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Review of HHRA for Bowdens Silver Mine (SSD 5765)

Reference: DTC270720-RF
11th September 2020

Prepared for:
NSW Department of Planning Industry and Environment

Prepared by:
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About the author:

Dr Roger Drew is the principal consultant of Drew Toxicology Consulting. 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. He has more than 40 years of toxicological and risk assessment experience in academia, industry and consulting. For 12 years he taught medical students at Flinders Medical Centre while undertaking research on the toxicology of chemicals and medicines. He then joined ICI Australia for 10 years as corporate toxicologist managing the Toxicology Unit and responsible for providing advice to the executive team, strategic business units and customers. For the last 20 years he has been an independent consultant servicing a range of industries and Government authorities. He has significantly participated in developing health based risk assessment practice in Australia. Dr Drew is one of just a few toxicologists in Australia certified by the American Board of Toxicology.

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

1. Introduction

Drew Toxicology Consulting (DTC) was engaged by NSW Department of Planning Industry and Environment (DPIE) to undertake a comprehensive review of the human health risk assessment (HHRA) completed for the proposed Bowdens Silver Mine environmental impact assessment (EIS). The scope of the review included:

a) Whether the assumptions used are reasonable, appropriate and suitably justified.
b) Adequacy of the HHRA methodology, analysis and assessment.
c) Identification of any areas of deficiency and recommendations to improve or resolve these issues.
d) Any recommendations (if required) for additional information to inform the assessment of the project.

The proposed mine is 2 – 3 km from the small town of Lue, NSW. There is currently no mining or other industrial activity at the site. To facilitate the HHRA review DTC was provided with internet links to the EIS; the HHRA was attached as Chapter 7. Thus the review has been performed on the HHRA as placed on public exhibition. Other information in the public arena directly relating to the proposed mine has not been accessed. Only material relevant to the HHRA has been read in the EIS.

The Secretary of the Department of Planning and Environment has prescribed environmental impact assessment requirements (SEARs) for the proposed mine. Thus the HHRA has stated objectives of addressing impacts to human health as outlined in the SEARS. These are:

- Air quality (particulates from crustal materials and heavy metals, in particular).
- Drinking water quality (surface and/or groundwater).
- Noise and vibration (including blasting).

The HHRA relies heavily on information provided to the authors by other specialist consultants that have contributed to the EIS; these relate to air quality (provided by dispersion modellers), groundwater, surface water and noise. Most important to the HHRA is the data supplied by the air dispersion modellers. The quality and veracity of the information was not evaluated by the risk assessors, the information was used on face value. This is usual. However it is also common for health risk assessors to liaise with the dispersion modellers with regards to the type of data required to undertake the HHRA. Any methodological shortcomings with the air dispersion modelling and/or inputs to the modelling are directly cascaded into the HHRA; defects in the supplied information mean the HHRA is also defected. Citations for the technical specialist reports from which data in the

HHRA has been sourced are provided in the HHRA. This review of the HHRA has not accessed those technical reports. The HHRA has been reviewed as a 'stand-alone' document; hence accuracy of data transfer from the technical reports to the HHRA has not been checked.

Recommendations/suggestions for addressing various aspects of the HHRA which, in this reviewer's opinion, require attention before the HHRA conclusions can be considered justified are **provided as bold text** throughout this report.

This review uses PM as abbreviation for particulate matter, the element symbol Pb for lead, and BPb for blood lead.

2. Review summary

2.1 What the HHRA has assessed.

There is an appreciated extensive description of the geology and soil, groundwater, and location of properties (including residences) around the mine. Maps and diagrams from the EIS are included to provide the reader with a view of town layout and outlying residences' in proximity to the mine. The demographics and expected health status of people in the town and surrounds is also provided. The latter was assumed to be similar to rural NSW. This is appropriate. It is concluded the area has a higher rate of cardiovascular and respiratory disease than the overall population of NSW, and that this may have some influence on the susceptibility of the population to environmental stressors. This however is not specifically accounted for in the HHRA other than health based guidance values used to compare exposure estimates are established to be protective of all sectors of the population, including persons with higher susceptibility. A table summarising mine related concerns raised by the community has suitably helped shape the HHRA.

The HHRA has assessed the possibility of acute and chronic health effects of dust predicted to be emitted from the proposed mine during four phases of mine development and operation. The assessment has considered exposure to:

- Dust *per se*, i.e. as particulates. This was done by:
    - Referring to the Air Quality Assessment undertaken by Ramboll (2020) in which predicted air concentrations of PM_{10} and $PM_{2.5}$, plus background (i.e. cumulative exposure) were compared against NEPM air quality guidelines.
    - Calculating a risk quotient (RQ) by dividing predicted concentrations of particulates in air by the corresponding air quality guideline.

    - Calculating incremental risk for all-cause mortality from changes in chronic (long term) exposure to $PM_{2.5}$ due to the project. The methodology and exposure-effect functions for this health effect were adopted from WHO.
- Pb from background exposure and from the mine by:
    - Firstly developing separate toxicity reference values (TRV, as an exposure dose) for young children and adults that from modelling by others was associated with blood lead (BPb) levels representing low levels of concern. The information was adopted from deliberations from a UK environment agency (DEFRA).
    - Secondly by comparing Pb intake from a number of exposure pathways (see below) with the developed TRVs to calculate risk indices (RI) for children and adults.
- Various other metals in airborne dust for:
    - Acute exposure by comparison of predicted 1 hour average metal concentration in airborne $PM_{2.5}$ with guidelines sourced from US EPA or Texas to generate acute risk indices (RI's).
    - Chronic (long term) exposure by:
        - Comparison of modelled annual average air concentrations of metals in $PM_{2.5}$ with chronic air guideline concentrations developed from various international agencies.
        - Comparison of metal intakes from specific exposure pathways with health based TRVs sourced from various agencies.
        - Consideration of dust (PM_{10}) deposition to roofs and soil to estimate metal ingestion from water collected in rain water tanks, directly from soil and indirectly from soil via home grown vegetables, beef, milk and eggs. Dermal exposure to metals in soil and tank water was included.
- Hydrogen cyanide (HCN) by comparing acute exposure (predicted 1 hour concentrations) with a health based guideline of the same averaging time from California.
- Chronic exposure to silica in $PM_{2.5}$ based on the silica content of the mined ore. Predicted annual concentrations of silica in $PM_{2.5}$ were compared with guidelines from California and Texas for protection of silicosis.

Risk quotients (RQ) (i.e. calculated exposure ÷ reference value) and risk indices (RI) are provided for individual metals, for specific pathways of exposure and as a total RI for all metals and pathways added together (i.e. the sum of all RQ's). The common terminology for the same calculations are hazard quotients (HQ) and hazard indices (HI's). The overall process is standard for HHRAs and appropriate for assessing potential health risks from the mine.

Depending on the substance and pathway being assessed, the health risk assessments were conducted for all potentially exposed residences (inclusive of privately owned and those owned by the mine), or only for private residences.

