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Human health risk assessment of lead from mining activities at semi-arid locations in the context of total lead exposure

Jiajia Zheng · Trang Huynh · Massimo Gasparon · Jack Ng · Barry Noller

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Abstract Lead from historical mining and mineral processing activities may pose potential human health risks if materials with high concentrations of bioavailable lead minerals are released to the environment. Since the Joint Expert Committee on Food Additives of Food and Agriculture Organization/World Health Organization withdrew the Provisional Tolerable Weekly Intake of lead in 2011, an alternative method was required for lead exposure assessment. This study evaluated the potential lead hazard to young children (0–7 years) from a historical mining location at a semi-arid area using the U.S. EPA Integrated Exposure Uptake Biokinetic (IEUBK) Model, with selected site-specific input data. This study assessed lead exposure via the inhalation pathway for children living in a location affected by lead mining activities and with specific reference to semi-arid conditions and made comparison with the ingestion pathway by using the physiologically based extraction test for gastrointestinal simulation. Sensitivity analysis for major IEUBK input parameters was conducted. Three groups of input parameters were classified according to the results of predicted blood concentrations. The modelled lead absorption attributed to the inhalation route was lower than 2 % (mean±SE, 0.9 %± 0.1 %) of all lead intake routes and was demonstrated as a less significant exposure pathway to children's blood, compared with ingestion. Whilst dermal exposure was negligible, diet and ingestion of soil and dust were the dominant parameters in terms of children's blood lead prediction. The exposure assessment identified the changing role of dietary intake when house lead loadings varied. Recommendations were also made to conduct comprehensive site-specific human health risk assessment in future studies of lead exposure under a semi-arid climate.

Keywords Lead · Health risk assessment · Inhalation · Ingestion · IEUBK · Semi-arid · Mining · Sensitivity analysis

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J. Zheng (✉) · T. Huynh · B. Noller
Centre for Mined Land Rehabilitation, The University of Queensland, Brisbane 4072, Australia
e-mail: jiajia.zheng@uqconnect.edu.au

M. Gasparon
School of Earth Sciences, The University of Queensland, Brisbane 4072, Australia

J. Ng
National Research Centre for Environmental Toxicology (Entox), The University of Queensland, 39 Kessels Road, Coopers Plains, Brisbane 4108, Australia

Introduction

Lead (Pb) is one of the most widespread heavy metal contaminants globally and can cause significant impacts on humans and biota (JECFA 2011). Inorganic lead is primarily absorbed by the human body through ingestion and inhalation and minimally via the dermal route (<0.3 %) (JECFA 2011; ATSDR 2007). Lead exposure is associated with a range of health effects, including neurological, mortality due to cardiovascular diseases, impaired renal function, hypertension, impaired fertility and adverse pregnancy outcomes, reduced sexual mutation and effects on dental health (ATSDR 2007; JECFA 2011). Neurodevelopmental effects have been identified as pivotal data for the risk assessment for children because of the low dose–response relationship and children's vulnerability to lead exposure (Bierkens et al. 2011; Jusko et al. 2008; Lanphear et al. 2000; JECFA 2011).

Lead is derived from mining and processing of its mineralised forms. Sustainable development of lead mining requires sound understanding of the potential environmental and health risks associated with its potential impacts on surrounding communities (ICMM 2007). Risk assessment is the

key process for developing management and communication tools provide better control of the adverse effects of lead on the population (Ricci 2006). Usually, a dose–response analysis is used within the health risk assessment framework (enHealth 2012b). In 2011, the Joint Expert Committee on Food Additives (JECFA) of Food and Agriculture Organization/World Health Organization withdrew the Provisional Tolerable Weekly Intake (PTWI) for lead and concluded that the previous lead PTWI level (0.025 mg/kg b.w. per week) could no longer be considered health-protective (WHO 2011). This action was taken because the dose–response analysis of lead was deemed to lack an indication of a threshold for the key effects on people. In order to assess the health risk associated with lead exposure for young children, the currently available Integrated Exposure Uptake Biokinetic Model (IEUBK) (U.S. EPA 2010) was utilised in this study. The IEUBK Model is a simulated site-specific risk assessment tool for rapid estimation of the aggregate lead exposure, uptake and biokinetic parameters for young children <84 months (7 years) (U.S. EPA 2010). It allows calculation of the risk associated with elevated blood lead in the population and provides the potential contributions from different lead exposure pathways (U.S. EPA 2010). Good correlations have been reported between measured blood lead test and IEUBK predicted blood lead for children living in different sites, such as USA (Hogan et al. 1998), Mexico (Carrizales et al. 2006) and Europe (Glorennec and Declercq 2007; Tristan et al. 2000), when sufficient data about lead exposure are given. It should also be noted that the IEUBK model is expected to yield more accurate prediction on a population level than for single individuals (Lewandowski 2009).

Lead absorption via inhalation is a less important pathway compared with lead from ingestion of various exposure sources in urban environments (ATSDR 2007; Pizzol et al. 2010), but this may not necessarily apply to arid or semi-arid mining locations which are characterised by low relative humidity during the dry season and increased resuspended soil dust into the atmosphere (Mitchell et al. 2010). Globally, there are numerous semi-arid areas where dusts care generated and dispersed by wind over large distances (Bryant 2013). An example of such a dry area is Lake Eyre Basin in continental Australia (Leys et al. 2011) from which continental dusts is dispersed over long distances to the main population centres on the east coast (Radhi et al. 2010). Whilst exposure via ingestion is well understood for city environments, little work has been undertaken to assess inhalation as an exposure pathway at mining communities in arid and semi-arid locations, including Australia. Particulate matter with a size <10 microns (PM_{10}) is a mixture of solid particles and liquid droplets found in the air and transported long distances on the continental scale and can be inhaled and captured by the nasopharyngeal region of the human respiration system (U.S. EPA 2007a). Airborne PM_{10} released from ore materials transporting, the smelters and the resuspension of fine particles from the surface are recognised as important sources of fine particulate matter that contains various metallic compounds (Sobanska et al. 1999) and primary lead pathways in lead–zinc mining and mineral processing activities described in several studies (McMichael et al. 1985).

