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12 April 2021

1st Floor, 12 Dangar Road Brooklyn NSW 2083

Attention: Scott Hollamby (scott@rwcorkery.com)

Proposal: Response to Drew Toxicology Consulting's Peer Review of the Bowdens Silver Project Human Health Risk Assessment

Environmental Risk Sciences Pty Ltd (enRiskS) has reviewed the *Review of HHRA for Bowdens Silver Mine* (*SSD 5765*) prepared by Drew Toxicology Consulting. A full response to relevant matters raised within the peer review document are provided in the attached response table.

The comments provided have assisted in further clarifying some aspects of the HHRA. This peer review is technical in nature and, as such, the response attached is also technical in nature and may not be readily interpreted by lay persons. The HHRA report has been revised and updated to incorporate various clarifications on matters raised in the peer review by Drew Toxicology Consulting to ensure the document appropriately addresses the matters raised and provides all readers with these clarifications. This ensures all the information relevant to the assessment is in the one updated report.

It should be noted, however, that the comments provided by Drew Toxicology Consulting have not changed the conclusions of the HHRA.

In regard to potential emissions to air during the project, the HHRA has evaluated risks posed from short term and long term exposures to particles or metals attached to particles in air as well as due to long term deposition onto soil and uptake into home grown produce or deposition into drinking water for all properties and other key locations (such as schools) within 4-6 km of the proposed mine. The HHRA found that dust emissions from the project would make a negligible contribution to exposure to particles and/or metals attached to particles and so no project related health impacts are likely for any member of the community.

I would be happy to discuss any aspect of the issues raised, or the response, with Dr Roger Drew and/or the NSW Department of Planning, Industry and Environment, as required.

Yours sincerely,

Dr Jackie Wright (Fellow ACTRA) Principal/Director Environmental Risk Sciences Pty Ltd



Response to Human Health Risk Assessment Peer Review

This document has been prepared in response to the *Review of HHRA for Bowdens Silver Mine (SSD 5765)* prepared by Drew Toxicology Consulting in September 2020. The response to comments presented within the peer review has been tabulated for ease of review. It should be noted that the HHRA report and modelling have been updated to include revised modelling of concentrations and deposition of copper and manganese and address comments received as part of the submissions process as well as the peer review. Importantly, none of these change the HHRA outcomes.

| No. | Peer Review Comments - Dr Roger Drew (11 September 2020) (note text has been paraphrased) | enRiskS Response | Relevant HHRA Section |
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| Over | view Comments | | |
| 1 | Section 2.3a: 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. 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). | The HHRA includes all the relevant details to allow the assumptions and calculations to be tracked and replicated. Contour maps are provided in the Air Quality Assessment (AQA) and it is not common practice to repeat them in the HHRA. The ability to do this is limited by the standard style requirements for the EIS established by the project managers, which ensures that repetition between technical reports is minimal. In addition, it is our experience that the details of the air quality modelling may not be easily understood by some members of the community, hence their inclusion may decrease HHRA readability. It is noted that Section 1.7 of the HHRA. | 1.7 |
| 2 | Section 2.3b: Exclusion of assessment of health risk at project-related residences (i.e. mine owned dwellings) for some substances and pathways is a concern. | In NSW, assessment of project owned residences (referred to in the HHRA as "Project-related" residences) does not have to be undertaken in the same way as undertaken for off-site dwellings. This is because there are usually limitations on who can occupy such dwellings and/or when the dwelling is occupied. For example, during operations some dwellings are retained unoccupied, utilised as an office etc. Notwithstanding, it is noted that the AQA provides assessment of project-related residences against the relevant community criteria for particulates, silica and hydrogen cyanide. Consideration is also given to noise received and noise management at project owned residences. | 4.2, 5.2.5, 5.3, 7.1, 7.4.1 |
| 3 | Section 2.3c: Notwithstanding compliance with regulatory air quality guidelines, assessment of health risks due to airborne particulates from the mine is restricted to assessment PM2.5, at private residences, to the health impact of death, and only to incremental increases. For some pathways and substances exposure to TSP and PM10 is also pertinent. | Detailed evaluation of the exposure response relationships for $PM_{2.5}$ (all endpoints) and PM_{10} (all endpoints) has been undertaken by the author. Understanding of these relationships has also been discussed with NSW Health. | Annexure A |



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| | | It is the case that there are a wide range of such relationships and that such relationships also exist for PM_{10} sized particles. It is noted though that the most sensitive relationship of any of them is the one for all causes of mortality related to $PM_{2.5}$. If a project is checked for issues related to this exposure response relationship and no unacceptable risks are found then they will also not be found for PM_{10} related endpoints. Some additional comments on this matter have been added to Annexure A of the updated HHRA. | |
| 4 | Section 2.3d: 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). | Please see response to item 2. | - |
| 5 | Section 2.3e: 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. | This relates to certain metal concentrations recorded within some rainwater tanks. It is important to note that the HHRA calculations have been undertaken using the highest predicted contributions from the Project at non-project locations. As such, the calculated risk represents the highest health risk to the community. Furthermore, using lead as an example, the predicted contribution to lead concentrations in rainwater tanks solely due to the proposed mine is 0.0000086mg/L or 0.00086 ug/L – so small as to be indistinguishable. As such, there is no concern that the Project would result in exceedance or have any significant contribution to lead within rainwater tanks regardless of their existing metal levels. | 4.6 |
| 6 | Section 2.3f: 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 | Please see response to item 24 which comprehensively addresses this and other comments relating to blood lead level guidance including toxicity reference values (TRV). | - |