2.2 HHRA results

Predicted cumulative (background plus predicted from mine) air concentrations of PM_{10} and $PM_{2.5}$ at privately owned residences are less than the relevant NEPM air quality guidelines.

The incremental risk (from mine emissions only) of dying from chronic exposure to $PM_{2.5}$ was calculated to be 3 $x10^{-5}$. Since this was "*below the unacceptable risk level of 10^{-4} outlined in the NSW EPA Approved Methods (NSW EPA 2016)"* [p7-133] the HHRA tacitly concludes there is no additional unacceptable risk of death from project related $PM_{2.5}$ emissions. The method of calculating the all-cause mortality is appropriate. This reviewer has not consulted NSW 2016, however there is only a 3-fold difference between the calculated and 'acceptable' risk of death. It is also noted acceptable risks of mortality for the general population are usually considered to be 1 in a million (10^{-6}) to 1 in 100,000 (10^{-5}), while acceptable occupational risk is 1 in 10,000 (10^{-4}).

For metal constituents in the dust particles (inclusive of Pb), if a RQ or RI is less than unity (i.e. <1.0) the health risk is considered to be low (described in the HHRA as acceptable). This criterion is a well-accepted bench mark for judging health impact acceptability from exposure of substances released into the environment. The calculated RQ's and RI's all comply with these criteria; some are one to two orders of magnitude lower.

The overall conclusion by the HHRA is "*Dust emissions from the Project would make a negligible contribution to these intakes* [i.e. background intakes] *and there would be no Project-related exposures that are considered to result in any health impacts for any member of the community*" [p7-14].

Comment: By and large the assessment methodology is appropriate. Based on the presented calculations it appears that health risks due to the proposed mine are low. However, and notwithstanding the appropriateness of the overall methodology, **there are numerous aspects (see below) of the HHRA that require attention before agreement with the conclusions of the HHRA can be made.**

2.3 Overview of issues

Problems with the HHRA mostly deal with data selection but some also with application of assessment methodology. These are broadly indicated below with details provided in Section 3 of this review.

a) The HHRA does not have the level of transparency in data selection or justification of assumptions that is expected, or required, if the reader is to readily assimilate the information and conclusions. This lack of clarity percolates through the detailed comments provided in Section 3. Particularly frustrating is the absence of dust and Pb dispersion maps and in the main text, lack of explicit description of the modelled data used in calculations (this stems from the absence of summary lists of contaminant concentrations predicted by the air dispersion modelling).

b) Exclusion of assessment of health risk at project-related residences (i.e. mine owned dwellings) for some substances and pathways is a concern.

c) Notwithstanding compliance with regulatory air quality guidelines, assessment of health risks due to airborne particulates from the mine is restricted to assessment $PM_{2.5}$, at private residences, to the health impact of death, and only to incremental increases. For some pathways and substances exposure to TSP and PM_{10} is also pertinent.

d) For metals there is also exclusion of project-related residences. In the absence of any indication these will be demolished, it is presumed by this reviewer these may be occupied by adults and children and therefore should be included in the assessment(s).

e) It is noted some existing media samples have metal concentrations higher than health based guidelines. The number and location of these ‘existing’ exceedances is not provided. It is therefore not possible to discern whether these are locations which may also have additional higher impact from mine emissions.

f) The use of a TRV for assessing exposure to Pb is questionable in the face of there being no identified threshold for some of the health impacts of Pb exposure. There are many questions relating whether use of a TRV is compatible with Australian public health objectives and also with the derivation of the TRV undertaken. This reviewer disagrees with some aspects and has offered, with justification, alternative inputs to the TRV derivation.

g) The risk measure (RQ) for exposure to airborne Pb appears to have been miscalculated.

h) For Pb, the overall RI is the sum of RQs. But the RQ for inhalation exposure is calculated differently than the other HQs and, in this reviewers opinion, the overall RI is not properly determined.

i) Noting the health effects of Pb are linked to BPb levels and public health policy is also based on BPb, It is this reviewer’s opinion the calculation of an incremental RI for Pb makes it difficult to determine whether there is, or is not a meaningful increase in BPb in adults or children from mine operations. It is strongly recommended incremental BPb be determined.

j) Some exposure pathways of metals are dependent on deposition from air to roofs and soil. The dust size used for this is PM_{10} despite it being clear that some residences are subject to TSP from the mine. Thus exposure to metals, including Pb, has been potentially underestimated.

k) International guidelines for crystalline silica do not appear to have been appropriately applied in the risk assessment. Also only silicosis is addressed, not other potential health effects.

3. Detailed comments

3.1 Health risks from particulates per se.

1. The HHRA uses the term 'inspirable' for particles around 10 microns and larger, and throughout the document 'inhalable' when discussing $PM_{2.5}$. However, more commonly applied terminology for particulate matter (PM) fractions is inhalable for particles ≤100 µm, thoracic for ≤10µm (PM_{10}) and respirable for ≤2.5 µm ($PM_{2.5}$). These size fractions correspond to regions of the respiratory tract where the majority of particulate size deposition occurs. 'Inhalable' particles can enter the nose, mouth and upper trachea (i.e. the extra-thoracic region), 'thoracic' particles can penetrate into the respiratory tract below the larynx including larger bronchioles (i.e. the nasopharyngeal/tracheobronchial region), and 'respirable' particles are those that penetrate into the alveolar part of the lung (CEN 1993). TSP is defined in NSW as particles ≤50 µm but other jurisdictions, e.g. Queensland, consider TSP as ≤100 µm. The particle size measured by the two TSP monitors in the vicinity of the mine is not provided; this depends on the fitted sampling heads. **It would be useful to have this information**. Notwithstanding regulatory, or environmental vs occupational, definitions for airborne particulate size it is well recognised that particulates of around 10 - 100 µm can enter the upper respiratory tract and penetrate down to the bronchiolar level. Inhalability of particles decreases as size increases, from ~77% for 10 µm to a plateau of 50% for sizes ~40 – 100 (US EPA 2019, Witschger 2006). **Therefore confining the HHRA to only $PM_{2.5}$ misses possible health effects from larger particulates.** The HHRA indicates at p7-33 "*that chronic diseases considered generally relevant to the assessment of health impacts related to coarse particulates* [i.e. those bigger than 2.5 µm] *and noise from mining activities include hypertension, heart disease, stroke and respiratory disease (including asthma)*". **With respect to particulates these health effects are not included in the HHRA.** It is noted the HHRA concludes the community around the mine has a higher prevalence of cardiovascular disease and hypertension than NSW in general.

2. The HHRA dismisses TSP as being of health concern, rather it described as "*more of a nuisance than a health hazard*" (p7-66). This reviewer agrees the weight of epidemiological

evidence supports the conclusion that the serious PM health effects are mostly associated with the fine particulates in air, i.e. ≤ $PM_{2.5}$, and it is appropriate to focus on this size fraction for chronic health effects. However the coarser particulates in air are not without health effects. They contribute to eye and upper respiratory tract irritation, and potentially to induction/exacerbation of respiratory and cardiovascular effects. This reviewer believes these are more than just 'nuisance' effects and **particulates > 2.5 µm should be included in the health effects assessment of particulates** (Section 5.2.2) even if statistically significant causal associations have not (yet) been established.