Since it is easier for airborne lead-containing particles to be mobilised and dispersed in an area with low precipitation, high potential evapotranspiration rate and periodic dry storm events (McTainsh et al. 2005), this study sought to test the hypothesis that the contribution of lead intake via the inhalation route and the soil and dust ingestion routes may be significantly higher at the locations near lead mining and mineral processing activities under such semi-arid climate condition. The following aspects will be examined to test the stated hypothesis and improve the understanding of lead exposure by: (1) investigating the potential sources for lead exposure to young children (0–7 years) from lead intake pathways via inhalation and ingestions routes; (2) evaluating the potential health risk of total lead exposure to young children living at the study site; and (3) using the IEUBK model to quantify the relative contribution of inhalation and ingestion pathways to the total lead exposure in the context of a site-specific human health risk assessment for children living at a semi-arid zone mining community in Australia. It was assumed that the dermal exposure pathway was negligible for this study as reported in the literature (ATSDR 2007). This paper considers the prediction of lead exposure within the constraints of the IEUBK model from the overall contribution of mine source plus natural background and historical additions (lead in paint, petrol, batteries and other minor sources). The authors, however, explored an approach to estimate the potential lead contribution from a relative background site. The direct relationship between dust generated from the mining activities and those found in the residential area is not within the scope of this paper.

Materials and methods

Study sites

The study area is located at the edge of the Lake Eyre Basin in the semi-arid region of continental Australia with historical and current lead mining (open pit) and smelting activities. The total lead emission to air, land and water from basic non-ferrous metal manufacturing at the study area during 2009–2010 was over 100,000 kg (NPI 2012). The lead mineralisation is enriched in a geological sequence, which is also exposed at the surface in part of the residential area. At the study, the primary lead ore product from deep mining is galena (PbS).

A comprehensive residential house-sampling program was conducted to select representative houses across different locations in the residential area of the study site. The residential house sampling program was conducted at 23 sites to ensure (1) an even geographical spread of house across the study area; (2) representative houses from a range of housing types and living standards; and (3) locations close to potential lead sources, such as the mining and mineral activities, natural outcrops and others to be sampled (Fig. 1). The majority of residential houses were located at approximately 0.5–6 km upwind of mining and mineral processing activities. The meteorology information showed that the prevailing annual winds (Fig. 1) are from east, south east and south directions at the study area (BOM 2012).

Sample collection

Air PM_{10}, soil and carpet dust samples were collected during the dry season to evaluate the actual lead exposure for children living at each of the study area sites.

Samples of airborne particulate matter with size <10 micron (PM_{10}) were collected from different locations across a city residential area, including eight indoor sites and 12 outdoor sites. The wind speeds were typically <30 km/h during the air particulate sampling periods with the mean wind speeds of approximately 14 km/h measured daily at a local meteorological monitoring site. Air PM_{10} collections were performed using membrane filters (PALL PVC disc membrane filter GLA 5000, 25 mm size, 5 μm pore size) in cyclones (SKC® Conductive Plastic Respirable Dust Cyclones, Cat. No. 225-62-25). The cyclones were set up in a pump system and run at sampling sites for approximately 7 days, with an air flow of ~2.2 L/min. The cyclones were placed at a height of about 1.8 m above ground and equipped with SKC® PCXR4 pumps that produced a constant flow rate of air (Fig. 2). The membrane filters were weighed with an electronic balance in the laboratory (Model C-38, ATI Cahn balance) to 0.01 mg following conditioning within the laboratory for "time" prior to weighing. Samples and blank filters were weighed and stored separately in new clean Petri dishes (MS ® polystyrene, 50 mm in diameter).

Five sites across the community were equipped with high-volume air particulates (HVA) samplers for routine monitoring purposes. HVA samples were collected over 24-h periods on a 6-day cycle. The total concentration data of lead in HVA samples were available for comparative purpose from the state regulatory authority.

Particles <250 μm are expected to have a higher chance of contributing to exposure through hand-to-mouth activity via the ingestion pathway (Ruby et al. 1996; Ng et al. 2013) and therefore are significant in terms of younger children. This <250 μm fraction was collected from soil and house dust samples for this study. Representative soil samples (0.5 kg) were taken from the garden of each study site (n=23). The samples were a composite collected at each site and made up of bare soil from surface (0–10 cm depth) selected from up to ten different points of each garden. The samples were collected using a stainless steel scoop and placed in a new sample

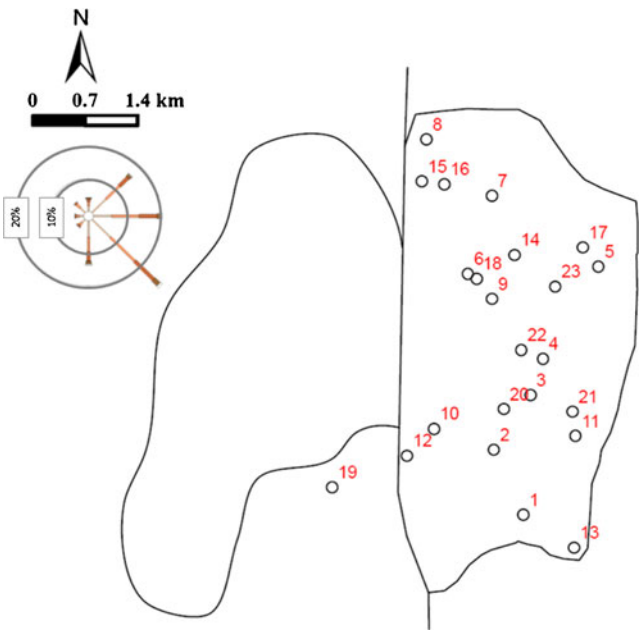

Fig. 1 Map of sampling sites (*circles* in the map indicating sampling sites)

Fig. 2 SKC® cyclone and sampling configuration in the backyard of a house for outdoor PM_{10} collection

polyethylene plastic bag. The whole of each soil sample collected was dried in the laboratory at 40 °C overnight and sieved to <250 μm (Ng et al. 2013). The samples were stored in 120 mL clear polypropylene container (SARSTEDT Australia, South Australia) and kept at room temperature until analysed.