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| | disagrees with some aspects and has offered, with justification, alternative inputs to the TRV derivation. | | |
| 7 | Section 2.3g: The risk measure (RQ) for exposure to airborne Pb appears to have been miscalculated. | We disagree with this statement. Please see response to item 8. | - |
| 8 | Section 2.3h: 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. | It is common practice to use inhalation specific TRVs (toxicity reference values) as well as ingestion specific TRVs to assess risks for the different types of exposure and to add these together to determine an overall picture of risk for an activity. It is not considered erroneous. | Annexure B (Section B5) |
| 9 | Section 2.3i: 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. | Please see response to item 24 which comprehensively addresses this and other comments relating to blood lead level guidance. | - |
| 10 | Section 2.3j: Some exposure pathways of metals are dependent on deposition from air to roofs and soil. The dust size used for this is PM10 despite it being clear that some residences are subject to TSP from the mine. Thus, exposure to metals, including Pb, has been potentially underestimated. | Deposition has used the rates based on TSP. This has not been underestimated. Also refer to response to item 34. | Annexure E (Section E2.2) |
| 11 | Section 2.3k: 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. | Please refer to responses to items 47 to 52. | - |
| 12 | Section 3.1: 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. | Such detailed information is generally not presented within assessment reports given that the sampling ports are generally standard sizing. The TSP inlet on the HVAS allows collection of all particle sizes suspended in air as opposed to (for example) a PM ₁₀ monitor that filters out larger particles. | - |
| 13 | Section 3.1: Confining the HHRA to only PM2.5 misses possible health effects from larger particulates. Particulates >2.5 µm should be included in the health effects assessment of particulates. In this reviewer's opinion there is compelling evidence in the EIS that exposure to particulates bigger than PM2.5 are expected to occur at residences and therefore their consideration should be included throughout the HHRA. | Please see response to item 3. | - |
| 14 | Section 3.1: Curiously PM_2.5 is lower in winter (June - August) when, in a country town, it would be expected wood heaters would be extensively | Section 5 of the AQA and Section 4.7 of the HHRA presents the existing environment information and discussion in relation to both PM_{10} and $PM_{2.5}$. | 4.7 |



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| | used. The possible explanation of higher rainfall in winter is not viable because the wetter months are December and January. This anomaly deserves comment. | At the outset, it is noted that, in the 2017 data set presented, there are no days in which the 24 hour $PM_{2.5}$ levels exceed the $25\mu g/m^3$ criteria. In relation to the pattern of lower and higher levels, the AQA explains for PM_{10} a number of events that may have influenced the average outcomes applied for assessment. This includes bushfires occurring during February 2017, dust storms in April 2017 and dust storms and hazard reduction burns in September 2017. These similarly correlate to the higher $PM_{2.5}$ levels. In addition, Lue is not densely populated, compared to other country towns such as Mudgee, and therefore it does not follow that a pattern of elevated $PM_{2.5}$ would necessarily occur within and surrounding Lue | |
| 15 | Section 3.1: When considering exposure and intake of metals that have systemic effects, particularly Pb, ingestion is a contributing pathway that has not been considered in the HHRA. | The HHRA has considered the ingestion of soil and dust for lead and all other metals, as it is standard practice for these types of HHRAs. It is noted that the soil and dust ingestion rates adopted in the assessment of ingestion of deposited dust to soil and indoor dust includes the inhalation and swallowing of larger particles as detailed by the USEPA (USEPA 2017), which clearly states that this pathway does not need to be assessed separately (i.e. already accounted for in the soil ingestion rates). The soil ingestion rates adopted for residents is consistent with those recommended by the USEPA (2017). | 5.2.3 Annexure E (Section E2.1) |
| 16 | Section 3.1: It is stated "This health endpoint [all-cause mortality] captures all other health effects found to be causally related to PM2.5 exposure and is the most significant in terms of calculating risks related to changes in PM2.5 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? It is this reviewer's opinion that health effects other than mortality should 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. | Please see response to item 3. | - |
| 17 | Section 3.1: It is recommended all residences, both private and project-related, be included in all parts of the HHRA. | Please see response to item 2. | - |
| 18 | Section 3.1: 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 | Commentary has been added to Annexure A of the updated HHRA indicating that the incremental $PM_{2.5}$ value used in the calculation of page 7-135 is the annual average increment for the maximum off-site location. | Annexure A |



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| | 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. | | |
| 19 | Section 3.1: 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 cumulative assessment was undertaken in the AQA, the results are summarised in Section 5.2.2.2 of the HHRA. The AQA confirms that there are no predicted exceedances of the cumulative PM_{10} or $PM_{2.5}$ NEPM criteria at any privately-owned residences. | 5.2.2.2 |
| 20 | Section 3.1: 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. | Some additional text providing further justification is presented in Annexure A of the updated HHRA. | Annexure A |
| 21 | Section 3.1: This reviewer does not consider the offered reason for conservatism in the calculation for incremental mortality risk (rainfall which would reduce dust emission on wet days and increase wet deposition) is valid. | This is addressed in more detail in the AQA report (Section 4.4) and some additional text has been included in the uncertainty discussion (Section 5.5) of the updated HHRA. It is noted that there is a significant margin of safety in risks related to dust deposition. Dust deposition would need to be more than 10 times higher for risks to be unacceptable. This would be sufficient to account for any variability in deposition, where calculated using various different assumptions. It is also noted that the highest increase in annual PM _{2.5} levels at any | 5.5 |
| | | privately-owned residence ($0.8 \ \mu g/m^3$ at receptor R7 in Year 9) for all modelled scenarios was selected to calculate the risk. As such, the calculated risk for the broader community would be lower. For example, the highest predicted increase in Annual PM _{2.5} within Lue is $0.2 \ \mu g/m^3$ (at multiple receptors). | |
| 22 | Section 3.1: Contour maps of dust concentrations for TSP, PM10 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). | Please see response to item 1. | - |
| 23 | Section 3.2-1: The use of gastric phase bioaccessibility data for the HHRA is conservative. It would be useful if a discussion such as above were included in the HHRA. | This information was provided in Annexure D of the HHRA (documentation provided by the University of South Australia). | Annexure D |



