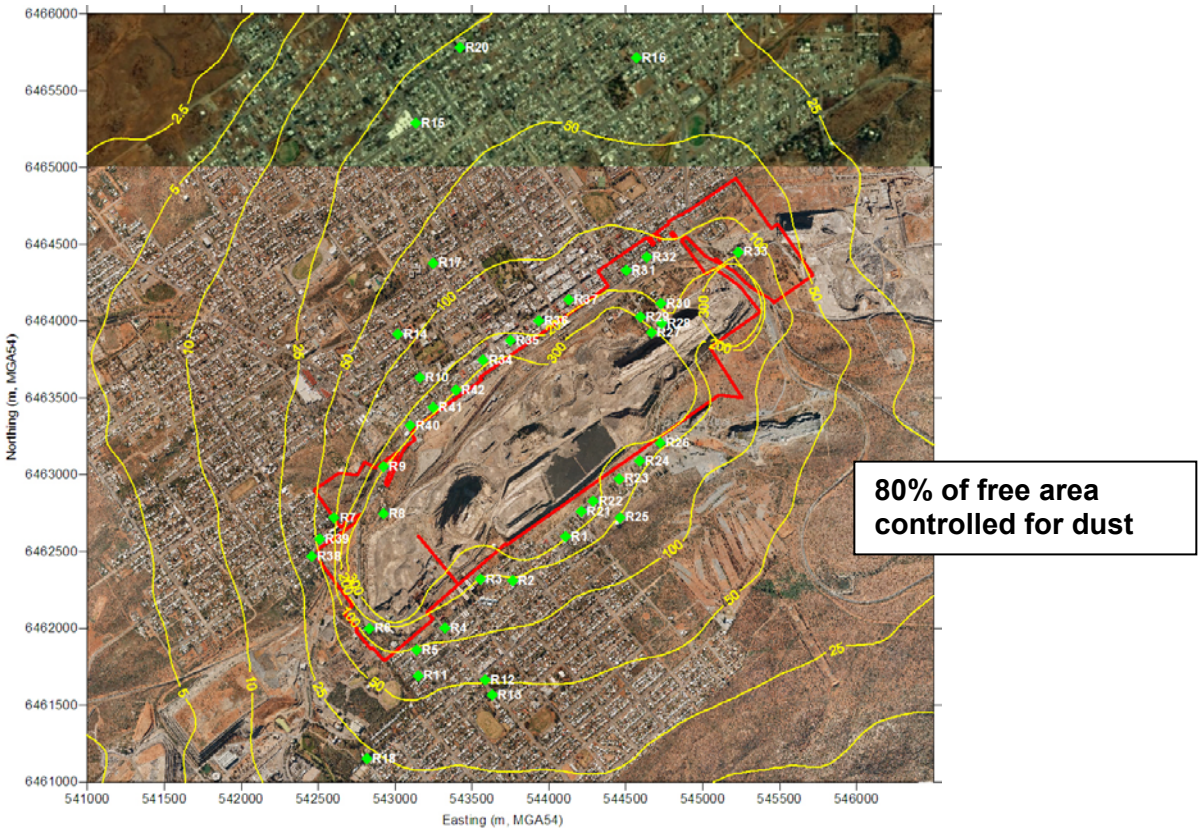


Figure 5.4.3: Impact of controlling free area dust lift off on Pb deposition ($mg/m^2/yr$).

5.4.2 Benefit of free area dust control on blood lead

Lead intake in the IEUBK model is a combination of external soil ingestion and ingestion of indoor dust, inhalation of outdoor dust and indoor dust. Figure 4.3.1 shows ingestion of soil and dust accounts for more than 95% of the incremental intake of lead. This of course needs to be overlayed on the existing background exposure to lead and the blood lead levels that result. Part of the Rasp mine proposal is that there will be additional control of dust lift off from the existing free area, Section 5.4.1 indicates such dust control will result in a beneficial blood lead outcome for people living near the mine site. It is however conceivable that 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.

As discussed in Section 2.2 recent reliable data for background soil concentrations in Broken Hill is not available. However limited information has been provided by the Greater Western Area Health Services (Section 2.2). This data is limited by the number of soil samples analysed (n = 148) from 2004 – 2008 relative to the total number of residences and by the fact that the data has not been obtained by random systematic sampling.

Geometric mean background soil concentrations for 2004 - 2008 were supplied to Toxikos by Boreland (2010) by district. The districts are those established by Boreland et al. (2002) using 1992 Broken Hill City Council soil data, and which subsequently have been divided into five risk zones (Lyle 2006, Boreland 2009a). In Section 2.2 the new data was grouped into the risk zones, according to the location of the 10 districts. The 2004 – 2008 data indicated risk zones 2 and 3 have similar concentrations of lead, and risk zones 4 and 5 have similar concentrations (Section 2.2).

The receptors analysed in this assessment have been mapped by risk zone (refer to Section 2.2), and have been grouped into risk zone 1, risk zones 2+3, or risk zones 4+5, based on locality. The average measured soil lead concentrations give an idea of approximate ranges of existing soil lead and for the purposes of assessing the impact of free area dust control have been grouped into areas of high, medium and low soil lead concentrations:

- **High soil Pb** = Risk zone 1: $\geq$ 1000 µg Pb/g soil
- **Medium soil Pb** = Risk zones 2+3: $\geq$ 300 – 1000 µg Pb/g soil
- **Low soil Pb** = Risk zones 4+5: < 300 µg Pb/g soil

These 'assumed' existing background soil lead levels were added to the expected incremental increase in soil lead (calculated from modelled lead deposition data after 15 years mine operation, assuming zero lead loss: refer to Section 2.3). The resulting combined soil lead levels were used to estimate blood lead levels for a 1-2 year old child hypothetically living at selected receptors in the high, medium and low soil lead areas since birth. A 1-2 year old child was selected, because this is the most sensitive receptor (Figure 5.4.1).

This blood lead modelling (inclusive of the assumed representative background soil lead levels) was conducted for Scenarios 1a (existing free area) and 3 (existing free area 80% controlled plus mine activities), in order to provide an analysis of the effect of implementing the free area dust controls on blood lead levels.

Receptors within the areas of high, medium and low assumed existing lead soil concentrations were further divided into groups according to the lead deposition associated with Scenario 3.