Indoor carpet dust samples that had accumulated in the carpets in houses were collected within the areas of the house frequently accessed by children, such as bedrooms, dining rooms and toy rooms, as recommended by U.S. EPA (2008). A carpet area of 10 m^2 was carefully cleaned with a Dyson stowaway DC-23-MOTORHEAD vacuum cleaner. Dust samples were then stored in a new 120 mL clear propylene (SARSTEDT Australia, South Australia) container. Indoor carpet dust samples were finer than 2 mm and were not sieved. As much of the lint/fluff as possible was removed manually from the sample before storage. A fresh wipe tissue was placed over the screen of the vacuum cleaner exit filter to prevent contamination. The vacuum cleaner was cleaned extensively with wipes between sample collections at different houses. The amount of dust collected at each site was weighed following collection.

Total concentration in samples following aqua regia digestion

All reagents used for sample digestion were obtained after double-distillation of analytical grade (Sigma-Aldrich Chemie GmbH, Steinheim, Germany) unless otherwise stated. Air particulate membrane filters were cut into four equal quarters using a stainless steel scissor. One quarter of each filter was then digested with 2 mL aqua regia (HNO_3/HCl= 1:3) at 140 °C in a hot plate overnight. The concentrations of lead in PM_{10} were determined by using Inductively Coupled Plasma–Mass Spectrometry (ICP-MS, Thermo Elemental X7 series, Waltham, USA). Approximately 1 g of prepared soil and indoor carpet dust samples were weighed and digested in aqua regia following the U.S. EPA method 200.2 (U.S. EPA 1994). The total concentrations of lead in soil and carpet dust were determined by inductively coupled plasma optical emission spectroscopy (Varian Vista, CA, USA).

Blanks and replicates from each batch were analysed for quality control purpose. Precision (as relative standard deviation) of the analysis was determined, based on duplicate analyses of duplicate sampling and analysing for selective samples. The RSDs for lead in eight replicates (n=2) were <11 %. Analytical accuracy was determined using a certified material NIST SRM 1648a (urban particulate matter, NIST, Gaithersburg, USA). About 77 % of lead from NIST SRM 1648a with RSD 7 % (n=5) was recovered by the aqua regia digestion procedure. Detection limits (3 times standard deviation, n=18) for ICP-MS for lead was 10 ng/L. Measurements of all blank filters (n=3) were lower than 178 ng/g for lead.

Lead total concentration analyses were carried out in the geochemistry laboratories of the School of Earth Sciences, The University of Queensland, for air particulate membrane filters and Queensland Health Forensic & Scientific Services (NATA accredited laboratories against ISO 17025) for soil and carpet dust.

In vitro bioaccessibility measurements using simulated gastro-intestinal fluid

The in vitro bioaccessibility (BAc) of lead in collected soil and carpet dust samples was examined using the comprehensive physiologically based extraction test (PBET) to simulate lead ingestion via the gastrointestinal tract conservatively (Ruby et al. 1993; Bruce et al. 2007). The analytical sequence comprised a 1-h gastric phase extraction and a 2-h intestinal phase in a water bath (37 °C) with oxygen-free conditions created by pumping argon gas into the simulation system (Table 1). Hydrochloric acid (reagent grade, 10 M) was used to adjust pH (1.3, 2.5 and 4.0) when making the gastric solution. One molar NaH_2CO_3 were added drop by drop to adjust the pH to 7 for the intestine extraction to mimic the different gastrointestinal phases (Table 1). Extracted solutions were taken from reaction vessels at 20 min, 40 min and 1 h after initiation of reaction at gastric phase. The final extracted solutions from both gastric and intestinal phases were filtered using disposable 0.45 μm syringe filters and diluted for analysis of lead by ICP-MS (Agilent 7500, CA, USA). The final BAc results reported in the current project were the average of both gastric and intestinal phases at different time frames (20, 40 and 60 min for gastric phase; 2 and 3 h for intestinal phase after initiation of reaction). The final extracted solutions were filtered using disposable 0.45 μm syringe filters and diluted for analysis of lead by ICP-MS (Agilent 7500, CA, USA). For each analytical batch, procedural blanks and replicates were included for quality control purposes. The certified reference material (TM 28.3, Environment Canada, Canada) was used to assess accuracy and precision of calibration standards. Reproducibility of bioaccessibility analysis (percent) for lead using PBET was 40 % RSD for the in-house quality control material (n=18), and procedural blank levels of lead were below detection limits. Lead bioaccessibility analyses using PBET for soil and indoor dust samples were conducted at the laboratory of the National Research Centre for Environmental Toxicology, The University of Queensland.

Data processing and analysis

All data were recorded and processed with Microsoft Office 2010 for Windows. The normality test (Shapiro-Wilk test) and non-parametric tests were conducted using the STATISTICA 11 program.

Table 1 Phases of gastro-intestinal simulations using PBET and their pH

Phases	Gastric phase			Intestinal phase
	Fasted-solution 1	Mean-solution 2	Fed-solution 3	Small intestine
pH	1.3	2.5	4.0	7.0
Gastric solution component	1.25 g of pepsin (activity of 800–2,500 units/mg), 0.50 g citrate (Fisher Chemical Co), 0.5 g of malate (Aldrich Chemical Co), 420 μL of lactic acid (Sigma Chemical), 500 μL of acetic acid (Fisher Chemical Co) to 1 L Milli-Q water.	Add 0.07 g bile salts (porcine) and 0.02 g pancreatin (porcine) to each reaction vessel containing gastro phase solution after pH adjustment		

The prediction of blood lead level for children (0–7 years old) was conducted with IEUBKwin v1.1 developed by U.S. EPA (2010). The IEUBK model inserts default values whenever site-specific information was not used (Table 2). The additional site-specific parameters that were used for the blood lead simulation of the study sites are summarised in Table 2. These included parameter inputs on air quality for the study area, dietary lead intake calculated based on the recent Australian total diet study (FSANZ 2003) in the case of no dietary survey conducted in the study area and lead concentration in drinking water for the study area (Table 2).