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| | Section 3.2-2: The HHRA does not contain discussion/rationale regarding the applicability of the DEFRA deliberations on Pb to the Bowdens HHRA: -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 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. -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. -Reasoning for adoption of the DEFRA BPb LLTC as a target should be included in the HHRA. It would be expected this would centre on applicability to the Bowden HHRA. -DEFRA (2014b) made a policy decision to use 3.5 µg/dl as the LLTC for the derivation of soil screening levels. Soil screening 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. -Should derivation of a TRV for Pb exposures be accepted for use in the HHRA, this reviewer recommends the BMDL01 for child neurobehavioural effects of 1.2 µg/dL as the target BPb (µg/dL) for conversion to a TRV (µg/kg/d). -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). | The DEFRA approach is blood lead modelling in reverse. That is, it takes blood Pb levels that are related to specific health effects (from studies) and back-calculates to exposure levels that may cause this. Hence, the HHRA is based on a blood lead modelling approach as it uses exposure levels to determine maximum intake to remain within acceptable blood Pb level limit. The DEFRA approach was as follows: - Ten possible points of departure (POD) were identified, ranging from 1.2 to 6.1 ug/dL (the effects considered included neuro-behavioural effects in children and cardiovascular and renal effects in adults). These 10 PODs were identified as the 10 possible LLTCs (low level of toxicological concern values). The US CDC goal of 5 ug/L (which is the same as the Australian goal) is also identified. - Three options for the LLTC were identified from the above 10 options – 1.6 ug/dL, 3.5 ug/dL and 5 ug/dL. The first two LLTCs were based on the data reviewed (i.e. a science position) and the last LLTC was identified as a policy position. - DEFRA then back calculated a daily intake corresponding to the target blood lead concentrations (LLTCs). For children the IEUBK model was used which is used in Australia for estimating exposures to lead by children. Three LLTCs were also recommended for consideration for children. Three LLTCs were also recommended for adults. - DEFRA did not make a single LLTC recommendation as it was noted that there were some significant risk management choices that were required in selecting the value. In this HHRA, the LLTC of 3.5 ug/dL or 1.4 ug/kg/day has been adopted. As noted above, this is a position based on science, not a policy position as indicated by the Reviewer. It is acknowledged that different scientists may arrive at different opinions following the review of the same toxicological data set, and the Reviewer. It is acknowledged that different scientists may arrive at different opinions following the review of the same toxicological data set, and the Reviewer. It | Annexure B |



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| | | In relation to policy, it is agreed that the HHRA needs to adhere to the Australian policy position and this is the approach that has been undertaken. In Australia, the NHMRC recommends that blood lead levels should not exceed 5 ug/dL. Hence, both the blood lead levels determined by DEFRA are more conservative than the Australian policy position. However, given that the Australian policy position is 5 ug/dL, the DEFRA blood lead level of 3.5 ug/dL was considered appropriate yet conservative and was adopted in the HHRA. It was felt that the DEFRA blood lead level of 1.6 ug/dL was overly conservative for an Australian HHRA hence this value was not adopted. It could also be argued that the DEFRA policy position LLTC of 5 ug/dL is relevant, however, this would be a less conservative position. | |
| | | From an overall perspective, the HHRA has found that the health risks from the proposed mine contribute less than 5% of the daily intake as related to the DEFRA blood lead levels. The contribution to lead levels from the proposed mine would be an even smaller proportion if the higher blood lead level of 5 ug/dL had been used. | |
| 25 | Section 3.2-2: It is strongly recommended BPb modelling also be undertaken in the HHRA. | Please refer to response to item 24. | - |
| 26 | Section 3.2-2: For reasons not explained, DEFRA chose the relationship for Pb dietary intake (μ g/kg/d) to determine the mean BPb concentrations (μ g/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. | Please refer to response to item 24. | - |
| 27 | Section 3.2-3: Comparing predicted Pb in PM2.5 with a guideline for TSP means the RQ for exposure to airborne Pb has been miscalculated. | A review of the NEPM indicates that this is correct and a sentence has been included within the updated HHRA that relates to compliance with the NEPM. The basis for the NEPM value is not transparent and given the lack of relevant data to review the value properly it is not considered suitable for use in risk assessment. As the assessment of lead uses an intake that is protective of a blood lead level (from blood lead modelling of all intakes), this is an intake over all pathways and hence the inhalation value used in the risk assessment has been revised to reflect the consideration of total intake from all pathways. The calculations and HHRA has been updated to reflect this change. | 5.2.6.2 Annexure B |



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| 28 | Section 3.2-3: 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. | Dust deposition based on TSP was used in these calculations. There has been no underestimation of risk. | Annexure E (Section E2.2) |
| 29 | Section 3.2-4: 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. | It is common practice to use inhalation specific TRVs as well as ingestion specific TRVs to assess risks for the different types of exposure and to add these together to determine an overall picture of risk for an activity. It is not considered erroneous. This is done in the NEPM in the derivation of the HILs and is common practice in any risk assessment. For lead, where the approach has adopted a total intake from blood lead modelling (refer to response to item 27) the inhalation values for lead have been updated within the HHRA. | 5.2.6.2 Annexure B |
| 30 | Section 3.3-1: What percentage of rainwater tanks have filters that may remove metals from the water? | That information was not provided/collected (and is unknown) otherwise it would have been mentioned in the HHRA. | - |
| 31 | Section 3.3-2: The following should be clarified. Table 4.4 (measurements of metals in tank water) does not indicate if the tank water data is pre- or post-cleaning. Blue text in the table signifies exceedance of a drinking water guideline but the number of exceedances is not shown. 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. | Table 4.4 includes the data for the survey. The data included were just the results taken for samples collected prior to cleaning. If the post cleaning samples were included the averages would have been lower. It is noted that National and State based guidance for using rainwater tanks for drinking water indicate that tanks should be cleaned regularly to remove sediment and that this is a responsibility of the homeowner. A total of 42 samples were collected. For cadmium there were 4 samples above DWG. For iron there were 2 exceedances. For lead there were 2 exceedances. For nickel there were 3 exceedances. The table notes have been updated within the HHRA with the information provided above. The tank cleaning was offered to homeowners as a community service and to allow the collection of the sediment. The samples prior to cleaning and of the collected sediment were collected on the same day. There was no trigger to clean the tanks following receipt of the analytical results for the water – the work had already been done. Detailed assessment of the likely causes of the exceedances was not included as it was not relevant to the assessment of the proposed mine nor was it appropriate to have detailed discussion of tank housekeeping for different households. | 5.2.6.1, Table 4.4 |