3. Although particle size is not indicated in Section 5.2.2.3 when discussing health risk of metals on particles (p7-69), inspection of the calculation spread sheets (p7-278 & p7-280) reveals only $PM_{2.5}$ is used. The rationale for excluding TSP and PM_{10} for assessing direct health effects from particulates or metals on/in the particulates is "*they would deposit out of the air (measured as deposited dust) close to the source and, if inhaled, are mostly trapped in the upper respiratory system*". It is agreed that $PM_{2.5}$ will travel further than other particulate sizes in TSP however no information is provided showing there will not be incremental increases in mine related TSP at locations of interest to the HHRA close the proposed mine. Indeed it is noted that:
    - For calculation of metals in tank water deposited PM_{10} is used. Indicating there are incremental increases in exposure to particulates larger than $PM_{2.5}$.
    - It is noted from the EIS (Fig 4.4.7 therein) that TSP from the project extends to some residences (both private and project related).
    - Acute guidelines from TCEQ are meant to be applied to PM_{10} and not $PM_{2.5}$ as in the HHRA.

    In this reviewer's opinion there is compelling evidence in the EIS that exposure to particulates bigger than $PM_{2.5}$ are expected to occur at residences and therefore their consideration should be included throughout the HHRA.

4. Figure 4.7b (p7-58) shows the 24-hour average $PM_{2.5}$ concentrations for 2017 measured by a monitoring station in Lue. Curiously $PM_{2.5}$ is lower in winter (June – August) when, in a country town, it would be expected wood heaters would be extensively used. The possible explanation of higher rainfall in winter is not viable because the wetter months are December and January. **This anomaly deserves comment.**

5. The HHRA is correct that inhaled large particles will be trapped in the upper respiratory tract and mostly swallowed (Footnote 10, p7-66), from a particle size perspective this particle ingestion does not present harm. However when considering exposure and intake of metals that have systemic effects, particularly Pb, this is a contributing pathway that has not been considered in the HHRA. Indeed for Pb, WHO (1999) considers the inhalable fraction (i.e. < 100 µm) are the particulates of interest (the HHRA only considers $PM_{2.5}$). **Despite acknowledging "*the digestive tract can also be an important avenue of lead absorption following inhalation*" (p7-139) this is not included in the Pb assessment.**

6. At p7-69 it is stated "*This health endpoint* [all-cause mortality] *captures all other health effects found to be causally related to $PM_{2.5}$ exposure and is the most significant in terms of calculating risks related to changes in $PM_{2.5}$ exposures".* Death is certainly is at the top of health effects. But **how does this capture the potential health effects that do not result in death?**

   In Annexure A (p7-127) it is stated "*The health effects associated with exposure to particulate matter vary widely.."* . Notwithstanding that evidence is strongest for mortality, for which exposure-response modelling is easiest to undertake, and mortality is what air quality standards are based upon, **is this reviewer's opinion that health effects other than mortality be addressed in the HHRA.** The possibility of non-lethal pulmonary/respiratory effects occurring in children is an important concern.

   In addition the HHRA does not have a discussion of risk factors that show/suggest an individual is, or is not, at greater risk to experience health effects from exposure to airborne particulates.

7. Also at p7-69 the maximum incremental risk for death to exposure to mine related $PM_{2.5}$ is reported, for "*privately-owned residences",* to be below the risk level considered to be unacceptable by NSW EPA, and therefore this risk of death is deemed acceptable. The receptor for the maximum incremental mortality risk is not identified. This reviewer could not find in the HHRA incremental risk calculations for other receptors, in particular for residences owned by the mine. It is noted maximum $PM_{2.5}$ air concentrations for <u>all receptors</u> is used for determining acute (p7-278) and chronic (p7-280) risk from metals on $PM_{2.5}$, it is therefore puzzling why incremental risk due to $PM_{2.5}$ *per se* in the HHRA has not included project-related residences as well as privately-owned residences. Similarly only private residences are considered when calculating metal concentrations in soil (p7-281) and therefore all soil related exposure pathways (ingestion and dermal), also only private residences are included

for calculating metals in rainwater tanks (p7-286), uptake into edible plants (p7-291) and eggs (p7-294). **It is recommended all residences, both private and project-related, be included in all parts of the HHRA.**

8. This reviewer agrees with the methodology adopted by the HHRA from WHO for assessing the mortality risk from $PM_{2.5}$ and the use of local data for baseline all-cause mortality. However the averaging time, presumably annual, for the incremental increase in PM concentration used in the calculations should be provided with the equations. Also the source of the annual $PM_{2.5}$ increment used in the calculation in Annexure A is not cited, nor is it indicated if it is the maximum within the modelling domain, and there is no information on increments elsewhere around the mine. **This hampers appreciation of the overall risk profile from PM exposure for the local population and should be addressed.**

9. Furthermore the cumulative risk (existing 'background' plus incremental) from PM exposure is not provided. Hence it is not known to the reader if the 'acceptable' risk for increase of death is on top of an already high risk for all-cause mortality from $PM_{2.5}$. **It is recommended this be addressed.** The risk benchmark chosen by the HHRA is 1 in 10,000 and NSW EPA is cited as the source. **It is recommended this risk level be explained in context of** generally accepted risk levels of 1 in 1,000,000 to 1 in 100,000 for the general public.

10. In Annexure A (p7-133) it is stated *"The calculation* [for incremental risk of death] *is also considered conservative as the air modelling has adopted conservative assumptions, in particular rainfall, which would reduce dust emissions on wet days and increase wet deposition*." This is paradoxical, the dispersion modelling has assumed dust control measures (using water) will be in place, if these controls are effective as assumed in the modelling then rain should not significantly reduce dust emissions from the mine, e.g. from road haulage. While rain will increase wet deposition this should only apply to background (non-mine) particulates and should not affect the calculation for incremental risk. **This reviewer does not consider the offered reason for conservatism in the calculation for incremental mortality risk is valid.**

11. Maps of groundwater bores and water dependent ecosystems, of rainwater tank sampling, and for the project air quality monitoring network are provided. But contour maps of dust concentrations for TSP, PM_{10} and $PM_{2.5}$ predicted in emissions from the project are inexplicability not included. This markedly hampers appreciation of exposures. **Dust contour maps should be included in the HHRA and discussed in relation to the background status of receptors (especially those closer to the mine).**

3.2 Pb health risk assessment

1. Bioaccessibility:

   The HHRA provides an understandable summary of bioaccessibility and bioavailability (p7-71), this is expanded in Annexure B. It is agreed site specific bioaccessibility should be used in the HHRA to adjust the bioavailability when calculating intake (absorbed dose).