The site-specific blood lead evaluation using IEUBK also required concentration data from each sampled site, and these outputs are demonstrated in the next section. The term "age-specific" estimation for IEUBK in Table 2 was related to different lead exposure scenarios due to physical and behavioural differences for each age group. That included the differences in time spent outdoor, ventilation rate, dietary lead intake, water consumption, daily soil and dust intake, alternative lead sources and other input details that all related to differences associated with children between the ages of 0 and 7 years. The input values of total lead concentrations and bioaccessibilities for different environment samples (soil, dust, water, food, air) were assumed to be the same for all 0–7-year age groups in terms of IEUBK prediction at each sampling site. The predicted blood lead concentration output was presented for each age group and the geometric mean of results for 0 to 7 age years for each sampled house.

Results

Lead concentrations in environmental media

The Shapiro-Wilk test results ($p < 0.05$) indicate that the total concentration of lead in garden soil and indoor carpet dust showed non-parametric distributions for data sets from all the sampling sites. Total concentration and bioaccessibility (%BAc) of lead in garden soil and indoor carpet dust from 23 sampling sites are given in Table 3. The total lead concentrations in soil samples ranged from 41 to 597 mg/kg (median milligrams per kilogram). Five out of twenty-three samples sites had total lead concentrations above the Australian National Health Investigation Level A (Health Investigation Level A) soil guidelines of 300 mg/kg Pb in soils from residential houses with garden or accessible soil, and schools (NEPC 1999). Three of the sites that exceeded HIL A criterion of lead were located less than 1 km away from the major industrial activities but upwind (Fig. 1). Two of the exceeding houses were located within the lead-enriched shale sequence that is part of the economic mineralisation. The total lead concentrations in indoor carpet dust samples varied from 150 to 3,400 mg/kg. The median total lead concentration (Table 3) in the indoor carpet dust samples was four times higher than that of the soil samples collected from the same house except for two of the samples sites where the lead concentration in carpet dust was over 30 times higher than that in the respective soils. The median lead concentration of all carpet dusts collected from 23 houses was used for IEUBK prediction for four of sampling sites which had no indoor carpet available.

The bioaccessibility adjusted lead concentrations were less than 125 mg/kg for soil and 389 mg/kg for indoor carpet dust samples from all sites (Table 3). The bioaccessibility adjusted lead concentrations had a maximum level of 125 mg/kg for all the soil samples and did not exceed HIL A criterion. The mean(±SE) lead bioaccessibilities were 20 %(±1 %) for soil samples and 16 %(±1 %) for indoor carpet dust samples.

The median level of lead in the total suspended particulate (TSP) from an air monitoring program collected for over 5 years from five monitoring sites across the residential area was 0.07 μg/m^3(Table 4). This median total lead concentration in TSP was applied in the IEUBK model (Table 4). The lead total concentrations in indoor air PM_{10} ranged from 0.01 to 0.25 μg/m^3, with the mean value of 0.05 μg/m^3 (Table 4). The ratio of lead concentrations between indoor and outdoor air PM_{10} varied between 21 % and 93 % (Table 4). The median level of the indoor/outdoor lead total concentration ratio (59 %) was used in the IEUBK blood lead prediction in this study (Table 4).

Table 2 Site-specific parameters for sampling sites using IEUBKwin v1.1 (U.S. EPA 2010)

Input	Parameters	Unit	IEUBK default	Site-specific data	Site-specific data sources	Note
Air data	Indoor air lead concentration (percentage of outdoor)	%	30	59	This study	
	Outdoor air lead total concentration	μg/m^3	0.1	0.07	This study	
	Time spent outdoor	h/day	1–4	IEUBK default		Age-specific
	Ventilation rate	m^3/day	2–7	IEUBK default		Age-specific
	Lung absorption percentage	%	32	IEUBK default		
Dietary data	Dietary lead intake	μg Pb/day	1.95–2.26	3.2–6.7	FSANZ (2003) with conversion to μg/day using mean body weight from U.S. EPA (2011)	Age-specific
	Dietary lead bioavailability	%	50	IEUBK default		
Water	Water lead concentration	μg/L	4	0.6	This study	
	Water consumption	L	0.2–0.59	0.271–0.414	U.S. EPA (2011)	Age-specific
	Water lead bioavailability	%	50	IEUBK default		
Soil/dust	Soil/dust ingestion weighting factor (per cent soil)	%	45	50	enHealth (2012a)	
	Outdoor soil lead concentration	μg/g	200	House-based	This study	
	Indoor carpet dust Pb concentration	μg/g	200	House-based	This study	
	Amount of soil/dust ingested daily	g/day	0.085–0.135	0.06, 0.1	enHealth (2012), U.S. EPA (2011)	Age-specific
	Soil lead bioavailability	%	30	House-based	This study	
	Indoor dust lead bioavailability	%	30	House-based	This study	
Maternal	Mother's blood lead concentration at childbirth	μg Pb/dL	1	1.2	Lesjak (2011)	
IEUBK output	Blood lead geometric mean	μg Pb/dL		House-based	Predicted by IEUBK program	