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| 32 | Section 3.3-3: The calculations for 'existing' exposure to metals in rainwater tanks in Annexure F use lower water concentrations than are provided in Table 4-4. 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. | Explanatory information has been added to Section 5.2.6.1 of the updated HHRA. Note, there was an adjustment which is why the values appear to be different. The average concentrations listed in Table 4.4 are the average concentration measured in the rainwater tank survey. The data in regard to metal levels in food include intake from drinking water. The levels in water used in the food surveys for each metal were subtracted from the average concentration in the rainwater tanks to ensure exposure via drinking water was not double counted as it was not possible to simply remove that pathway from the food survey information. For a number of metals (copper, cobalt, chromium and mercury), the levels assumed to be present in drinking water used in the food survey were higher than was present in these rainwater tanks so no additional exposure via these rainwater tanks was added into the assessment of existing exposures. | 5.2.6.1 Table 4.4 |
| 33 | Section 3.3-4: Units in the equations don't cancel. Volume of rainwater (VR) from the equation is m3 (currently no units for VR in Table E6). Nevertheless, it appears this has been converted to L prior to undertaking the calculations. Suggest 10³ be added to numerator of the VR equation and VR units in Table E6 indicated as L. A unit conversion of 1000 cm3/m3 is missing from the Cw equation. No units for Cw in Table E6. | The equation presented in the report and partly in the spreadsheet had an error in the units listed for bulk density, which should be g/cm ³ , and the report equation did not include the conversion from m ³ to L (included in the calculations). The equation and units for all parameters have been revised in the report and spreadsheets to ensure these are all correct. This corrects the documentation but does not change the calculation. This has been clarified in the updated HHRA. | Annexure E |
| 34 | Section 3.3-5: 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 PM10 the proportion of metal in the particle may also be different. | The deposition calculations have been based on TSP for deposition to soil and uptake into plants and livestock, and rainwater tanks. The dust deposition used in the calculation of impacts to water quality in rainwater tanks has been revised to be consistent. This has increased the calculated RI for ingestion and dermal contact with water from rainwater tanks, however the RI remain very small and there are no changes to the outcomes of the assessment. This has been clarified in the updated HHRA report. | Annexure E |
| 35 | Section 3.3-6: 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/m2/yr and to roofs as 0.1793 mg/m2/yr. Both are presented as the maximum for a private | Please see response to item 34. | Annexure G |



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| | residence thus the same location, surely the deposition rates should be the same for roofs and soil. This difference needs to be reconciled. | | |
| 36 | Section 3.3-7: 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. | The Kd (soil-water partition coefficient) listed in the RAIS database is the soil water partition coefficient. It is the value used to estimate partitioning of chemicals dissolved in water to the soil through which the water is passing or vice versa. The Kd values adopted are commonly used in all risk assessments, and for metals are assumed to be sufficiently conservative to be independent of the soil type. There are no studies that provide Kd values for lead based on soil type etc. Further the risks related to ingestion of lead dissolved in water in rainwater tanks is very low and hence any minor variability in the Kd value (should there be any justification to select a different value for soil type etc [which there is not]) would make no change to the risk outcomes. | - |
| 37 | Section 3.4-1: It is this reviewer's opinion 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. | The approach adopted in the HHRA is standard for such assessments and has been discussed with and accepted by NSW Health. It is noted that air dispersion modelling occurs in 1 hour increments and has been undertaken in accordance with the Approved Methods for the Modelling and Assessment of Air Pollutants in New South Wales (EPA, 2016). The ability to accurately model smaller time increments is questionable as dispersion modelling can generally only model in hourly timesteps for hourly averaged emissions. It is noted that the AQA reported and the HHRA assessed the maximum 1-hour average (i.e. 100 th percentile) recorded in all modelled years (i.e. the highest value out of four scenarios each with 8,760 1-hour increments). | - |
| 38 | Section 3.4-1: This reviewer does not know what " asymptomatic, non- sensory effects" may be. This should be clarified (Footnote to Table 5.1). | This terminology comes directly from the definitions of these terms as specified at https://edms.energy.gov/pac/TeelDef AEGL-1 is the airborne concentration (expressed as ppm [parts per million] or mg/m3 [milligrams per cubic meter]) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, nonsensory effects. However, these effects are not disabling and are transient and reversible upon cessation of exposure. | - |