   - Oral bioaccessibility was determined from soil samples taken from14 locations from the mine lease; 12 were surface samples (0 – 5 cm) and at 2 locations three samples were at greater depth (5 – 15 cm, n = 2, and 30 – 60 cm, n =1). For one location there is a hint that bioaccessibility is lower at greater depth (18.6%, 14.6% vs an average for all surface samples of 35.4%) but this was not observed for the other sample taken at 5 – 15 cm (44.4%). Thus it is uncertain if the bioaccessibility of Pb from ore at depth is different from bioaccessibility of Pb in surface soil where it may have been oxidised. The latter being more soluble. It is therefore appropriate that the average bioaccessibility for particle size <250 µm from all samples (33%) has been applied in the HHRA. It is likely this overestimates the bioaccessibility of mined rock and ore, but perhaps not if they are left on the surface and the Pb becomes oxidised.
   - It is noted that bioaccessibility from smaller particles, as expected, is greater than from larger ones. This is evinced by the testing for surface particulates of size <2 mm (5.5% bioaccessibility) but for <250 um the bioaccessibility is 35.4%. It might be expected bioaccessibility for smaller particulates (PM_{10} & $PM_{2.5}$) may be higher than the average used for $PM_{<250}$. This might result in underestimation of bioaccessibility of ingested PM_{10} and $PM_{2.5}$.
   - Bioaccessibility was determined by Uni SA with an *in vitro* dissolution method that mimicked stomach fluid. Although the method can also incorporate mimicking intestinal fluid, only the gastric phase of the test was done. Pb is absorbed from the small intestine, not the stomach. Pb dissolves in gastric acid (pH ~2) but solubility at intestinal pH (~8 – 9) is much less and some of the Pb solubilised in the stomach drops out of solution in the intestine and is not available for uptake into blood. Thus Pb solubility data from the gastric phase of the test overestimates the actual bioaccessibility of Pb in soil. **This is conservative for the HHRA. It would be useful if a discussion such as above were included in the HHRA.**

2. Use of TRV for Pb:

The HHRA (Annexure B) has a good discussion of the disposition in the body and effects of Pb. The HHRA correctly reports that a tolerable daily intake (TDI)[1] for Pb was withdrawn by WHO, and by many other international organisations. This was because a population threshold for neurological deficits in young children could not be identified. The critical effects from Pb in adults are cardiovascular and renal. It is also acknowledged in the HHRA there are other health effects associated with Pb exposures but data is not as strong for those. The adverse effects of Pb in children and adults correlate with blood lead levels (BPb). Hence public health goals, such as from the NHMRC in Australia and international organisations, are articulated in terms of target BPb levels. As acknowledged in the HHRA, these are not bright lines between safe and unsafe. In other words there is a risk of adverse health effects from any exposure to Pb that increases BPb over background levels.

Notwithstanding the above, the Bowdens Silver Mine HHRA has developed a TDI for Pb in children and adults in terms of dose (µg/kg bw/d). It is based on BPb modelling presented[2] by the UK Department for Environment, Food and Rural Affairs (UK DEFRA) technical consortium team who provided the support documentation for regulatory derivation of acceptable Pb in various soils in the UK. **The HHRA does not contain discussion/rationale regarding the applicability of the DEFRA deliberations on Pb to the Bowdens HHRA.** Below are comments on the DEFRA technical support document (UK DEFRA 2014a) and adoption of information from it in the HHRA.

a) What is not recorded in the HHRA, and generally unappreciated by many risk assessors, is the dose-response for loss of IQ points in children is steeper at low BPb levels than at higher levels. Thus there is legitimate concern for even quite small increases in BPb potentially occurring from non-background (non-dietary) exposures. It is a public health goal in Australia to eliminate or minimise unnecessary exposures to Pb; this is acknowledged in the HHRA.
b) The HHRA has minimal description of the UK DEFRA process(es). The narratives below are this reviewer's summary and interpretation/applicability of the DEFRA work to the HHRA. DEFRA mathematically modelled BPb concentration and effect (i.e. response) data to establish a Low Level of Toxicological Concern (LLTC) for BPb in children and adults. This modelling used benchmark dose (BMD) software freely available from US EPA to determine the LLTC. LLTC is a term unique to DEFRA but is operationally used by DEFRA to establish 'allowable' concentrations of substances,

[1] The TDI is the toxicity reference value (TRV) for substances that have a threshold mode of toxicological action.

[2] The information in the cited DEFRA document appears to be largely from a review and modelling undertaken by EFSA. The EFSA work has not been interrogated by this reviewer.

not just Pb, in soil in the same way as a No Observed Adverse Effect Level (NOAEL) or BMD could be used. The process tacitly assumes a threshold, or at least an acceptable incidence of adverse effect in Pb exposed populations (see below).

c) For Pb, the LLTC is a BPb level corresponding to a certain incidence of adverse effect. The selected incidence is different for different effects and is one that the data curve fitting can accommodate. Thus the LLTC is the BMD (as a BPb level) that is associated with the adverse effect at the selected incidence level in an exposed population. An interpretation is that it represents an 'acceptable' incidence of effect[3]. The DEFRA process takes this 'target' BPb, after applying a safety factor, and converts it to a 'target' dose (exposure intake) of Pb (i.e. a TDI equivalent) using different software developed by US EPA and also available free. The factor applied by DEFRA was 1, i.e. no safety factor[4]. The LLTC when converted from a BPb (µg/dL) to a dose of Pb (µg/kg/d) is the TRV used in the Bowdens HHRA. **It is this reviewer's opinion the selection of adverse effect, effect incidence level, and appropriateness of mathematical model are health/science policy decisions that should be informed by the appropriate authority, or at least discussed/justified in the HHRA.** Note rationale for alternative selections to those adopted in the HHRA is provided below.

d) The modelling steps used by DEFRA are 'state of the science' procedures for dose response assessments. While these processes were originally designed for substances with threshold mode of action they are also amenable to non-threshold substances if cognisance is applied to their use. Conceptually the DEFRA process is applicable to HHRAs that include Pb exposures. However, where the public health goal is for no, or only insignificant increases in child and adult BPb from non-dietary Pb exposures, the measure of the objective (i.e. change in BPb) is obfuscated by using a TRV based on dose. **It is strongly recommended BPb modelling also be undertaken in the HHRA.**

e) Notwithstanding the DEFRA processes may be applicable to assess the health risk from Pb exposure it is this reviewer's opinion options from DEFRA, other than the ones selected by the HHRA, are more applicable and compatible with Australian public health goals for Pb. The HHRA states (p7-147) "*For this assessment it is appropriate to adopt the value* [for LLTC] *of 3.5 µg/dL*." The only justification offered

[3] A BMD_{01} and BMD_{10} (as BPb, µg/dL) respectively represent an incidence of effect in 1 and 10 persons out of 100 that have that particular concentration of Pb in their blood.