Table 3 Summary table of sensitivity analysis of IEUBK

IEUBK input parameter	IEUBK default value by U.S. EPA	Sensitivity index[a]	Group
Indoor/outdoor lead concentration ratio (dimensionless)	30	0.00	C
Outdoor air value (μg/m^3)	0.1	0.17	B
Dietary intake (μg/day)	1.95 – 2.26	0.65	B
Water lead (μg/L)	4	0.59	B
Soil lead concentration (mg/kg)	200	1.95	A
Dust lead concentration (mg/kg)	200	2.00	A
Maternal blood lead concentration at childbirth (μg/dL)	1	0.05	C
Alternative lead intake (μg/day)[b]	Not given	1.2	A
Soil bioaccessibility (%)	30	0.42	B
Dust bioaccessibility (%)	30	0.32	B
Water bioaccessibility (%)	50	0.14	B
Diet bioaccessibility (%)	50	0.16	B

[a] The sensitivity index are the standard deviation of predicted blood lead ratios when the scale of certain input parameter increase to ten times of the default value or decrease to 1/10 of the default value, assuming no changes in other parameters but IEUBK default values

[b] Indicates the prediction using 50 % bioavailability of the alternative lead source

IEUBK blood lead prediction

The blood lead level predictions for young children (0–7 years) using IEUBK for all sampling sites are given in Fig. 3. The geometric mean of the predicted blood lead levels varied between 1.5 and 7.1 μg/dL with a mean level of 3.5 μg/dL (95 % CI, 3.0–4.0). None of the sites indicated blood lead levels exceeding 10 μg/dL, as recommended by the Australian Government (NHMRC 2009). There were also slight variations in age-specific blood lead predictions for different age groups. The younger age groups, i.e. 0.5−1 year-, 1–2 year- and 2–3 year-old children, were the most vulnerable groups, as found in an epidemiological study (Lyle et al. 2006).

The IEUBK model simulates a standard normal distribution using the calculated geometric mean and selected geometric standard deviation (GSD). To derive the distribution around the geometric mean, the IEUBK model uses the GSD—a value describing the spread around the geometric mean (GM) of blood lead concentration. GSD measures the relative variability in PbB of children of a specified age, or children from a hypothetical population whose Pb exposures are known. It also encompasses biological and behavioural differences, measurement variability from repeat sampling, variability as a result of sample locations and analytical variability. The recommended default value for GSD (1.6) was derived from empirical studies with young children where both blood and environmental lead concentrations were measured (U.S. EPA 2007b). From the standard normal distribution, the IEUBK model also calculates the probability that a child's blood lead level will exceed a user-selected PbB level of concern (10 μg/dL in this study) according to the equation $z=(\ln(10)-\ln(GM)/\ln(GSD)$, shown as "Exceedance" in Fig. 3.

An effort was made to identify the more important exposure route contributing to the blood lead for young children based on assessing the influence of input data on the magnitude of predicted blood lead concentration using the IEUBK model. The input sources of lead in the IEUBK program were air, diet, water and ingestion of soil and dust (U.S. EPA 2007b). The contributions from these five input sources to the site-specific risk assessment in the study area were plotted based on the IEUBK prediction for all 23 sampling sites (Fig. 4). The median contributions of lead intake from the diet, soil and dust sources compared with total lead exposure were 24 %, 21 % and 54 %, respectively (Fig. 4). In contrast, lead from water and air routes only showed median levels of 1.1 % and 0.9 %, respectively, to predicted blood lead levels in children (Fig. 4).

Table 4 Summary statistics for lead levels in residential exposure media

	Lead total concentration (mg/kg)		Lead bioaccessibility -adjusted concentration (mg/kg)[a]		Lead bioaccessibility (%)	
	Soil	Indoor carpet dust	Soil	Indoor carpet dust	Soil	Indoor carpet dust
n	23	19	23	19	23	19
Mean	208	873	44	122	20	16
SD	147	657	35	75	5	7
Minimum	41	150	5	28	11	6
25th percentile	115	515	17	70	15	13
Median	186	650	30	124	20	14
75th percentile	224	993	59	146	22	19
Maximum	597	3,400	125	389	30	38

[a] Lead bioaccessibility of soil and indoor carpet dust was estimated for gastrointestinal simulation using PBET method

Table 5 Summary table of lead in air particulates

	Lead total concentration in outdoor PM_{10} ($\mu g/m^3$)	Ratio of indoor lead/outdoor lead total concentration (%)	Historical lead total concentration in TSP for 5 year ($\mu g/m^3$)
n	12	8	1,353
Mean	0.05	59	0.39
SD	0.08	24	0.86
Minimum	0.01	21	0.001
25th percentile	0.01	39	0.03
Median	0.02	59	0.07
75th percentile	0.03	74	0.35
Maximum	0.25	93	1.98[a]

[a] 95 percentile level

Sensitivity analysis of IEUBK model (This follows IEUBK blood lead prediction

Sensitivity analysis is a used to determine how "sensitive" a model is to changes in the value of the parameters of the IEUBK model. It was conducted for the pharmacokinetic parameters used in the IEUBK model (Dong and Hu 2012). The geometric standard deviation of IEUBK predicted blood lead levels for young children was U.S. EPA recommended default value of 1.6 %, which was derived from empirical studies with young children where both blood and environmental lead concentrations were measured. In this study, the authors tested the sensitivity of the key IEUBK input parameters to help in characterising uncertainties of blood lead prediction using the IEUBK model, as shown in Table 5. Based on the sensitivity index, the IEUBK input parameters were divided into three groups: (1) Group A is the most sensitive group in which parameters include soil and dust total concentrations and alternative lead ingestion if applicable in a study area; (2) Group B in which the parameters show moderate sensitivities to changes, including outdoor air concentrations, dietary intake, lead concentration in drinking water and bioaccessibilities of lead containing materials; and (3) Group C in which predicted blood lead levels have insignificant changes when the values of the parameters in this groups decrease or increase. These include indoor/outdoor lead concentration ratio and maternal blood lead concentration at childbirth.