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| | | AEGL-2 is the airborne concentration (expressed as ppm or mg/m3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting, adverse health effects or an impaired ability to escape. AEGL-3 is the airborne concentration (expressed as ppm or mg/m3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience life-threatening adverse health effects or death | |
| 39 | Section 3.4-2: There is no discussion of the basis for the TCEQ 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. | It is agreed that the requested information would be interesting, however, adding this information to the HHRA would not change the value of the adopted guidelines or the outcomes of the HHRA. It is also noted that publicly available information that outlines how guidelines from overseas were derived is not always available. It is noted that the exposures being evaluated really are chronic environmental exposures with limited potential for acute exposures to be of concern. This is included to ensure that the peak exposures (which may occur once or more than once) are addressed. | - |
| 40 | Section 3.4-3: 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 PM10. This appears to be misapplication of TCEQ guidelines. | The footnote to Table 5.1 was unclear and has been revised in the updated HHRA report to indicate that this relates to metals in and on particulates. The derivation of the TRVs for metals by TCEQ has considered the dose reaching the lungs in each of the relevant studies considered in the evaluation. Hence it is appropriate to consider and use $PM_{2.5}$ exposure data for comparison with these (and other) acute or short-duration guidelines, where it has been assumed that 100% of $PM_{2.5}$ reaches the lungs. The reference to PM_{10} is a policy approach for application of the air guidelines in that jurisdiction, and does not reflect the basis for the derivation. | Table 5.1 |
| 41 | Section 3.4-4: 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 | The approach adopted in the HHRA is standard for such assessments and has been discussed with and accepted by NSW Health. It is noted that the exposures being evaluated really are chronic environmental exposures with limited potential for acute exposures to be of concern. This is included to ensure that the peak exposures (which may occur once or more than once) are addressed. | - |



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| | 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. | | |
| 42 | Section 3.5-1: p7-281: Deposition rate for various metals is not indicated if this is for TSP, PM10 or PM2.5 (unlikely). | Please refer to response to item 44. | - |
| 43 | Section 3.5-2: 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. | These terms refer to the same value. This has been clarified in the updated HHRA report. The calculation has determined the concentration in soil at the end of 70 years of deposition arising from the proposed mine. This is intended to ensure the calculation is conservative. | Annexure E Annexure G |
| 44 | Section 3.5-3: It is not indicated if deposition rate to soil (DR, mg/m2/yr) is a maximum (where?), an average over the life of the project or, an average over a lifetime (70 years). | The deposition to soil calculates a soil concentration at the end of 70 years of deposition when mixed into the top 1 cm or 15 cm of soil depending on the exposure scenario. The deposition rate used in the calculation was the value at the maximum affected location and the maximum value at private residences. This is described in Annexure E Section E2.2. | Annexure E |
| 45 | Section 3.5-4: 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/m2/yr and to roofs as 0.1793 mg/m2/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. | Please refer to response to item 34. | Annexure G |
| 46 | Section 3.5-5: 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 ($0.33 \times 50\% = 16.5\%$). It would be best to include a bioaccessibility parameter (BAc) in addition to bioavailability (B) in the equations. | The text on page 7-71 of the HHRA clearly outlines that multiplying the bioaccessibility by the absorption is how this value is calculated. It is not considered necessary to repeat this point on every occasion where this value is used. | 5.2.6.4 |
| 47 | Section 3.6: There is no discussion in the HHRA regarding how PM2.5 silica concentrations were determined based on data provided by the air modellers. | This is discussed in the AQA (please refer to Sections 6.5 and 7.8) and is not repeated in the HHRA. | - |
| 48 | Section 3.6-1: 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 | A toxicity summary is now included in Annexure C and Section 5.3 of the HHRA has been updated accordingly. | 5.3 Annexure C |



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| | evidence for which has been growing since the above agencies conducted their toxicological reviews. | | |
| 49 | Section 3.6-2: 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. | A toxicity summary is included in Annexure C of the HHRA that includes a discussion of the cited guidelines. | Annexure C |
| 50 | Section 3.6-3: Due to the difference in how fine particles are measured for environmental exposure (2.5 μ m) vs occupational (50% cut of 4 μ m), OEHHA says in a screening HHRA (such as done for silica in the Bowdens HHRA) its guideline should first be compared with crystalline silica in PM10. If the silica concentration in PM_2 5 modelled at a receptor is less than 3 μ g/m3 but PM10 is greater than 3 μ g/m3, it is indicated further investigation is needed. | This is now discussed in the toxicity summary provided in Annexure C of the updated HHRA. Annexure C notes that Victoria (EPA Victoria 2007) has adopted the OEHHA (2005) guideline for RCS as PM _{2.5} and that this approach has also been adopted in the HHRA. There is no change in HHRA outcomes. | Annexure C |
| 51 | Section 3.6-4: The guidelines from TCEQ have not been applied as intended by the agency. As 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. It is noted the predicted maximum air concentration for any residence is greater than the ESL for lung cancer. | Both lung cancer and the TCEQ ESLs are discussed in the toxicity summary now provided in Annexure C of the updated HHRA. Annexure C indicates that IARC is clear that the determination that RCS is carcinogenic relates only to occupational exposures. For this reason, the TCEQ (2009) cancer guideline has not been adopted in this HHRA. There is no change in HHRA outcomes. | Annexure C |
| 52 | Section 3.6: Overall, a more scholarly discussion of health effects and risk than that provided in the ³ / ₄ 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. | Additional information has been added to the HHRA to address the comments as discussed in responses to items 47 to 51. The toxicity summary in Annexure C of the HHRA provides a comprehensive discussion of potential risks and health effects as well as guideline intake values. It remains the conclusion of the HHRA that there are no health risk issues of concern in relation to community exposures to crystalline silica derived from Project operations. | Annexure C |
| 53 | Section 3.7: It would be germane to provide the biological reasons for lack of health effects occurring at low HCN so non-toxicologists can also appreciate there are no long term outcomes from chronic exposure to low concentrations of hydrogen cyanide. | Annexure C has been updated with a more detailed toxicity summary with the main part of the report adjusted accordingly. | Annexure C |
| 54 | Section 3.8-1: For particulates 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). | Crustal dust is discussed in a number of locations through the report and in Section 5 in particular. The discussion notes that combustion derived particles appear to be more of concern than crustal dust. | 5.2.2.1 |