- **High deposition** = $\geq$ 0.3 g/m^2/yr
- **Medium deposition** = 0.1 – 0.3 g/m^2/yr
- **Low deposition** = $\leq$ 0.1 g/m^2/yr

These two receptor classifications were used to establish a risk matrix for judging whether the dust control of free areas in association with mine operations (i.e. Scenario 3) resulted in lower blood lead levels relative to existing circumstances (i.e. no mine and no associated free area dust control) (Table 5.4.6). The benefit was judged as poor, good or very good according to the percentage decrease in predicted blood lead level.

Predicted blood lead levels for a 1-2 year old were modelled using IEUBK for three situations (Figure 5.4.4):

(A). Assuming a range of existing soil lead concentrations that encompassed the high, medium and low soil lead categories without additional deposition from free areas or mine activity.

(B). As (A) above but including 15 years of lead deposition from 'existing free areas' (i.e. Scenario 1a, no mine).

(C). As (A) above but including 15 years of lead deposition from 'existing free areas' 80% controlled and 15 years of mine operation (i.e. Scenario 3).

Figure 5.4.4 is an example of the analysis of one receptor, Receptor 3, the second highest impacted receptor location in the air dispersion modelling domain and the highest outside the boundary of the mine lease. The first two graphs at the top show the decrease in soil lead concentration and corresponding decrease in incremental blood lead levels with increasing percentage of 'free area' controls. Receptor 3 is an example of a receptor with a high annual deposition rate (0.4 $g/m^2/yr$), located in a location with high background soil lead: $\geq$ 1000 µg/g, risk zone 1.

Regarding Figure 5.4.4:

The graph in the middle shows predicted blood lead levels for a 1-2 year old child at

- varying assumed existing soil lead levels without additional deposition from free areas or mine activity.(blue line, A),
- as above but including 15 years deposition from 'free area' with controls and no mine (S1a) (red line, B), and
- assumed variable existing soil concentrations including 15 years deposition from free areas ('80% dust control') plus the mine (green line, C).

The graph indicates that at an assumed soil lead concentration of 2500 µg/g, for example, the predicted typical blood lead level for a 1-2 year old at this location is 4.4 µg/dL without additional exposure, and would be expected to rise to 5.3 µg/dL in 15 years if no controls are put into place on existing 'free areas' (assuming no loss of soil lead over 15 years), and exposure assumptions remain the same over that period. However, the typical blood lead level of a 1-2 year old is only predicted to rise to 4.7 µg/dL (as opposed to 5.3) after 15 years if 'free areas' dust emission is controlled as

intended, and the mine operations went ahead. This is a benefit reduction in blood lead level of 11.3%.

The fourth graph at the bottom shows the percentage decrease in predicted blood lead if the existing soil concentration varied from 100 to 2,500 µg/g soil. Since Receptor 3 is located in an area (RZ1) of presumed high existing soil concentration, the soil concentrations may be somewhere between 1,000 to 2,500 µg/g soil. The corresponding reduction in blood lead levels achieved with control of free area dust and mine operations is 11 – 24% (Table 5.4.5).

These 'beneficial' decreases in blood lead come about because:
1) the IEUBK blood lead modelling indicates the ingestion pathway accounts for >95% of blood lead and
2) the additional dust controls result in a smaller increase in residential soil lead that would otherwise occur if the status quo for the free areas of the mine lease remained for 15 years.

The same figures have been generated for additional receptors (refer to Appendix 7) and the beneficial decreases in blood lead summarised in Table 5.4.5. The percentage decrease in blood lead for each receptor was calculated by the Excel spread sheets used to generate the graphs in Appendix 7.

Figure 5.4.4: Impact of varying levels of controls on soil Pb, incremental blood Pb and total blood Pb at assumed existing soil levels for a 1-2 year old child.