[4] DEFRA states "...[although] *a margin of 1 has been included for each endpoint. It could be argued that margins of up to a maximum of 10 could be used. There are no precedents as to what generic margins might relate to low risk for these non-cancer non-thresholded endpoints. Therefore, there is no generic guidance (akin to nominal ELCRs) that could be applied here for lead*." See also relevant parts in comment (g) below.

in the HHRA for this target BPb is the BPb modelling in the cited DEFRA document used software (IUBEK) that has also been used in Australia. **Reasoning for adoption of this particular BPb as a target should be included in the HHRA.** It would be expected this would centre on applicability to the Bowden HHRA.

f) DEFRA presented results from BMD modelling for three of the endpoints (neurobehavioural in children, adult renal toxicity and hypertension in adults) associated with BPb levels. The lowest of these was a $BMDL_{01}$ of 1.2 µg/dL for neurobehavioural effects in children from the EFSA deliberations.

From the available modelling three options for the LLTC (the target BPb) were put forward to DEFRA for consideration for derivation of acceptable Pb in different soils.

- 1.6 µg/dL (BMD_{10} for adult renal toxicity).
- 3.5 µg/dL (slightly lower than $BMDL_{01}$ for adult hypertension, lower than the median BMD_{01} from all models for child neurobehavioural effects, and approximately the BMD_{20} for adult renal toxicity).
- 5 µg/dL as a policy choice compatible with US CDC.

For selection of the LLTC, the consortium technical team undertaking the work made it clear that significant risk management choices based on policy needed to be made. They therefore did not make a recommendation for which of the LLTC choices they put forward should be adopted. DEFRA (2014b) made a policy decision to use 3.5 µg/dL as the LLTC. It is noted the DEFRA work relates to setting 'Category 4 Screening Levels' in soil[5]. In doing this DEFRA balances the potential adverse effects that regulatory intervention[6] may have against the risks of the contaminant in soil to human health. Soil screening levels apply to existing contaminants in soil and are not intended to be levels to which new, or existing industry are permitted to pollute up to. **It is therefore questionable whether the policy deliberations of DEFRA are strictly applicable to proposed mining projects in Australia. Advice should be sought on this**.

g) Given the non-threshold nature of Pb neurobehavioural effects in children this reviewer considers a BMDL (the lower 95% confidence limit on the modelling fit to the data) is more appropriate than the central tendency of the data modelling (i.e. the

[5] Category 4 soil screening levels are concentrations of a contaminant in soil that present acceptably low levels of risk for the soil use(es).

[6] These include property blight, anxiety and stress related health impacts, mobilisation of contaminant during remediation, environmental impacts of remediation, cost of investigation and possible remediation.

BMD) for the critical effect(s). Also a low incidence for neurobehavioural effects is appropriate. The lowest incidence from BMD modelling available for this end point from the DEFRA document is 1%. Since the neurobehavioural impacts in young children is higher at low BPb (steeper dose response) the piecewise linear model is more appropriate than logarithmic or linear models.

Should derivation of a TRV for Pb exposures be accepted for use in the HHRA, this reviewer recommends the $BMDL_{01}$ for child neurobehavioural effects of 1.2 µg/dL as the target BPb (µg/dL) for conversion to a TRV (µg/kg/d).

This BPb is the same as that recommended by EFSA for characterising risk[7] to children from Pb exposure. If used as a target for incremental Pb exposure from the mine, the suggested $BMDL_{01}$ is compatible with keeping BPb in children as low as possible. The combined aspirational BPb, from dietary background exposures (< 2 µg/dL) plus the incremental target BPb, for the HHRA would be 3.5 µg/dL. This is also consistent with the goal of minimising any additional increases in BPb over background.

For most substances an uncertainty (safety) factor of 3 would be applied to the BMDL to account for variability between humans not embedded in the BPb-effect modelling, i.e. kinetic differences. However this would result in the target BPb being markedly below current background concentrations for children (< 2 µg/dL) and not realistically attainable. Not applying an uncertainty factor to the point of departure is an additional reason to use the $BMDL_{01}$ rather than the BMD_{01} or BMD_{10}

DEFRA notes 1.2 µg/dL is the value chosen by the EFSA CONTAM panel as a reference BPb level in the context of Margin of Exposure assessments. DEFRA indicates EFSA stated "*protection of children against the potential risk of neurodevelopmental effects would be protective for all other adverse effects of lead, in all populations*." The modelling presented in UK DEFRA (2014b) gives the lowest BMD for adults as 1.5 µg/dL as a $BMDL_{10}$ for renal toxicity. **It is therefore recommended by this reviewer that 1.2 µg/dL, as a target BPb, be applied to both children and adults, for calculation of a TRV (should a TRV be accepted as an acceptable measure for Pb exposure).**

[7] By 'Margin of Exposure' method.

h) Converting the target BPb into a dose (µg/kg/d) was done by DEFRA using the IUBEK and ALM, respectively child and adult, BPb models from US EPA in reverse. The above suggested BPb target (1.2 µg/dL) is not one of the BPb levels converted to a dose by DEFRA. The relationships between BPb and intake/uptake in Figure 2.4 of the DEFRA document can be used, as done by DEFRA. For reasons not explained, DEFRA chose the relationship for Pb dietary intake (µg/kg/d) to determine the mean BPb concentrations (µg/dL) in children that would equal the target BPb levels listed above. This relationship was also used, without justification, in the HHRA. **It is unclear why the BPb-dose relationship for uptake (from all pathways) should not be used in the Bowdens HHRA multi-pathway exposure for Pb exposure.** This is an increase of 5 µg/dL blood lead per µg/kg bw/day uptake vs ~2 µg/dL per µg/kg/d for the dietary relationship.

3. Calculation of Pb in environmental media

a) The RQ for inhalation exposure is made using Pb in $PM_{2.5}$ for comparison with the NEPM air quality guideline (p7-278 & p7-280). However, the Federal Register of Legislation (Aust Gov 2016) on the NEPM Air Quality Measure states at Schedule 2 "*Measurement of lead must be carried out on Total Suspended Particles (TSP) or its equivalent*". Thus comparing predicted Pb in $PM_{2.5}$ with a guideline for TSP means **the RQ for exposure to airborne Pb has been miscalculated.**

b) Exposure pathways for Pb that are dependent on deposition of Pb from air have used Pb deposition to roofs and soil from PM_{10}. Where residences are predicted to experience TSP from mine activities, Pb in TSP should be used. **Pb exposures from pathways reliant on deposition are potentially underestimated.**

4. Calculation of overall risk index for Pb

For Pb exposure pathways, other than inhalation, individual pathways RQs are calculated by dividing the intake for the pathway by the TRV. The RQ for the inhalation pathway is calculated (inappropriately see above) by dividing the predicted air concentration (in $PM_{2.5}$) by the NEPM air quality guideline. The overall RI is the sum of all the HQ's. However a very different inhalation HQ is likely obtained if intake of Pb from inhaled particulates is compared to the TRV. **Since the inhalation HQ in the HHRA is calculated differently to the other pathways HQs (air guideline or Pb TRV at denominator) it is erroneous to add them together to give a RI.**

3.3 Assessment of metals in tank water

1. p7-50: What percentage of rainwater tanks have filters that may remove metals from the water?