Discussion

Potential sources of lead and the relevant risks

This study used the approach of a site-specific human health risk assessment in the context of total lead exposure for children 0–7 years old that are relevant to assess lead exposure at the semi-arid study site. It considered lead

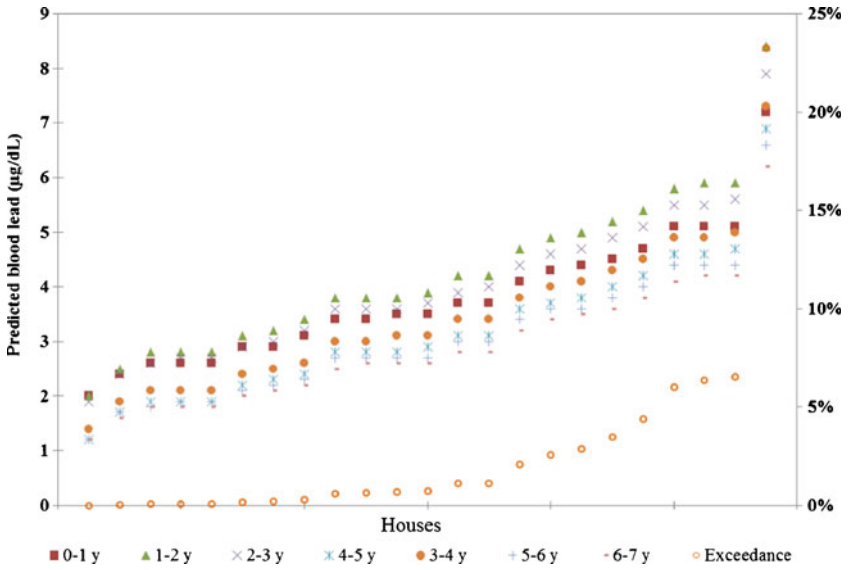

Fig. 3 IEUBK predicted blood lead level geometric mean (micrograms per deciliter) for individual age group (0–1, 1–2, 2–3, 3–4, 4–5, 5–6 and 6–7 years old) and probability of exceedance

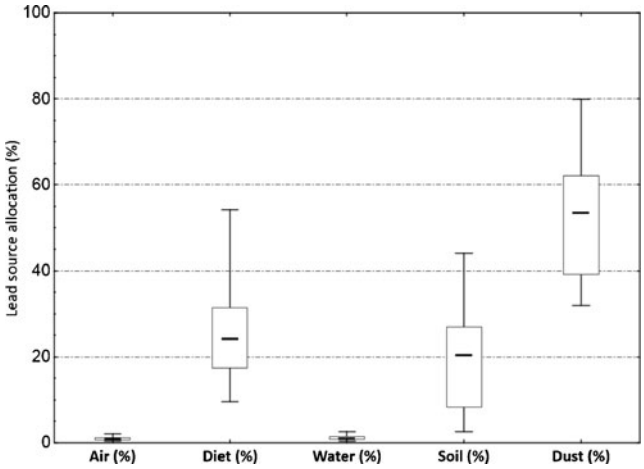

Fig. 4 Lead source allocations of IEUBK blood lead prediction for sampling sites (*solid line* = median, *boxes* = lower and upper quartile and *whiskers* = maximum and minimum values)

intakes from all possible sources, including air, drinking, dietary, soil, dust and alternative sources with the exception of dermal exposure. Since the study sites were located close to mining and mineral processing activities in the semi-arid area, it is important to separate the relevant risks from the natural background and the disturbances evoked by the anthropogenic activities.

There were no blood lead surveys for children living at the study site prior mining activities at this and other semi-arid mining communities in Australia. It is, therefore, impossible to compare the blood lead concentrations of pre- and post-mining activities directly. However, the predicted blood lead levels for samples from the background sites can be used to fill this gap. The Australian central desert dust is characterised by high Al/Si and Fe/Si ratios that indicate a continental origin of dust (Radhi 2010; Radhi et al. 2010). Comparison of the Pb/Si and Si air particulate concentrations for Lake Eyre dust (Radhi 2010) with Pb air particulate concentrations from this study showed that the lowest measured Pb values at the study site approached continental Pb air particulate concentrations. This comparison gave an indication of lead air particulate background on a regional scale. A summary of total Pb concentrations in the natural outcrop and sediments from dry fluvial material at upstream background sites reflect a low natural background, with the mean (SD) total lead concentration of 8.4 (2.7) mg/kg ranging from 5 to 11 mg/kg (n = 4) and median 8.9 mg/kg. The maximum background lead level of 11 mg/kg may generate blood lead geometric mean of 1.1 μg/dL (GSD 1.6 %) based on IEUBK modelling following a site-specific parameter setting (Table 2). The sampled house with minimum lead exposure had predicted blood lead level of 1.5 μg/dL (GSD 1.6 %). These two site-specific predictions for background sites indicated likely blood lead levels under lead-mining free condition at an arid zone location if the children experience certain lead intake as shown in Table 2.

Alternative lead intake sources for children living in the study area could be lead consumption of homegrown food and the lead-containing house paint. The IEUBK model prediction of blood Pb in this study was calculated based on a national intake average as reported in the Australian dietary survey (2003). For children who regularly consumed food grown from home gardens where the lead concentrations in soil were elevated, the lead exposure risk from the dietary route could be higher than the findings in this study. Homegrown food items including vegetables and fruits are not commonly featured in this semi-arid study site, and they were not tested. However, this can be considered should the residential setting change in the future. Unleaded petrol was introduced to Australia in 1986, and leaded-petrol was phased out nationally in 2002 after its first presence in Australia in 1935 (Australian Government 2009). The lead content in domestic paint in Australia was reduced to 1 % in 1965, 0.25 % in 1992 and further decreased to 0.1 % in 1997 from the initial level of 50 % before 1965 (Australian Government 2012). Since most of the houses in the study area were built after late 1960s or renovated in recent times, leaded-paint as an alternative source was not considered.