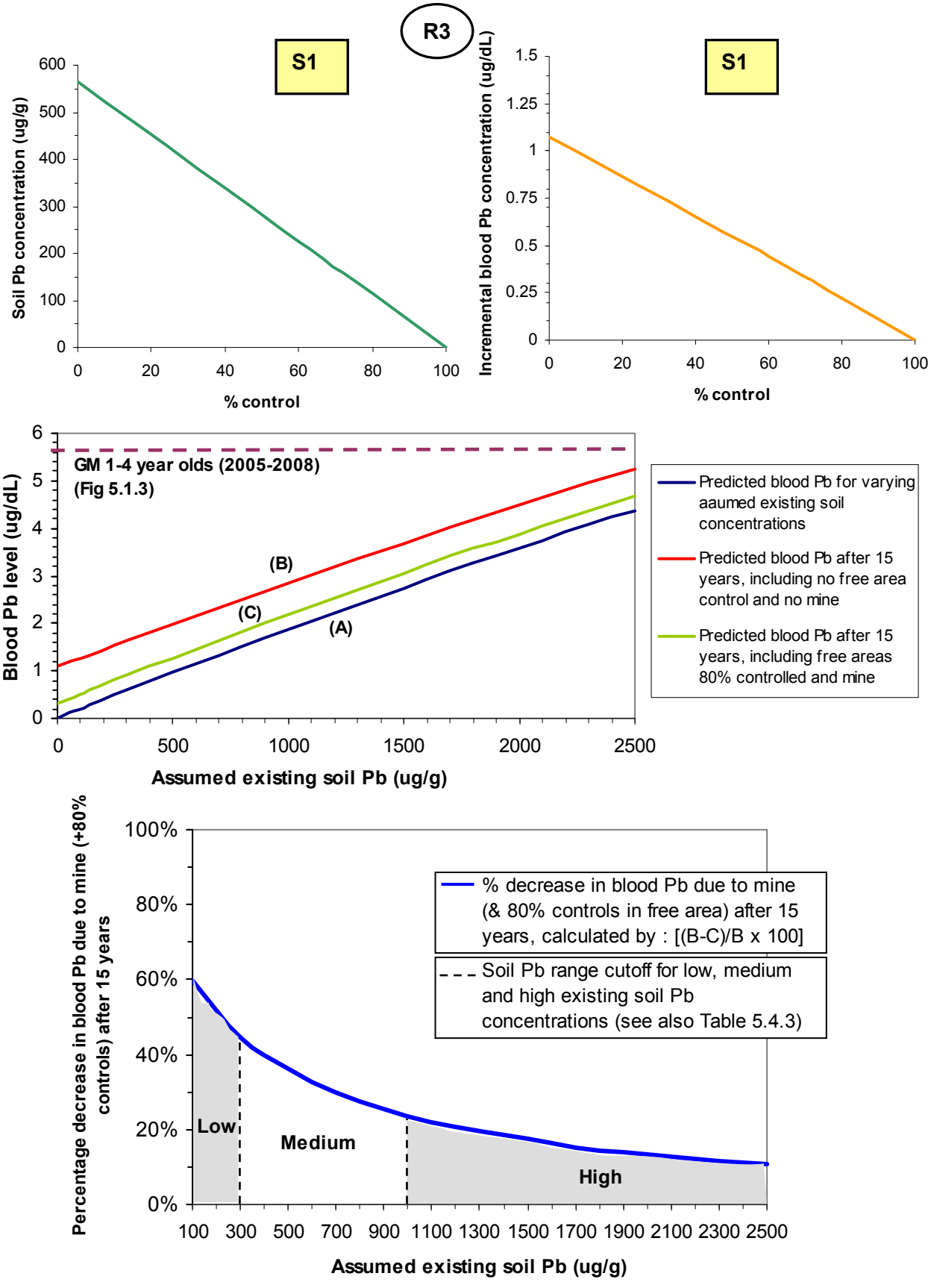


Table 5.4.5: Percentage decrease in blood lead for a 1-2 year old child as a result of dust controls associated with mine operation at selected receptors

Selected Receptor	Predicted total cumulative dust deposition ($g/m^2/yr$)	Percentage decrease in blood Pb (%)
R1	0.3	14-30
R2	0.3	9-21
R3	0.4	11-24
R4	0.2	7-17
R5	0.2	13-31
R9	0.4	27-49
R10	0.2	21-42
R11	0.1	10-25
R12	0.05	9-15
R14	0.07	6-16
R17	0.03	3-8
R18	0.03	2-4
R23	0.3	26-48
R32	0.2	19-41
R36	0.2	23-46
R38	0.07	6-18

Because the existing soil concentrations at the receptors is not known with precision assumptions have been made as to the likely soil concentration being low, medium or high based on 2004 – 2008 soil data for Broken Hill and the risk zones that the data may represent (Section 2.2). Equally the 'beneficial' reduction in predicted blood lead is also imprecise as it depends on the existing soil lead concentration. So as not to convey an impression of accuracy when it is not warranted receptors have been grouped into one of three qualitative categories based on the predicted **decrease** in blood lead levels as a result of implementing dust controls on the 'free areas'. The categories relate to whether the reduction in blood lead can be perceived as poor, good or very good.

- Poor: <10% decrease in blood Pb
- Good: 10-20% decrease in blood Pb
- Very good: $\geq$ 20% decrease in blood Pb

Whether or not a specific receptor falls into a certain category is dependent on the existing soil lead concentration (high, medium or low) and the annual lead deposition at the receptor (high, medium, or low).

Table 5.4.6 is a ‘beneficial’ risk matrix to qualitatively show the decrease in blood lead levels for a 1-2 year old child that could be expected as a result of controls implemented in Scenario 3 (cumulative, free areas 80% dust controlled, 15 years mine life) vs. Scenario 1a (no mine, 0% ‘free area’ controls) at the sensitive receptors grouped according to dust deposition and assumed existing soil lead levels.

Although no receptors could be modelled for the high risk zone/low deposition rate, the low risk zones/medium or high deposition rate situations, their associated decreases in blood lead due to ‘free area’ controls associated with the mine have been predicted in the risk matrix by extrapolation of the modelled results from analyses undertaken such as that depicted in Figure 5.4.4.

A square containing two colours denotes that the range of percentage reduction for the receptor(s) overlap the allocated percentage decrease for a poor, good or very good outcome as described above.

From Table 5.4.6, it is evident that the greatest benefit of ‘free area’ dust controls occurs at locations where existing soil lead concentrations are low or medium and lead deposition is medium or high.

Table 5.4.6: Predicted percentage decrease in blood lead for mine vs no mine (i.e. existing situation) for a 1-2 year old child [b, d, e]

Assumed existing soil Pb		Pb deposition ($g/m^2/yr$)		
	(µg/g)	Low (≤ 0.1)	Medium (0.1 - <0.3)	High (≥ 0.3)
Low (RZ 4+5)	≥ 100- < 300	R12 R13 R15-20	None [g]	None [g]
Medium (RZ 2+3)	≥ 300 – 1000	R7 R11 R14 R38-39	R5-6 [c] R10 (R25[a]) R28 R30-33 R36-37	R9 R21-24 R26-27 R29 R34-35 R40-42
High (RZ1)	≥1000	None [g]	R4	R1- 3 [f] (R8) [d]

[a] R25 is a water tank that was included in the air dispersion modeling in order to assess its suitability as a future monitoring site

[b] R11-20 are schools in Broken Hill, but have been treated as residences in this assessment.

[c] R6 is not actually in risk zone 2, but has been treated as such due to its vicinity to it and lack of background soil measurements for this area.

[d] R8 is located on the mine lease, and has been included in risk zone 1.

[e] R21-33 are assumed to have background soil concentrations associated with risk zone 3, due to their proximity to this zone.

[f] R1 is assumed to have background soil concentrations associated with risk zone 1, due to its proximity to this zone.

[g] No receptors in this risk zone at the nominated deposition rate.

Legend

Poor	≤ 10% decrease in blood Pb associated with project
Good	10-20 % decrease in blood Pb associated with project
Very Good	≥ 20 % decrease in blood Pb associated with project

5.5 Discussion and conclusions

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.

A benefit matrix for amelioration of increases in blood lead concentrations over the life of the proposed mine has been constructed. The matrix consists of low, medium and high existing soil concentrations determined from 2004 – 2008 soil lead data and location of receptors in historically established 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.

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.

6. Cancer risk assessment

Lifetime cancer risk for carcinogens whose mode of action is 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 air borne carcinogens, the "unit" is generally 1 $\mu g/m^3$ and depending on the nature of the data used to determine the carcinogenic potency, the numerical value refers to the probability of developing, or dying of cancer. Thus a life time exposure to 1 $\mu g/m^3$ of a substance may carry a risk of 1 chance in 200 of developing cancer; this is often interpreted as meaning, if 200 people were exposed to 1 $\mu g/m^3$ for their lifetime then one individual may develop cancer. This probability is expressed as 0.5 in 100, or 0.5×10^{-2} per $\mu g/m^3$, written as 0.5×10^{-2} $(\mu g/m^3)^{-1}$. The target acceptable risk band adopted in this assessment is 1×10^{-6} to 1×10^{-5}, i.e. with a lifetime exposure there is a chance developing a tumour between one in one hundred thousand and one in a million.