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| 55 | Section 3.8-1: 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 95th or 99th percentile. | The data on the existing exposures for rainwater tanks was used to determine "typical background exposure". The NHMRC Drinking water guidelines includes "typical values for Australian drinking water" – which they state for lead as ranging up to 0.01 mg/L with typical concentrations less than 0.005 mg/L. Out of 42 samples collected prior to cleaning of the tanks only two tanks exceeded 0.01 mg/L lead. This is essentially in line with the NHMRC guidance for drinking water supplies in Australia. It is also noted within the HHRA that tank water quality can vary over time from factors including how recently the tank has been cleaned out. Given that tanks were cleaned as part of the sampling process, the use of the pre-cleaning values already provides conservatism for these locations. Application of 95 th and 99 th percentile values for assessment would not provide value. Furthermore, given that this section (4.6) is about the existing environment, changing the value used makes no difference to the potential contribution from the proposed mine. For lead, the predicted contribution to concentrations in rainwater tanks solely due to the proposed mine is 0.0000086mg/L or 0.00086 ug/L – so small as to be indistinguishable. As such, there is no concern that the Project would result in exceedance or have any significant contribution to lead within rainwater tanks. Therefore, the approach taken is considered appropriate. | 4.6 |
| 56 | Section 3.8-1: For soil, only limited data are suggested to be available. Why is it limited? | Extensive data is available for the Mine Site and surrounds for which ready access for sampling was available. However, the geographic extent was relatively limited beyond this area. Notwithstanding, it is considered sufficient for the purpose of assessment. Notably, the adopted background value of 50mg/kg is reasonably consistent with the geometric mean of 13.3mg/kg recorded by the Macquarie University within and surrounding Lue. The wording in the uncertainties section has been updated (Section 5.5 of the updated HHRA). | 5.5 |
| 57 | Section 3.8-2: 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? | Table 4.2 indicates that soil samples collected from the mine site away from the proposed main open cut pit reported lead concentrations <u>all</u> below 50 mg/kg. The wording in the report and uncertainties section has been updated to confirm a total of 388 soil samples were collected and clarify the data. A figure is also attached to this response (refer to Attachment A) to provide an understanding of the extent of soil sampling and distribution of Pb concentrations. | Table 4.2 |



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| 58 | Section 3.8-2: 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? | Table 4.2 refers to information relevant for defining the <u>existing environment</u> . It is about the current situation as it stands not about what could occur if the proposed mine is approved. That is discussed in Section 5 of the report. The table provides information on dust in and around buildings in Lue and then information about the mine site soil. The information provided about the soil on the mine site covers two aspects – levels of lead in the soil within the area containing the orebody where mining will occur (i.e. 1.5-1380 mg/kg) <u>and</u> also the soil around the rest of the mine site away from the ore body – this soil had lead below 50 mg/kg in all samples and so the assessment assumed 50 mg/kg as a worst case assumption for the existing lead levels in that soil. | Table 4.2 |
| 59 | Section 3.8-2: There is only one sample for Pb in soil in Lue, why has Pb in agricultural soil being used for the town? | The company proposing the mine has ready access to the mine site and parts of their exploration licence area for detailed investigation. Sampling included areas adjacent to Lue's northern boundary. The town is also noted to be small with most receptors on agricultural land similar to the areas around the mine site. Whilst dust wipe samples were collected from 20 buildings within and surrounding Lue, it is acknowledged that soil sampling within Lue was limited. Notably, Macquarie University undertook soil sampling within Lue, collecting a total of 34 samples and recording a geometric mean of 13.3mg/kg and range of 4.7mg/kg to 300mg/kg of lead. Section 5.5 of the updated HHRA includes further discussion regarding this additional soil data. | 5.5 |
| 60 | Section 3.8-2: 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? | This detail is outlined within Table 6.3 of the AQA. It notes that for lead it was assumed that waste rock had 0.02% lead or 200 mg/kg, for ore it was 0.32% lead or 3,200 mg/kg and for soil around the mine it was 0.009% lead or 90 mg/kg. The air quality modelling used emission factors for relevant particles from different activities and applied the concentrations for soil, waste, and ore as relevant to each activity. | Table 6.3 |
| 61 | Section 3.8-2: No data is given in the HHRA for Pb, or other metals, in mine ore. | Such data is included in both the main EIS and the AQA. It is noted that Section 1.7 of the HHRA clearly outlines the other technical reports relied upon for the HHRA. | 1.7 |
| 62 | Section 3.8-2: 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. | Dust wipe samples were collected from 20 buildings (18 residences, the Lue Public School and Lue Hotel). Whilst the objective was not to identify lead | 4.3 |



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| | 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. | paint, the only building for which lead paint was indicated and subsequently identified was the Lue Public School. This does not rule out the presence of lead paint within the other buildings. Such recommendations (included in the uncertainties section) are outside the scope of the HHRA as the HHRA focuses on assessing potential impacts of the proposed mine on human health. It is also re-iterated that the estimated contribution from the proposed mine is small. Notwithstanding, it is noted that Section 4.8.9 of the EIS confirms that, should development consent be granted, an information package providing an outline of the existing potential exposure pathways to lead and other metals and ways in which exposures can be reduced would be provided to all residents in the locality. This would include, but not be restricted to, the potential for lead paint to be present. | |
| 63 | Section 3.8-2: It is reiterated the assessment has relied on modelling of emissions presented in the Air Quality Assessment. While the uncertainties associated 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. | Please see response to item 1. It is also noted that Section 1.7 of the HHRA clearly outlines the other technical reports relied upon for the HHRA. | 1.7 |
| 64 | Section 3.8-2: 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. | A response to the matter of rainfall within the modelling is addressed in item 21. In relation to management measures, the HHRA states that the modelling has incorporated a range of preventative dust management measures. It is then also stated that further reactive and corrective measures are expected to be implemented which would result in lower dust emissions. It is not correct to say this is a deficiency. As required, the AQA provides a summary of the best management practices and how these have been applied (or where they are not applicable / have not been applied). It is not practicable to include all reactive or corrective measures within the air quality model and it is a common requirement for management plans to include a trigger action response and contingency measures. As such, reactive and corrective measures have been outlined within the AQA and EIS. Furthermore, conservatism is built into the modelling, such as modelling years with the greatest volume of material being handled. It is therefore appropriate to consider that the modelling is undertaken showing the typical | - |