2. On p7-51 it is listed that water samples prior to tank cleaning, sediment samples during tank cleaning, and water samples post cleaning were obtained from 84 water tanks in the immediate area of the proposed mine. **The following should be clarified.**
    a) Table 4.4 (measurements of metals in tank water) does not indicate if the tank water data is pre- or post-cleaning.
    b) Blue text in the table signifies exceedance of a drinking water guideline but the number of exceedances is not shown.
    c) It is not indicated in the HHRA if tanks with existing exceedances of drinking water guidelines are at locations impacted by dust deposition onto roofs, or if these found exceedances resulted in the tanks being cleaned.

3. The calculations for 'existing' exposure to metals in rainwater tanks in Annexure F (p7-271 & p7-272) use lower water concentrations than are provided in Table 4-4 (p7-51). The data in Table 4-4 are presented as a summary of measurements obtained from the tank water sampling campaign. There is no indication in the HHRA that these measured metals concentrations should be adjusted for assessing risk. Nor in the Annexure is the data source for tank water metal concentrations provided. **These issues should be addressed.**

4. Units in the equations on p7-264 don't cancel.
    a) Volume of rainwater (VR) from the equation is m^3 (currently no units for VR in Table E6). Nevertheless it appears this has been converted to L prior to undertaking the calculations (p7-286). Suggest 10^3 be added to numerator of the VR equation and VR units in Table E6 indicated as L.
    b) A unit conversion of 1000 cm^3/m^3 is missing from the C_W equation. No units for C_W in Table E6.

5. p7-286 . The mass of dust deposited to roofs appears to have been calculated in the HHRA as only derived from PM_{10}. Surely there are larger dust particles from the mine, and general area, that will be in the air and be deposited on soil and roofs (especially closer or downwind from the mine). Intuitively, just using PM_{10} as the source of dust will underestimate the deposited dust and metal, and hence also in tank water. This will have greater impact for Pb and Zn than other metals as these are the metals with the highest current deposition rates. Furthermore, inspection

of TSP contours provided in the EIS clearly show residences are impacted by TSP (Figure 4.4.7, p4-93). **It is recommended deposition calculations be based on TSP.** It is also noted that larger particles may not only provide higher mass of metal but relative to PM_{10} the proportion of metal in the particle may also be different.

6. It is noted the deposition rate (DR) in calculations for metals in tank water is different to that used for calculating soil concentrations. For Pb the deposition to soil is given as 0.7667 mg/m^2/yr and to roofs as 0.1793 mg/m^2/yr. Both are presented as the maximum for a private residence thus the same location, surely the deposition rates should be the same for roofs and soil. In Figure 5.4 (p7-80) ingestion of soil and water are respectively the second and third largest contributor to overall risk. **This difference needs to be reconciled.**

7. It is noted Kd (soil: water dissociation constant) for estimating the amount of dissolved metal in tank water are sourced from US EPA RAIS. It is further noted that Kd for Pb and Li are more than an order of magnitude higher than for other metals. Since Kd is in the denominator for calculating the tank water metal concentrations a higher Kd means a lower water concentration. **A rationale for adopting the Kd's from RAIS in this risk assessment is encouraged to be provided.** Particularly for Pb since tank water accounts for a high proportion of the total project related Pb exposure for children. It would be expected that the requested rationale address soil type, plus the form and source of lead.

3.4 Acute inhalation guidelines.

Section 5.2.2.3, p7-69

1. For some metals Protective Action Criteria (PACs) from the Office of Environment, Health, Safety & Security, US Department of Energy (DOE) have been adopted by the HHRA as inhalation guidelines for assessing possible health effects from direct acute exposure to mine air emissions. They have 1 hr averaging times. However, effects (e.g. sensory irritation) from airborne exposure to chemicals or particulates may be experienced within a much shorter period than 1 hour. Also fluctuations of airborne concentrations can vary within an hour; with concentrations being higher or lower than the 1 hour average. Thus the 1 hour average of the adopted guideline does not match the exposure time eliciting the effect, and is arguably not an assessment of "*peak short-term*" exposures as claimed in the HHRA (p7-69). It is this reviewer's opinion, notwithstanding US DOE regulatory guidance for use of PACs, that the 1 hour acute inhalation guidelines for assessing the likelihood of experiencing the acute health effects described in the assessment ("*discomfort, irritation, or certain asymptomatic, non-*

sensory effects") (Table 5-1 footnote) are not necessarily "*protective of adverse health effects from exposure to these pollutants within the general population, including sensitive individuals*" (p7-69) as indicated in the HHRA. The 1 hour average concentration is one of convenience since this is what is commonly provided by the air dispersion modellers.

It is suggested the 'acute' health effects should be assessed against peak concentrations within the 1 hour average. These can be provided by the air dispersion modeller for periods nominated by the health risk assessor (e.g. 10 min), or the 1 hour average already provided could be adjusted with assumed/justified peak:mean ratios, or by Turner's power law.

Also, this reviewer also does not know what "*asymptomatic, non-sensory effects*" may be. This should be clarified (Footnote to Table 5.1).

2. For other metals, 1 hour 'acute' air guidelines from the Texas Commission on Environmental Quality (TCEQ) have been adopted. TCEQ has produced detailed support documents which, from the citations in footnotes to Table 5.1, the authors of the HHRA appear to have accessed. However there is no discussion of the basis for these acute guidelines in Annexure C (Toxicity Summaries for other Metals). **It would be appropriate to at least provide the endpoint that each TCEQ guideline addresses and how the 'point of departure' was manipulated.** Sensory irritation of eyes and upper respiratory tract may be due to the particulate nature of the metal and therefore independent of the actual amount of metal (soluble?) in the particulate. The same applies to PAC guidelines, which are rebadged AGELs, EPRGs or TEELs from US agencies that usually have documentation containing the health effects addressed by the acute guideline.

3. This reviewer has not accessed the TCEQ documents however footnotes to Table 5.1 of the HHRA indicate "*For metals these* [guidelines] *relate to concentrations in particulates <10 microns in size*". However it is apparent the calculations for the acute hazard index (HI) are made using air concentrations for the metal in $PM_{2.5}$ rather than PM_{10} (p7-278). **This appears to be misapplication of TECQ guidelines.**

4. The acute and chronic guidelines adopted in the HHRA assume thresholds exist for the health effects potentially induced by the substances assessed. At p7-69 it is stated in relation to the acute and chronic guidelines "*Where exposures are below these thresholds, no adverse health effects would occur*." This is generally the case, or at least adverse health effects are not expected. However, **it needs clarifying** that the health effect (whether

adverse or not) for which the 'acute' 1 hour guideline is assumed to offer protection assumes a single exposure only. It only pertains to the averaging time linked to the guideline, i.e. for effects that might occur within that exposure time or soon after. It does not necessarily offer protection for either the same effects, or other effects, that might occur with multiple single exposures if they are frequent during a day or over several days. 'Acute or short-term exposure' is defined in the HHRA (p7-9) as "*Contact with a substance that occurs only once or for a short period of time, typically an hour or less, but may be up to 14 days*". For metals the HHRA only deals with 1 hour or chronic (a year or more). **Health effects associated with intermediate exposure are not addressed.**

3.5 Exposure to metal concentrations in soil

Annex F, p7-281

1. p7-281: Deposition rate for various metals is not indicated if this is for TSP, PM_{10} or $PM_{2.5}$ (unlikely).