Lifetime cancer risk = lifetime average air concentration $(\mu g/m^3)$ x unit risk factor $(\mu g/m^3)^{-1}$

$= A_C\ (\mu g/m^3) \times UR\ (\mu g/m^3)^{-1}$

In this risk assessment literature values of carcinogenic potency have been used without any attempt to evaluate the veracity of the value. Where several unit risk values are in the literature, the value indicative of the highest potency has been used except where there is appropriate precedence for either an Australian authority or the WHO using a different value for deriving a standard, in which case the latter has been used in the risk assessment.

It is common practice to assume cancer risks due to different genotoxic carcinogenic air pollutants is additive, summing the individual cancer risks is used to estimate a total lifetime risk of developing cancer (Morello-Frosch et al. 2000, Tam and Neumann 2004, Pratt et al. 2000). However unit risk estimates are upper bound 95% confidence estimates and do not reflect the central tendency or average. When several upper bound estimates are added together there is the question of whether the predicted cancer risk is plausible. The greater the number of carcinogens being considered the more unlikely, in theory, the true risk for each carcinogen will lie near the upper bound estimate. The process of adding cancer upper bound cancer risk estimates together is inherently conservative. Cogliano (1997) has shown that the resulting risk estimate becomes increasingly improbable the greater the number of risk estimates, but not necessarily misleading. However to obtain a cancer risk estimate closer to the true risk Cogliano (1997) considers central estimates are more plausible. Unfortunately central estimate unit risk factors are not readily available.

Of the chemicals of potential concern evaluated in this risk assessment, four metals (As, Be, Cd, and Ni) are considered genotoxic carcinogens (Appendix 1). These four metals have been assessed for incremental cancer risk via inhalation exposure. In addition, As was assessed for its potential to induce cancer via ingestion.

Overall the emissions from the mine site do not pose an inhalation nor oral carcinogenic risk to persons living around the site.

The highest calculated total cancer risk is approximately 12 x 10^{-7} (Figure 6.1, Receptor 8, Scenario 3). This is within the commonly accepted risk band of between one in a million and one in one hundred thousand[18] (Table A1.1.4). Furthermore this is an aggregate of the individual inhalation cancer risks for Cd, Be, Ni and As), as well as the incremental oral/dermal cancer risk for As. The incremental cancer risk spreadsheets have been reproduced in Appendix 8. For this HRA evidence was not located to indicate the inhalation carcinogenic risk

[18] To the best knowledge of Toxikos an official acceptable carcinogenic risk level for Australia has not been formally announced by any agency. In the US a risk of 1 in a million is regarded as being *de minimus* and is the risk level used by the Australian NHMRC for establishing drinking water guidelines for genotoxic carcinogens. However many of the risk assessment guideline documents for Australia recognise the level of carcinogenic risk deemed to be acceptable is a matter for the community as a whole or the community bearing the risk to decide. In New Zealand an incremental risk level of 1 in 100,000 per lifetime (1 x 10^{-5}) is considered as being acceptable (NZ MfE 1997, 1999, NZ MoH 2000). This is a policy decision based on Ministry of Health deliberations for derivation of public health guidelines for New Zealand and the objective of protecting 'almost all' individuals. There are also examples in Australia where a lower risk level than 1 x10^{-6} has been used for evaluation of public health impacts or establishment of standards, for example the Air Toxics NEPM.

from these agents are additive, it is also conservative to include the oral/dermal cancer risk for As.

From Figure 6.2, it is evident that Cd, followed by As, are the highest contributors to the incremental cancer risk.

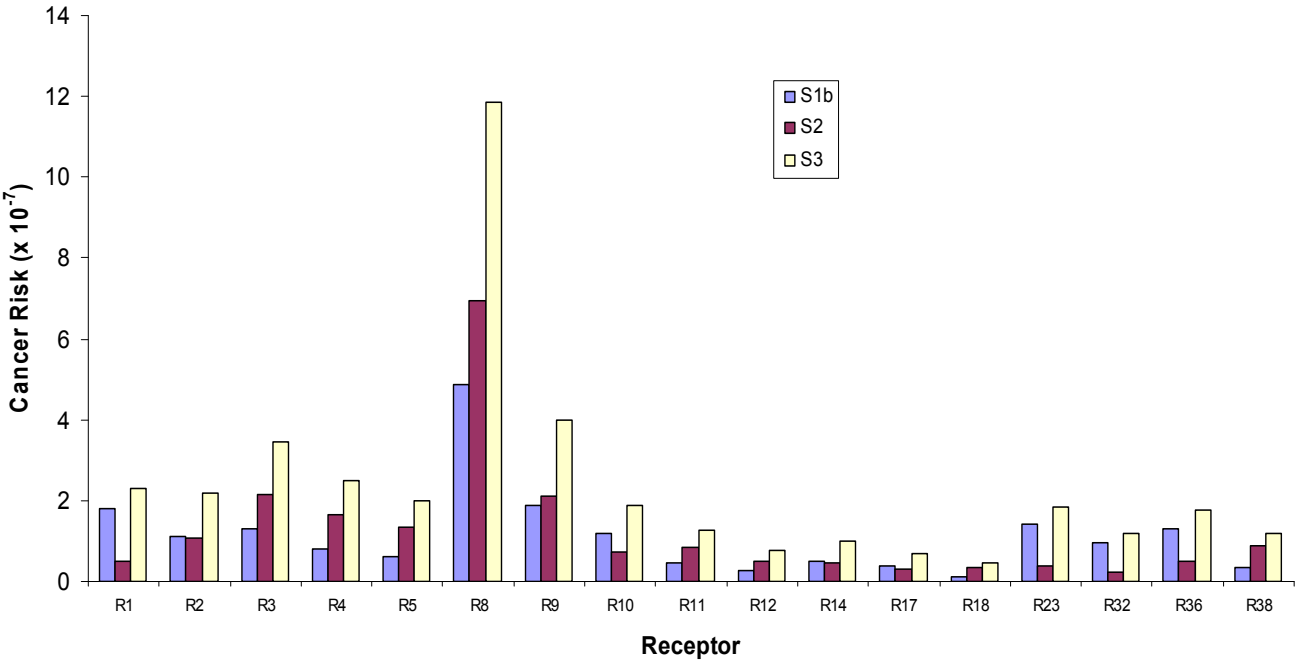


Figure 6.1: Incremental cancer risks at selected receptors for the three scenarios evaluated (S1b, S2 and S3).
Note that all incremental cancer risks are within or lower than the risk usually deemed 'acceptable' by agencies in Australia (10^{-5} or 10^{-6}). The total cancer risk in this figure includes oral as well as inhalation cancer risk for As.