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| | | worst case which then indicates how critical the management measures will be to the project. If the risks are low with the adopted preventative management measures, then the need for further reactive and corrective measures is less critical to the success and acceptability of the project and to comply with relevant regulations. If the risks are higher, then it is clear to regulators that control measures are critical and additional preventative measures (or a change in the project) may be required. For the Bowdens Silver Project no non-compliances with air quality criteria has been predicted and the contribution to potential health impacts has been assessed as low. | |
| 65 | Section 3.8-3: 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 difficult to respond to this comment as specific queries have not been identified. We can confirm that the HHRA is based on the outcomes of the AQA which has been undertaken in accordance with the required guidance. The HHRA has also complied with the required guidance. The source/s of conservatism in each site-specific HHRA will differ, depending on the data that is available. This is part of what makes each HHRA site-specific. Broad brush statements about site-specific HHRAs are considered problematic for this reason. Irrespective of the above, the HHRA has adopted maximum concentrations e.g. maximum modelled averages in air, maximum deposition for 70 years, background lead concentrations in soil of 50 mg/kg etc. | - |
| 66 | Section 3.8-3: 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. | Noted. | - |
| 67 | Section 3.8-3: 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 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. | Please see item 2 in relation to the private residences. The commentary on metals in rainwater tanks refers to deposition of particles from the mine onto the roofs of houses. The particles can then wash into rainwater tanks which introduces these metals into the water. The only place this is mentioned is in the discussion of uncertainties in Section 5.5. The idea that water in the rainwater tanks may be a dominant pathway is in relation to the wash off of particles into the tank and the fact | 5.5. Figure 5.3 |



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| | | that the lead (or other metals) can dissolve into the water. Once dissolved they are potentially more available for uptake by people when they use the water for drinking etc when compared to lead in dust. | |
| | | Figure 5.3 of the HHRA shows that intake from water is the second most important pathway for existing exposures. The small addition due to the proposed mine again shows that lead in the tanks is the second most important pathway. This does indicate that drinking water from the rainwater tanks is a dominant pathway. This figure has been updated to include the full legend, that was missing. The point of the comment in the uncertainties section was to note that the exposure assessment calculations were conservative. | |
| 68 | Section 3.9-1: In the HHRA 'exposure' and 'intake' appear to be used interchangeably. These are not defined in the list of commonly used terms. | The use of these terms throughout the HHRA has been reviewed and amended where needed. A definition for exposure is already listed in the list of commonly used terms and this definition has been reworded for clarity. A definition for intake has been added to the list. | Commonly Used Terms |
| 69 | Section 3.9-2: 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). | The document has been further reviewed to check for typographical issues. A response to specific comments on Annexure B is provided below. | Annexure B |
| 70 | Section 3.9-2: Annexure B - 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 it is indicated typical Pb absorption in adults is 10%. | It is agreed that WHO guidance indicates a higher level of absorption of lead for children compared to adults. A conservative choice was made for this assessment – to assume the rate of absorption for children is relevant for all life stages. This has been made clearer in the report. | Table 5.2 |
| 71 | Section 3.9-2: Annexure B - 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. | Toxicity summaries for all chemicals are now provided in Annexure B. | Annexure B |



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| 72 | Section 3.9-2: Annexure B - 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. | The sources of information used in this section (on page 7-138) include Food Standards Australia and New Zealand, the National Health and Medical Research Council, the NSW Environment Protection Authority (formerly DEC) and the guidance document used to assess background soil concentrations for metals specified in the NEPM. In addition, these are the same sources used in the NEPM Schedule B7 Appendix A1 to show how the background exposure to lead was calculated for the determination of the HILs. Where necessary, the values listed in Annexure B have been updated. It is not clear in what way these are sources that are considered to lack credibility given that the Health authorities across Australia accepted this information in the NEPM and they are considered to be the premier sources of such information in Australia (especially FSANZ and NHMRC). A discussion on background concentrations of lead in the environment has been included in the HHRA, as is standard practice in these type of assessments, and consistent with the toxicity summarises for the other chemicals. The adopted approach for lead is outlined later on in the toxicity summary. | Annexure C |
| 73 | Section 3.9-2: Annexure B - For a lot of the information it is not indicated if it applies to children or adults. | The toxicity summary for lead has been reviewed to ensure that, where relevant, information is attributed to either adults or children. Figure B1 provides a useful graphic with a summary of health effects of lead exposure >10 ug/dL for adults and children. More detailed information on the critical effects for adults and children is provided below Table B1. The adopted TRVs for adults and children are summarised at the end of the Annexure. | Annexure B |
| 74 | Section 3.9-2: Annexure B - In the 2nd full paragraph 10% of Pb in soil is meant to be 100%. | The HHRA report has been amended accordingly, however it is noted that this does not change the outcomes of assessment. | Annexure B |
| 75 | Section 3.9-2: Annexure B - Table B1; >10 µg/dl should be <10 µg/dl. | The HHRA report has been amended accordingly, however it is noted that this does not change the outcomes of assessment. | Annexure B |
| 76 | Section 3.9-3: 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? | Table 5.2 on Page 7-70 lists the toxicity reference values and the dermal absorbance and dermal permeability values used in this assessment. Dermal permeability refers to absorption via the skin from water while dermal absorbance is absorption via the skin from soil. All metals evaluated for this proposed mine have a dermal permeability listed in the USEPA funded Risk Assessment Information System database – RAIS. It is noted that absorbance from soil is not relevant for many metals. This is because the metals are well attached to the particles and don't easily detach to move | Table 5.2 Annexure B |