Potential health risk of total lead exposure to young children

The predicted blood lead levels in the study site (Fig. 4) can be further fit in the following groups: (1) Group PbB >10 μg/dL (above the Australia level of concern); (2) Group PbB 5–10 μg/dL (above U.S. CDC blood lead reference level); and (3) Group PbB<5 μg/dL. The maximum predicted blood lead level was 7.1 μg/dL, and no site was in Group (1). There was one site that exceeded U.S. CDC reference blood level of 5 μg/dL representing Group (2). The majority of sampled sites (95 %) in this study were from Group (3).

Decrements in intelligence quotient (IQ) have been reported (JECFA 2011). Recent cohort studies reported children had impaired intellectual function with blood lead concentrations below 10 μg/dL (Jusko et al. 2008; Lanphear et al. 2005). The exposure level of 0.3 and 1.9 μg/kg b.w. per day are associated with a population decrease of 0.5 IQ point and 3 IQ points (JECFA 2011). The average estimated IQ point decrements were 3.9 (95 % CI, 2.4–5.3), 1.9 (95 % CI, 1.2–2.6) and 1.1 (95 % CI, 0.7–1.5) associated with an increase in blood lead concentrations from 2.4 to 10, 10 to 20 and 20 to 30 μg/dL, respectively (Lanphear et al. 2005). It was also identified that children with a maximal blood lead level <7.5 μg/dL showed greater lead-associated intellectual decrement compared with those with a maximal blood lead level ≥7.5 μg/dL (Lanphear et al. 2005). No safe blood lead level in children has been determined, and children are generally more vulnerable than adults (ATSDR 2007; JECFA 2011).

The last national blood lead survey for Australians was conducted in 1995, and the geometric mean for 1−4-year-old

children (n =1,575) was 5.05 μg/dL (Donovan 1996). There were significant decreases in blood lead concentrations globally after the phase-out of leaded-petrol (U.S. CDC 2013; Schuhmacher et al. 1996; Strömberg et al. 2008; Wu et al. 2011). The comparisons of blood lead levels for children living at the study site and international sites were made in Table 6. The geometric mean and 95 percentile of IEUBK predicted blood lead levels in this study at a mineral-rich region site were relatively higher than the results from recent U.S.A and Canada national blood lead surveys and some European countries (Table 6). The arithmetic mean of IEUBK predicted blood lead levels was much lower than the blood lead test results for children living in arid area like Karachi, Pakistan and a subtropical area like Durban in South African (Table 6). Semi-arid and arid Australia central desert area has been identified as the continental dust source to other cities on the coast (Leys et al. 2011; McTainsh et al. 2005). The spatial relationship between aridity and dust storm activities was also established (McTainsh et al. 1989; Ekström et al. 2004). The significant role of desert was identified to be the strongest source of different dust storms in Australia history (Shao et al. 2007; Knight et al. 1995; Aryal et al. 2012; McTainsh et al. 2005). Conditions with severe soil moisture deficits and reduced vegetation cover were created due to a severe drought and periods of high temperature anomalies prior to the dust events (Shao et al. 2007; McTainsh et al. 2005; Leys et al. 2011; Gabric et al. 2010). The maximum dust load associated the 22–23 October 2002 dust event in Australia was around 5 Mt based on the model estimations, moderate size compared with the magnitude of a northeast Asian dust storm (Shao et al. 2007; McTainsh et al. 2005). These studies indicated that significant dust materials are very likely to be generated at the study area. The relatively higher site-specific blood lead predictions at the study site, compared with the results from recent U.S.A and Canada national blood lead surveys under urban conditions (Table 6), are likely to be associated with the dust from anthropogenic activities in the context of semi-arid climate condition where more airborne particulates can be generated.

Lead source allocations of IEUBK prediction

The lead source allocation calculated using the IEUBK program to estimate children's blood lead level is given in Fig. 4. Lead absorption via inhalation was demonstrated to be less significant compared with the ingestion pathway. Lead from the inhalation pathway showed <2 % of contributions to IEUBK predicted blood lead levels in children for all sampling sites, with a mean of 0.9 % (SE 0.08 %) (Fig. 4). It highlighted the dominant role of the ingestion pathway (soil and dust ingestion) over the inhalation pathway at the study area, despite it being characterised by semi-arid dusty

Table 6 Summary table of blood lead survey in different countries

Location	Australia	U.S.A	Canada	Germany	France	Portugal	Belgium	Karachi, Pakistan	Durban, South African
Screen time	2008	2009–2010	2009–2011	2003–2006	2008–2009	2006–2007	2003–2004	2006–2008	2005
Data source	This study[a]	(U.S. CDC 2013)	(Statistics Canada 2013)	(Kolossa-Gehring et al. 2007)	(Oulhote et al. 2013)	(Smolders et al. 2010)	(Schroijen et al. 2008)	(Ataur Rahman et al. 2012)	(Batterman et al. 2011)
Age group	0.5–7 years	1–5 years	3–5 years	3–5 years	0.5–6 years	1–2 years	14–15 years	6–60 m	8–16 years
n	23	836	495	315	484	120	1679	269	358
Arithmetic mean (μg/dL)	3.5	n/a	n/a	n/a	n/a	2.7	n/a	22	5.3
Standard deviation	1.3	n/a	n/a	n/a	n/a	n/a	n/a	10.4	2.1
Geometric mean (μg/dL)	3.26	1.17	0.93	1.91	1.4	1.9	2.17	n/a	n/a
95 % confidence interval (CI)	0.51	1.08–1.26	0.86–1.0	1.81–2.02	1.27–1.5	1.6–2.2	2.08–2.26	n/a	n/a
50 percentile (95 % CI)	3.2	1.15 (1.03–1.27)	0.93 (0.86–1.0)	1.96	1.3	2.4	2.2	20.5	5.3
95 percentile (95 % CI)	4.91	3.37 (2.63–4.11)	2.1 (1.8–2.3)	3.99	3.28	5	4.54[b]	n/a	9

n/a means data not available

[a] Blood lead levels in children predicted by IEUBK program

[b] Given as 90 % percentile value

climate conditions with active lead mining and mineral processing activities. Limited data for urban scenarios described lead contributions from the inhalation route that were normally less than 6 % of the total (Davies et al. 1990; Dong and Hu 2012). The IEUBK default value for bioavailability of lead via the inhalation pathway is 32 %, which was applied in this study, as shown in Table 2. The application of a site-specific bioaccessibility data was achieved but did not indicate that this is a sensitive parameter of lead total exposure for young children according to sensitivity analysis (Table 3).