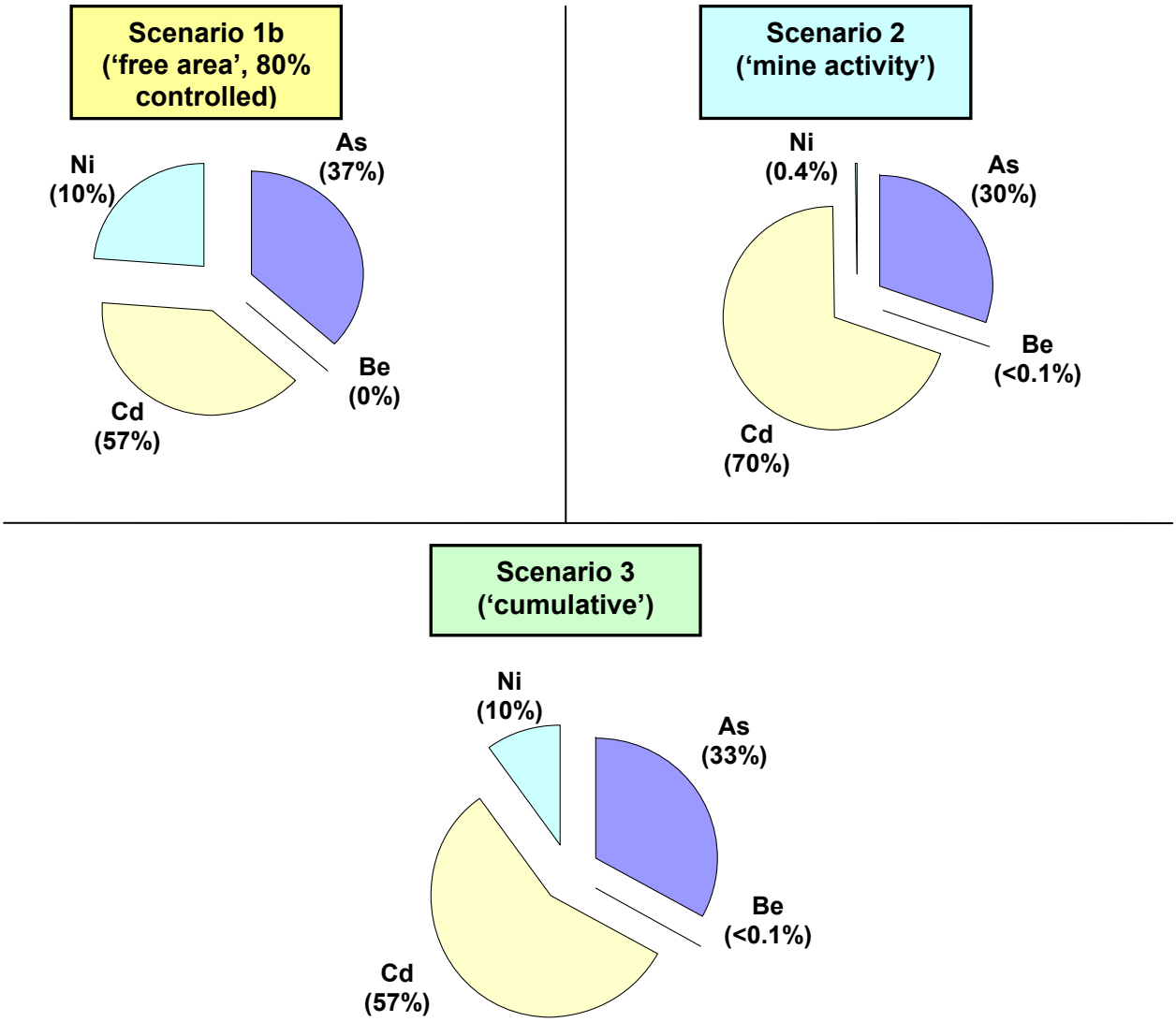


Figure 6.2: Percentage contribution of metals to incremental cancer risk for Receptor 8 by scenario.
Note the As risk includes oral as well as inhalation cancer risk.

7. Uncertainty Analysis

7.1 General uncertainty

In interpreting the calculated risks associated with assumed exposure to emissions from the refinery, uncertainties associated with the assessment need to be considered. The risk assessment process involves a number of steps (e.g. exposure assessment, toxicity assessment and risk characterisation), each of which incorporates the use of assumptions and simplifications to manage uncertainty or lack of knowledge about the 'true' value. Without such assumptions and simplifications it would not be possible to quantitatively evaluate the potential for health effects. Although uncertainties in the risk assessment may influence its accuracy, the assumptions used to cope with unknown data for specific parameters err on the side of safety and therefore bias the evaluation to over estimation of health risk. This is appropriate for an assessment for possible impacts on public health. It must be realised however the conservatism embedded in any one value is at least additive, most times multiplicative, with conservatisms in other values such that the cumulative or compound conservatism incorporated into the assessment can be very large. This is especially so when gross, unrealistic default parameters are used in lieu of measured data.

This section contains a general qualitative discussion of the major uncertainties and their potential influence on the health risk assessment. The 'big picture' uncertainties fall into the following major categories.

- Those associated with exposure estimation.
- Receptor specific uncertainties.
- Contaminant specific uncertainties.

Questions commonly asked of a risk assessment are:

- Are GLCs accurately predicted?
- What is the variability in emission levels?
- Have background exposures been taken into account?
- What is the effect on susceptible subpopulations?
- Is there interaction between emission components for causing health effects?

For the proposed Rasp mine the above questions are addressed in Table 7.1, it presents a listing of the major areas of uncertainty for the refinery emissions only.

Elsewhere in this report, when particular risks or health endpoints are discussed/assessed, information on the uncertainty is provided to enable the reader to integrate the uncertainties with the assessment that has been performed at that point in the report.

In Section 5.4.2, two exposure circumstances have been compared so that the influence of free area dust control could be determined. The scenarios are Scenario 1a, i.e. leaving the existing free areas of the mine site in their present condition for the next fifteen years versus Scenario 3, the exposures predicted to occur with free area dust control (80% efficiency) together with the mine operations. These two sets of exposure have been evaluated using the same parameter values, consequently differences in children's predicted blood lead levels between the two scenarios are largely independent of the actual parameter values that have been applied.