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| | | through the skin. For metals dissolved in water, they are more likely to be in a form that can move through the skin. It is usual for experiments to determine such permeability to be undertaken using soluble salts of these metals. As a result, it is more likely than not for metals (including lead) to move through the skin when present in water to which a person may be exposed. This is why the RAIS includes a dermal permeability value. Including this pathway in the assessment means that the assessment has not underestimated the potential for exposure arising from the proposed mine. Some additional text has been added to the updated HHRA report to clarify this. | |
| 77 | Section 3.9-4: 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. | Table 4.1 footnotes have been amended within the updated HHRA. | Table 4.1 |
| 78 | Section 3.9-5: 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. | The HHRA has been updated in response to this comment. | Section 4.3 |
| 79 | Section 3.9-6: 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. | The number of samples has been included as a footnote to Table 4.2 within the updated HHRA. An additional analysis of variability is not considered warranted noting that the HHRA is focused on assessing potential health risks from the proposed mine and not from the existing background conditions. In addition, a detailed understanding of the existing situation is not critical to the HHRA given the estimated negligible contribution from the proposed mine. | Table 4.2 |
| 80 | Section 3.9-7: Table 4.3: The number of samples and percentage exceedance should be included. | The number of samples has been included as a footnote to Table 4.3 within the updated HHRA. An additional analysis of percentage exceedance is not considered warranted noting that the HHRA is focused on assessing potential health risks from the proposed mine and not from the existing background conditions. In addition, a detailed understanding of the existing situation is not critical to the HHRA given the estimated negligible contribution from the proposed mine. | Table 4.3 |
| 81 | Section 3.9-8: Emissions of fine particulates (as PM2.5 and PM10) from diesel combustion in mining equipment are included in Section 5.2 ('Assessment of impacts from dust emissions'). This reviewer could find no | The primary emissions of concern from diesel equipment are $PM_{2.5}$ particles. These particles have been assessed in considerable detail in the assessment. This is flagged in dot point 2 in the list describing what types of | 5.1 |



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| | mention of diesel emissions. It is therefore unclear whether diesel emissions, from mine equipment and or road transport) have actually been assessed. | impacts have been assessed on page 7-51. Furthermore, Section 6.3 of the AQA provides a breakdown of the contribution of diesel emissions for both PM _{2.5} and PM ₁₀ . Detailed information has also been included within the HHRA about the health effects of both particles based on size as well as when metals are attached to such particles as is needed for the proposed mine. Further separate assessment of diesel emissions has not been included due to the primary source of particles being from crustal materials (soil, waste and ore) and the key chemicals being the metals attached to those particles. | |
| 82 | Section 3.9-9: 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. | Please see responses provided to items 47 to 53. | - |
| 83 | Section 3.9-10: Legend in Figure 5.3 does not correspond to what is in the bar chart. | Figure 5.3 has been updated within the updated HHRA and all the legend included (which was missing). | Figure 5.3 |
| 84 | Section 3.9-11: It would be appropriate to mention whether exposure to lithium will impact treatment of bipolar disorder. | Given that existing lithium levels in soil are well below guidelines for low density residential land and additional exposures from the proposed mine do not change that conclusion, it is not considered necessary to provide a detailed assessment of this matter. The dosage of lithium taken by a patient needing this medication is well in excess of what could be sourced from the existing environment or from this proposed mine. The ingestion TRV used in this assessment was 0.002 mg/kg bw/day. If an adult patient (70 kg bw) takes 1 tablet of 250 mg/tablet of lithium carbonate per day then the dose they receive is 3.6 mg/kg bw/day. The dose from the environment is negligible based on this assessment. It is also noted that the lithium toxicological profile in Annexure C of the human health risk assessment includes reference to guidance from the USEPA which also states that intakes from food and water are considered negligible in comparison to therapeutic use. | Annexure C |
| 85 | Section 3.9-12: 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. | References to hazard quotient (HQ) and hazard index (HI) have been removed from the updated HHRA report. | - |

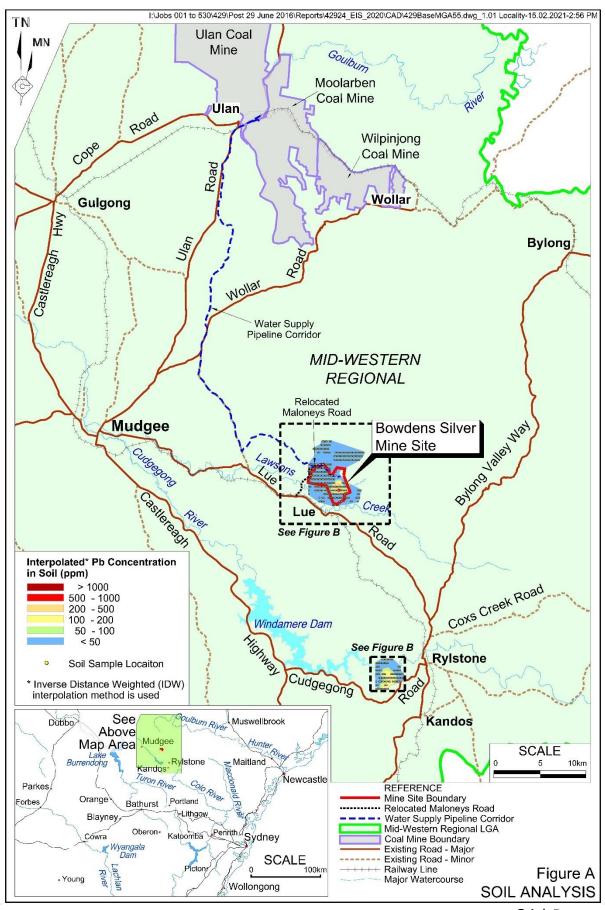


USEPA 2017, *Update for Chapter 5 of the Exposure Factors Handbook, Soil and Dust Ingestion, EPA/600/R-17/384F*, National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, Washington. <<u>https://www.epa.gov/expobox/about-exposure-factors-handbook</u>>.



Attachment A: Soil analysis maps





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