2. The equation on p7-281 has a term 't' defined as "*accumulation time"* but in the text box beneath there is a term 'T' defined as "*duration of deposition".* It is unclear whether these are meant to be the same. 'T' is assigned a value of 70 years (i.e. a default lifetime) "*as per OEHHA guidance".* However the life of the mine is considerably less than 70 years.

3. Nor is it indicated if deposition rate to soil (DR, $mg/m^2/yr$) is a maximum (where?), an average over the life of the project or, an average over a lifetime (70 years).

4. It is noted the DR in calculations for metals in tank water is different to that used for calculating soil concentrations. For Pb the deposition to soil is given as 0.7667 $mg/m^2/yr$ and to roofs as 0.1793 $mg/m^2/yr$. Both are presented as the maximum for a private residence thus the same location, surely the deposition rates should be the same for roofs and soil. This difference needs to be reconciled.

5. The bioavailability of Pb from soil for children and adults is depicted as 16% (p7-284 & elsewhere). It is not indicated in Annexures that this incorporates the average bioaccessibility of 33% (p7-71) from dust and soil (0.33 x 50% = 16.5%). It would be best to include a bioaccessibility parameter (BAc) in addition to bioavailability (B) in the equations.

It is recommended the above be clarified and the implementation of the OEHHA recommendation in the HHRA justified.

3.6 Assessment of silica

The assessment of silica in the HHRA is undertaken by comparison of predicted air concentrations of silica in $PM_{2.5}$ based on the amount of quartz in mined ore. The data was provided by the air modellers, there is no discussion in the HHRA regarding how $PM_{2.5}$ silica concentrations were determined. **But there should be.** It is stated at p7-84 "*the maximum predicted concentration of silica (as $PM_{2.5}$) at all locations (Project related and privately-owned)* [is] *predicted to be 0.76 µg/m³ and at all privately-owned residences predicted to be 0.21 µg/m³ (as an annual average)*".[8] This has been compared with guideline values of 3 μgm^3 ($PM_{2.5}$, annual average) from Vic EPA (2007), a guideline from California (OEHHA 2005) indicated to be comparable, but value not supplied in the HHRA[9], and 2 μgm^3 from Texas (TCEQ 2009). On this basis the HHRA concluded "*there are no health risk issues of concern in relation to community exposures to crystalline silica derived from Project operations*". The following comments are made.

1) The HHRA only indicates exposures may cause silicosis and maybe also lung cancer. There is no mention of tuberculosis/silicotuberculosis, chronic bronchitis, small airways disease, emphysema or associations with a variety of autoimmune diseases, the evidence for which has been growing since the above agencies conducted their toxicological reviews. **This is a deficiency.**

2) While information is provided in Annexure B of the HHRA for metals, there is no discussion of the basis of the cited guidelines for crystalline silica. It is therefore not possible to judge their applicability to the Bowdens mine HHRA. **This should be rectified.**

3) OEHHA (2005) specifies the guideline of 3 $\mu g/m^3$ as applicable to concentrations of particles having a size range (and reactivity) similar to those measured in the occupational studies as defined by ACGIH. For 'respirable' particles this is a 50% cut point of 4 µm. OEHHA notes some particles of size 10µm do deposit in the alveolar region. Due to the difference in how fine particles are measured for environmental exposure (2.5 µm) vs occupational (50% cut of 4 µm), OEHHA says in a screening HHRA (such as done for silica in the Bowdens HHRA) its

[8] This 3.6 fold difference between project-related residences and private residences might also apply to metals in $PM_{2.5}$, including Pb.

[9] Consultation of OEHHA (2005) shows 3 μgm^3, it is possible Vic EPA (2007) just adopted this.

guideline should first be compared with crystalline silica in PM_{10}. If the silica concentration in $PM_{2.5}$ modelled at a receptor is less than 3 µg/m^3 but PM_{10} is greater than 3 µg/m^3, it is indicated further investigation is needed. **The OEHHA silica guideline in the HHRA has not been applied as intended by OEHHA.**

4) TCEQ have an extensive technical support document detailing the basis of their air guidelines for crystalline forms of silica. For chronic exposures three values have been set.
    - A chronic reference value (ReV) of 2.0 µg/m^3 applied to particles ≤ 4 µm for protection against silicosis. (See above comment on the OEHHA guideline for appropriateness of this to $PM_{2.5}$).
    - An environmental screening level (ESL) set to 0.3 ReV of 0.6 µg/m^3. ESLs are used in TCEQ's air permitting process to evaluate air dispersion modelling' s predicted impacts, i.e. precisely what has been done in the HHRA. If predicted airborne levels of a constituent do not exceed the screening level, adverse health or welfare effects are not expected. If predicted ambient levels of constituents in air exceed the screening levels, it does not necessarily indicate a problem but rather triggers a review in more depth. **It is noted the guidelines from TCEQ have not been applied as intended by the agency. Also the predicted maximum air concentration for any residence is greater than the ESL for silicosis, some discussion in the HHRA on the different guidelines from TCEQ, how they are applied, and how predicted silica concentrations from the mine comply, is warranted in the HHRA.**
    - A long term ESL for air permit reviews (i.e. as for the Bowdens mine). This is 0.27 µg/m^3 applied to particles ≤ 4 µm and set at a risk of 1 in 100,000 for lung cancer mortality. **It is noted the predicted maximum air concentration for any residence is greater than the ESL for lung cancer.**

Overall, the above indicates a more scholarly discussion of health effects and risk than that provided in the ¾ page devoted in the HHRA to silica is warranted. There is insufficient information in the HHRA to allow agreement with the conclusion regarding crystalline silica.

3.7 Assessment of hydrogen cyanide

The predicted maximum 1 hour average concentration (5.9 µg/m^3) of hydrogen cyanide at any receptor is markedly lower than the lowest 1 hour guideline of 340 µg/m^3 cited in the HHRA. This guideline is from California and is for protection against loss of coordination and loss of

consciousness due to cellular hypoxia of the central nervous system. These occur very shortly before death. It is agreed the acute risk of health effects from hydrogen cyanide exposure from the mine is very low.