Table 7.1: Uncertainties in the risk assessment for mine dust emissions and potential effect on health risk assessment outcome.

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
Exposure Estimation *From air dispersion modelling as provided to Toxikos by ENVIRON.*	Toxikos has not assessed the information provided by ENVIRON for its representativeness for metal deposition to soil at receptors, or metal content of PM_{10}. However ENVIRON (2010a) note predictions for dust deposition and airborne TSP and PM_{10} are predicted worst case concentrations.	The calculated increase in receptor soil concentrations has a major influence on estimation of lead exposure and blood lead levels. This is based on worse case deposition information from ENVIRON (2010) which therefore results in likely overestimation of incremental soil metal concentrations at the receptor.
	Metal emissions from existing free areas were estimated by ENVIRON (2010) based on the metals profile established from site-specific sampling so it might be anticipated the estimations may be representative. However ENVIRON (2010a) note the 0% control efficiency scenario was overestimated dust deposition rates when compared to on-site dust deposition measurements. It was thought this is likely to be due to sections of the existing free areas having crusted areas with lower erosion potentials.	For lead it is considered the modelling estimations from the free areas are likely to be over predictions (ENVIRON 2010a). Since the free areas are the main contributor to receptor soil concentrations this will tend to over estimate the intakes when characterising the risk via the TDI and estimating increases in blood lead levels. However this overestimation will also decrease the apparent difference in blood lead levels between the 0% free area dust control when compared with an assumed 80% efficiency dust control of the free areas.
	The assumption of 80% efficiency of dust control from free areas is considered by ENVIRON to be reasonable goal when compared to the claims of 95% efficiency by the manufacturers of the dust suppressants.	Free area dust control is a critical aspect of the risk assessment. If the overall efficiency is less than 80% then estimated lead intakes in the risk assessment will be under predicted.

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
Receptor soil calculations	The incremental soil metal concentrations were calculated assuming 15 years of mine operation, deposition into the top 2 cm of soil, and no loss of the deposited lead over the 15 years. These aspects are explored further in Section 7.2.	Smaller mixing depth will increase soil concentrations and a larger one will decrease them. The 2 cm is the default mixing depth used by US EPA (2005) based on measurements of dioxins in soil from incinerator fallout (Section 2.3). The assumption of zero loss of mine related deposited dust will tend to over estimate exposure and blood lead levels.
Background exposures	Data for background (i.e. existing) soil lead concentrations is not available for individual receptor locations. This information has been generated from 2004 – 2008 data supplied by the Greater Western Area Health Services (GWAHS) and transformed into assumed background soil levels according to 'risk zones' in Broken Hill previously defined by GWAHS. This data was not from a systematic random survey but for households which requested soil lead to be measured or where soil lead was measured to investigate the lead source of children with high blood lead. The upper 95th confidence level (CL), or close to the 95th CL, was the assumed background soil concentration. For lead the maximum concentration measured in the reticulated water was coupled with the upper 95th percentile water intake.	The assumed background soil lead concentration while being potentially representative of a small number of receptor locations within a risk zone tends to over estimate the background soil concentration for most. Thus the risk assessment for lead is conservative, i.e. is biased to overestimating risk. Background exposures for other metals was not available. However compared to the TDI for these metals the incremental intake was very small and therefore unlikely to markedly change the existing exposures to these metals. Background intake of lead from these sources (diet and water) over estimate the intake and risk.

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
Intake of lead from existing soil	The background intake of lead from existing soil is an important contributor in the calculations of total lead intake for comparison to the TDI (Section 4) and for examining the influence of free area dust control on predicted blood lead levels (Section 5.4.2). The actual bioaccessibility of lead from the existing receptor location soils at Broken Hill is unknown. The HRA has assumed the bioaccessibility from these soils is similar to that from the free areas of the mine site. Here the basic underlying assumptions are that most of the lead in residential soils has been derived from dusts coming from mine areas in Broken Hill, and the bioaccessibility of lead from other mines is similar to that in dust from the Rasp mine. These assumptions do not consider lead from other sources such as lead paint or past automobile emissions from leaded petrol.	Estimations of total lead intake (i.e. inclusive of background) for comparison with the TDI may have been under or overestimated. While the numerical values for the intakes may change, this does not alter the conclusion that background intake dominates the calculated total lead intake. Or that incremental intake of lead from the proposed mine has negligible influence on existing lead intakes. The predictions of incremental blood lead levels are not affected by this bioaccessibility assumption as existing background soil lead concentrations were not included. The predictions of blood lead levels in the evaluation of dust control benefits (Section 5.4.2) may be under or overestimated. However this section compares the 'benefit' of dust control between scenarios assuming the same existing soil lead concentration and the same bioaccessibility. Hence the relative differences (i.e. 'benefit') between the scenarios will remain even though the blood lead levels may change with a different bioaccessibility.

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
Receptor Uncertainty There may be people within the dispersion area of mine emissions that are more susceptible/vulnerable than others to developing health effects if they are exposed to lead and other metals from the mine site.	For lead, the metal of most concern, public health risk has been characterised by comparing high end take (incremental plus background) to the TDI developed by WHO/JECFA. This has been set to protect the most sensitive individual, toddlers and young children, against increase in blood lead levels. It is widely accepted by health authorities that the effects of lead are well correlated with blood lead levels. Intrinsic variability (i.e. toxicodynamic variability) has not been identified as a major contributor to the effects of lead. When a given population is exposed to the some environmental concentrations of lead, the variability in effects occurs due to variability in blood lead levels. This is the result of behavioural differences between persons resulting in different personal exposures (e.g. extent of time outdoors, extent of hand mouth activity, personal and house hygiene etc).	Impact on the conclusions of the HRA is minimal. Because: 1. Worst case lead air concentrations and deposition have been used, which together with high end background exposure, ensure the most exposed persons is included in the HRA. 2. Worst case and/or high end exposure assumptions were used as input to the IEUBK model. In predicting blood lead levels, the IEUBK model calculates the most likely blood lead level that would occur if a large population were exposed to the same exposures. The IEUBK model also predicts the percentage of such a theoretical population that could have blood lead levels higher than the determined level of concern (10 µg/dL).This is based on the observed blood lead variability, geometric standard deviation, in US children that are in the same lead contaminated environment. There is uncertainty regarding the applicability of the US children blood lead variability to the population of Broken Hill. However appropriate data were not available to make this input parameter site specific. 3. The most impacted receptor locations (R8 & R3) have low risk of exceeding the relevant health guidelines (i.e. the TDI and critical blood lead levels).
Contaminant Uncertainty *Defining toxicological potency of emission components.*	The HRA relies on regulatory guidelines (i.e. TDIs) established to protect public health. These have been sourced from reputable authorities (e.g. WHO) who establish the guideline to ensure the most susceptible portion of the population are protected.	It is unlikely the guidelines used will fail to be protective of all or nearly all individuals. This is the very essence of the philosophy for creating public health guidelines.

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
There may be interactive health effects between emission components.	Regardless of effect or mode of toxicological action, additivity of either dose or effect has been assumed to occur between emission components. See Section 4.6.	This practice causes the HRA to grossly overestimate the risks to combined exposure to emission components. Nevertheless the resulting hazard index at the most affected receptor is about 0.02. Therefore a health effects interaction between the metals in dust from the mine site is considered unlikely. There is minimal effect on the conclusions of the HRA.

7.2 Incremental soil estimations (loss and soil mixing depth)

Loss of deposited lead:

The influence of loss of deposited lead (0, 10%, 25% or 35% per year) has been explored in Figure 7.2.1, for Scenarios 1, 2, and 3. As might be expected soil concentrations are significantly lower when some loss of deposited lead is assumed. In contrast to the assumption of no loss of deposited lead applied in the risk assessment, lead soil concentrations tend toward a plateau signifying steady state between input and loss rather than linearly increasing with time. Moreover the higher the assumed loss of lead from soil, not only are total soil lead levels lower after 15 years but steady state is achieved sooner.

It would appear that a soil loss component of between 1 – 10% potentially has a marked effect on soil lead levels during the life of the proposed mine. Thus the assumption of no loss of lead from soil once deposited is conservative and results in over prediction of lead intake and blood lead.

The depth of assumed mixing of deposited lead with receptor soil has a marked influence on the resulting calculation of soil lead (Figure 7.2.2). The risk assessment has been conducted using the US EPA (2005) recommended default mixing depth of 2cm for untilled soils (Section 2.3). This is based on investigations of dioxin depth profile from emissions from incinerators. In the absence of other information this seems to be a reasonable mixing depth to adopt for this risk assessment. However if in reality the mixing depth is less than 2 cm then incremental soil lead concentrations will be underestimated and the HRA will under estimate the risks. This is however counterbalanced by the assumption of no loss of deposited lead. How these two opposing factors interact is unknown. There is a direct linear correlation between soil mixing depth and blood lead levels, doubling the mixing depth halves predicted blood levels (Table 7.2)

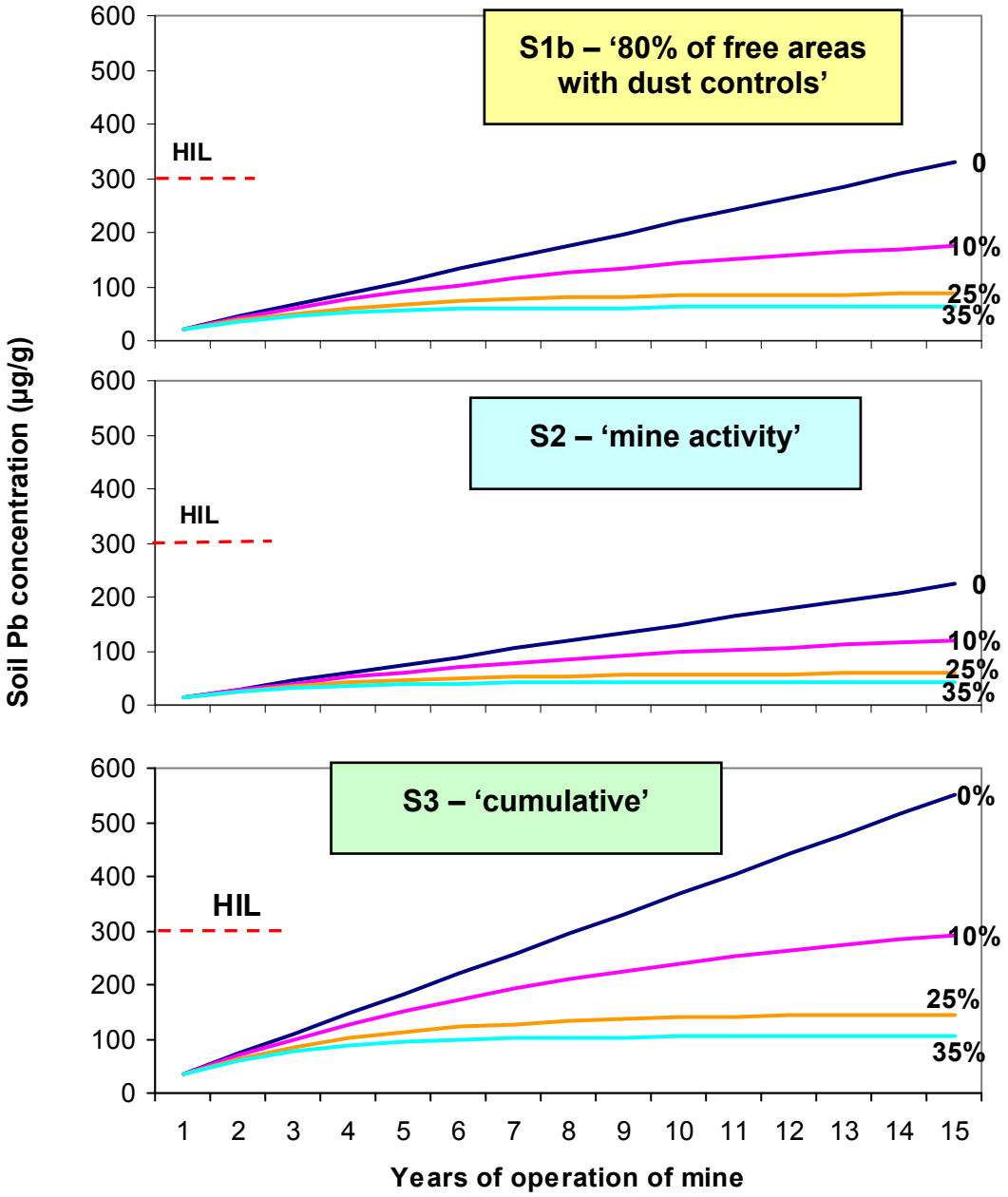


Figure 7.2.1: Predicted soil Pb concentration (µg/g) for receptor 8 assuming 0, 10, 25 or 35% annual loss of soil Pb.