Regarding chronic risk the HHRA states "*the protection of acute inhalation effects associated with hydrogen cyanide is expected to be protective of chronic health effects*." (p7-85). While this reviewer agrees this is the case, there is recorded in the literature neurological effects in workers exposed to non-lethal HCN concentrations. **It would be germane to provide the biological reasons for lack of health effects occurring at low HNC so non-toxicologists can also appreciate there are no long term outcomes from chronic exposure to low concentrations of hydrogen cyanide.**

3.8 Uncertainties

1. General comment:
    - The discussion of uncertainties in the HHRA is very broad. Based on the conclusion that exposures, as calculated with modelling, are very low the HHRA states "*there is no basis for undertaking any specific sensitivity analysis on the individual parameters chosen in these models as the variability in such an assessment would be very low*." While this is true for many of the substances and pathways assessed it may not be for all, particularly Pb. See detailed comments above.
    - For particulates *per se* it is surprising comment is not made in the HHRA regarding potential differences in health effects from crustal dust vs those from combustion derived particulates which underpin the health guidelines (WHO 2007).
    - For existing exposures it has been assumed the average concentration of metals in rainwater tanks is sufficiently representative of current exposures. A reason is not provided. It would be useful to have also used 95^{th} or 99^{th} percentile.
    - For soil, only limited data are suggested to be available. Why is it limited?

2. Data.

    It is stated "*Most soil is reported to have a lead concentration less than 50 mg/kg. To be conservative existing soil concentrations for lead have been assumed to be 50 mg/kg."*
    - The term "most" is noted. The number of soil samples is not provided in the HHRA. How many are above the chosen 50 mg Pb/kg?
    - From Table 4.2 (p7 -45) it appears the 50 mg Pb/kg is for soil away from the proposed open cut. Why hasn't the average, or a high percentile, been used?
    - There is only one sample for Pb in soil in Lue, why has Pb in agricultural soil being used for the town?

- Pb in soil in the mine open cut area is 1.5 - 1380 mg/kg, what concentration(s) were used to calculate Pb in dust emissions?
- No data is given in the HHRA for Pb, or other metals, in mine ore.
- It is recommended in the HHRA the presence of Pb paint in soil at the school be addressed by the school or Department of Education. Is Pb in soil from house paint likely common for residences at Lue, or elsewhere? If so then cumulative exposures to Pb will be underestimated. It could be a recommendation from the HHRA that this be further investigated.
- It is reiterated the assessment has relied on modelling of emissions presented in the Air Quality Assessment. While the uncertainties associate with the air dispersion modelling are outside the HHRA, even so there are numerous aspects of the emission modelling that should have been brought into the HHRA to inform the reader.
- It is indicated the dust emission modelling incorporates dust management measures. It is indicated the modelling is conservative because:
  - Further management measures would be employed. This suggests deficiencies in the modelling since any prospective management of dust should be included.
  - Rainfall has not been accounted in the modelling. Comment has previously been made regarding the fallaciousness of this logic for supporting conservatism in the HHRA.

3. Exposure assessment:
   - Most HHRAs invoke use of high end data measurements, or estimates, of substance concentrations in environmental media as a main source of conservatism. This HHRA has not done so.
   - It is stated conservative assumptions (not indicated which) have been used for predicting uptake of metals into home-grown produce (fruit, vegetables, eggs, meat, and milk). While this is true for the individual exposure pathway, contribution of metal exposure in these foods to the total RI is very small. Any conservatism in these exposure pathways has minimal impact on the risk assessment.
   - It is indicated metals in rainwater tanks is a "*dominant*" exposure pathway. This reviewer has previously noted the assessment is only for private residences. Figure 5.4 (p7-80) indicates for emissions from the mine inhalation of $PM_{2.5}$ (!) is the dominant pathway in the overall risk index for Pb and some other metals. It has been commented this is not the same as intake for Pb.

3.9 Other comments

1. In the HHRA ‘exposure’ and ‘intake’ appear to be used interchangeably. These are not defined in the list of commonly used terms.

2. Annexure B unfortunately suffers from a variety of conflicting information and important typographical errors (numerous spelling mistakes throughout the report and annexures have not been commented on).
    - At p7-71 it is stated 50% absorption is adopted for lead from all pathways. It is not indicated this is for both children and adults. The reader only finds this out by interrogating the calculation spread sheets in Annexes F & G, even there it is not explicit. While it is conservative, a reason for not using adult specific Pb absorption is not provided. Furthermore in Annexure B (p7-139) it is indicated typical Pb absorption in adults is 10%.
    - Most information for Pb is relegated to Annexure B. Cyanide and silica have separate identified sections in the main text where health risk associated with their exposure is provided. This is not so for Pb, which is strange given that this is arguably the potential emission of most concern. The absence of a dedicated section for Pb within the report means the reader has to hunt various annexes for exposure/intake methods and parameter values specific for Pb.
    - Background intake (exposure) information for Pb lacks credible references in some instances. It is not clear how or what information is used in the HHRA.
    - For a lot of the information it is not indicated if it applies to children or adults.
    - In the 2nd full paragraph 10% of Pb in soil is meant to be 100%.
    - Table B1; >10 µg/dL should be <10 µg/dL.

3. Annexure F – spread sheet calculations:
    - In Annexure B dermal absorption of Pb is indicated to be negligible (agreed). Hence the pathway it is not included in Pb exposure from soil. However dermal absorption is included when calculating Pb intake from water. It is not explained why one media and not the other?

4. Table 4.1 has footnote (C = CCME guideline) that is not in the table. What does the blue text in the table signify? It should be confirmed in table footnotes the number of samples analysed and also the depth of the samples.

5. The HHRA does not indicate what existing soil concentrations have been used. It is left to the reader to deduce from spread sheets in Annexures G & F that for metals other than Pb it is the mean value from Table 4.1. This should be made explicit in the HHRA.

6. Table 4.2: Dust indoors - An indication of the sample numbers and variability would be useful to show if the exceedances of the guideline(s) are common.

7. Table 4.3: The number of samples and percentage exceedance should be included.

8. P7-62 indicates emissions of fine particulates (as $PM_{2.5}$ and PM_{10}) from diesel combustion in mining equipment is included in Section 5.2 ('Assessment of impacts from dust emissions'). This reviewer could find no mention of diesel emissions**. It is therefore unclear whether diesel emissions, from mine equipment and or road transport) have actually been assessed.**

9. Acute guidelines for crystalline silica and hydrogen cyanide are not included in Table 5.1 and 5.2. It appears silica is assessed for chronic effects not acute, and cyanide for acute but not chronic.

10. Legend in Figure 5.3 does not correspond to what is in the bar chart.

11. It would be appropriate to mention whether exposure to lithium will impact treatment of bipolar disorder.

12. The HHRA assesses potential for risk of exceeding health guidelines as risk quotient (RQ) and risk index (RI), however in Annexures hazard quotient (HQ) and hazard index (HI) are reported.

4. Outcomes of health risk assessment

The HHRA "*has concluded that impacts derived from the Project make a negligible contribution to overall exposures to these metals and there are no health risk issues of concern relevant to the Project (including construction and operational phases). These conclusions apply to all members of the community, adults and children as well as sensitive individuals*."

This conclusion is primarily based on calculated exposures being less than health based guidelines, for individual substances the difference is substantially less. However issues are raised in the above

comments regarding the selectivity of using data for PM_{10} (for dust deposition) and $PM_{2.5}$ (for exposure to metals), the assessment of Pb and crystalline silica, and only assessing exposure of private residences for some substances. **Consequently, although the conclusion may turn out to be correct, this reviewer is unable to concur with the stated conclusion until the comments are addressed.**

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