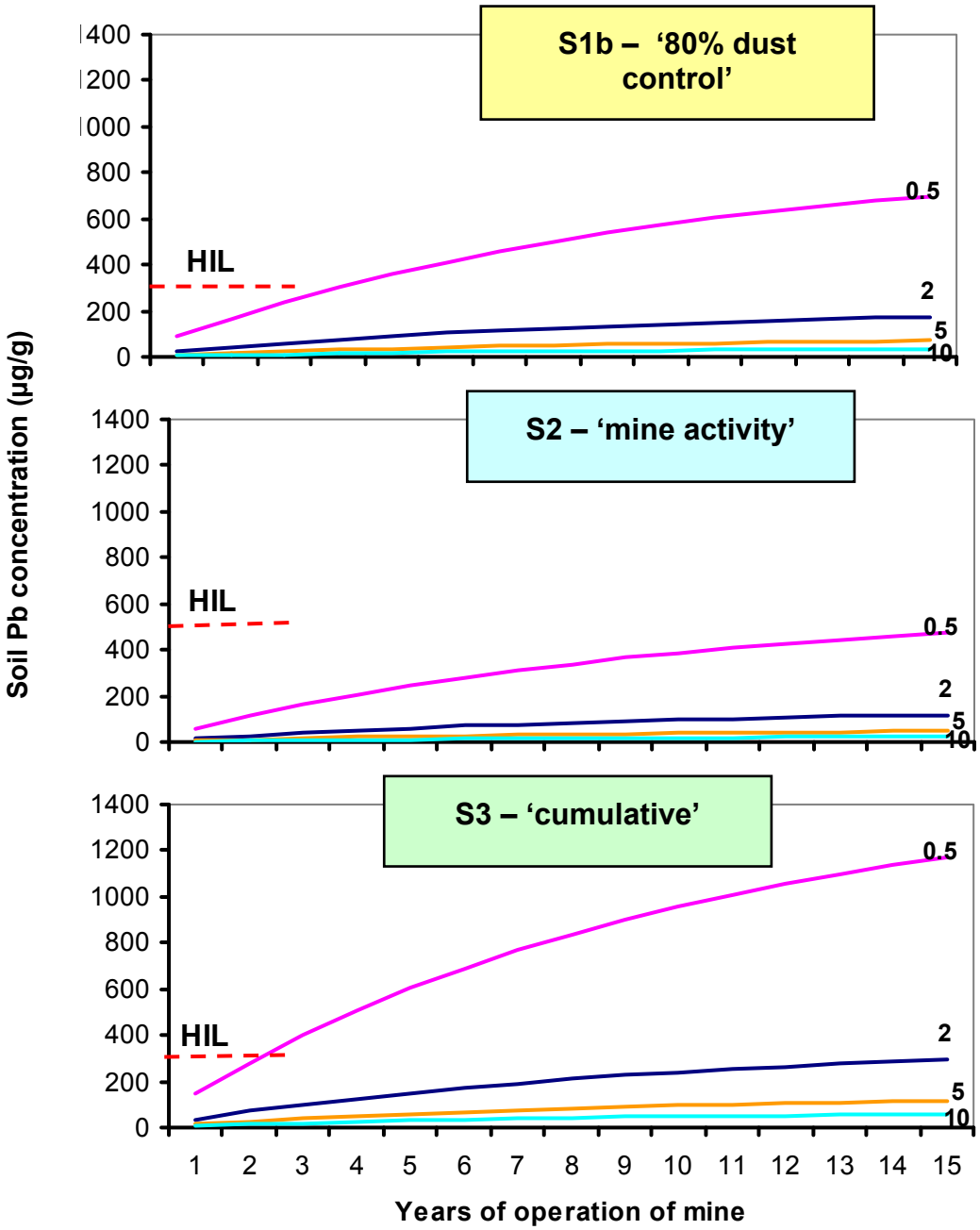


Figure 7.2.2: Predicted soil Pb concentration (µg/g) for Receptor 8 assuming 0.5, 2, 5 and 10 cm mixing depth and 10% loss of annual soil Pb.

7.3 IEUBK

A sensitivity analysis was conducted in IEUBK to determine the effect of altering the ratio of indoor dust to outdoor soil lead on incremental blood lead levels for a 1-2 year old child for Scenario 3. This was done for two receptors: Receptor 8, which is the receptor with the highest predicted annual lead deposition, and Receptor 14, which has a lead deposition closer to the median of all the receptors. Table 7.1 shows there is an increase in predicted blood lead when the indoor dust to outdoor soil Pb ratio is increased. The default value for this parameter in IEUBK is 0.7, this was raised to 1.5 based on, albeit limited, data for Broken Hill.

Table 7.1: Effect of changing indoor dust to outdoor soil lead ratio on blood lead levels in a 1-2 year old child

Ratio of indoor dust Pb: soil Pb	Blood Pb level (µg/dL)	
	R8	R14
0.7 : 1	0.49	0.03
1 : 1	0.59	0.04
1.5 : 1	0.75	0.05
2 : 1	0.9	0.06

Also investigated was the effect of changing the soil mixing depth on predicted blood lead levels for a 1-2 year old child for Scenario 3 at Receptors 8 and 14. Table 7.2 shows there is an inverse linear relationship between mixing depth and blood lead levels. This is as expected since reducing the soil mixing depth increases the soil lead concentration potentially derived from the deposition equation (Equation 2.1).

Table 7.2: Effect of changing soil mixing depth on blood lead levels in a 1-2 year old child

Soil Mixing depth (cm)	Blood Pb level (µg/dL)	
	R8	R14
0.5	2.41	0.2
1	1.46	0.1
2	0.75	0.05
5	0.31	0.02
10	0.16	0.01

Similarly, there is also an inverse linear relationship between the assumed annual percentage loss of soil lead from soil and the incremental predicted increases in blood lead for a 1-2 year old, any loss of soil lead will reduce the amount of lead to which children will be exposed.

Table 7.3: Effect of incorporating different percentages of lead loss from soil on blood Pb levels in a 1-2 year old child

Percentage soil Pb loss per year (%)	Blood Pb level (µg/dL)	
	R8	R14
0	0.75	0.05
10	0.4	0.03
25	0.2	0.01
35	0.15	0.01

8. References

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