The relative contribution of ingestion routes, i.e. water, diet, soil and dust, varied within the same sampling site. Lead from water showed insignificant contributions (<2.6 %) to the predicted blood lead levels in children for all sampling sites (Fig. 4). The other three parameters of lead exposure, namely, diet, soil and dust were the dominant inputs to children's blood lead predictions, as indicated from sensitivity analysis (Table 3). The highest contributions of lead from the diet, soil and dust sources for the different sampling sites were 54 %, 44 % and 80 %, respectively (Fig. 4). The contribution of diet changed for all sampling sites depending on the lead loading in the house, as discussed in previous section. The contribution of diet exceeded that of soil or surface carpet dust for 14 sampling sites (n =23) whereas there were relatively lower lead levels in environmental matrices (soil and indoor carpet dust).

It is worth noting that the contribution of dietary Pb was comparable to that of soil and dust, with a median level of 24 % (Fig. 4). The site which showed the maximum lead contribution from diet (54 %) had 41 % of lead contribution from soil and dust ingestion and only 2 % via inhalation. In comparison, the houses with higher predicted blood lead levels were generally associated with a lesser diet contribution from diet and more significant contribution from soil and dust. The site with minimum lead contribution from diet (0.4 %) had 89 % of lead contribution from soil and dust ingestion. In the case of background site, dietary intake contributed 81 % of total lead exposure, compared with 11 % from the background soil itself. The contributions of diet with maximum and minimum levels were correlated with the lowest and highest predicted blood lead levels for all sampling sites, respectively. In particular, the contribution of dietary compared with the total lead intake ranged from 24 % to 54 % for the predicted blood lead group of 1.5–3.2 μg/dL, which is lower than the median blood lead level predicted by IEUBK. For the group higher than 3.2 μg/dL, the contribution of dietary took only 10 % to 22 %.

The site with maximum dietary Pb contribution also had the lowest contributions from soil and dust ingestion. It also had the lowest predicted blood lead levels for children. Meanwhile, the sites with relatively higher blood lead prediction levels are likely to be linked with higher bioavailable lead concentrations in environmental media. It is therefore clear that the highest children's blood lead levels are driven by lead exposure to environmental media via the ingestion pathways (soil and indoor dust materials), particularly for those houses with heavy lead loadings. The finding from this study further highlights the important role of personal hygiene and house environment in the context of semi-arid climate with particular reference for surface dust for all the residences, especially for children.

Conclusion

The study successfully investigated and quantified the potential lead exposure to young children (0–7 years) living in a mining community in a semi-arid zone from five different lead intake pathways (air, diet, water, soil, and dust) using the IEUBK Model in the absence of the PTWI for lead. The results showed that the ingestion of soils and settled dusts is the driving factor in terms of lead exposure by children in a semi-arid industrial residential location. The lead source contributions from water were lower than 2.6 % of all intake pathways for all sampled houses. The dietary intake of lead contributed up to 54 % of blood lead for children living in the houses with low lead exposure conditions, compared with only <0.4 % for the houses with heavy dust and soil loading of lead, which would normally correlate with higher blood lead levels in children. In particular, the lead absorption via the inhalation pathway was lower than 2 % of the total exposure for all sampling sites.

The following novel aspects were also addressed that relate to the semi-arid condition:

1. The understanding is improved of the lead sources and their contributions to children's blood lead level at a semi-arid location with the presence of current mining and mineral processing activities. Total lead exposure assessment showed that the ingestion of soils and settled dusts is the driving factor in terms of lead absorption by children living at the study site. The dominant contribution of the ingestion pathway to the blood lead concentrations for children living in a semi-arid area like the study site was not clear or well documented until the results of this study were generated. Lead intakes via diet, soil and dust were the dominant inputs to children's blood lead prediction. The inhalation exposure is demonstrated as a less indicative factor of children's blood lead level.
2. It identified the role of dietary to lead intake for children who are exposed to various levels of lead loadings.
3. An approach was explored to estimate the potential lead contribution from the background sites when no blood lead surveys were conducted before mining started at the study area.
4. A sensitivity analysis was conducted for major IEUBK input parameters and identified three levels with various sensitivities when the input values were adjusted at different scenarios.

Assessment of lead exposure via the inhalation route under the site-specific health risk assessment framework is still recommended because, under a shift of the chemical and physical properties of lead-containing particles, it can become more bioavailable and therefore the resulting contributions from inhalation routes can potentially be increased. For a sound understanding of lead exposure via both inhalation and oral ingestion pathways and the influence of bioaccessibility, however, it is recommended to conduct a comprehensive site-specific human health risk assessment by including a survey of local dietary and behaviour profile of children. Personal hygiene and regular house cleaning and maintenance are highly recommended for children living at such study areas. Under a site-specific human health risk assessment framework, a better understanding of the indoor lead levels in the community, the characteristics of the potential fugitive lead-containing dust from the mine site and natural mineralisation sites in the study area should be considered in future studies considering the higher dust level in this semi-arid area.

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References

Aryal R, Kandel D, Acharya D, Chong MN, Beecham S (2012) Unusual Sydney dust storm and its mineralogical and organic characteristics. Environ Chem 9(6):537–546. doi:10.1071/EN12131

Ataur Rahman M, Rahman B, Saeed Ahmad M, Blann A, Ahmed N (2012) Blood and hair lead in children with different extents of iron deficiency in Karachi. Environ Res 118:94–100. doi:10.1016/j.envres.2012.07.004

ATSDR (2007) Toxicological profile for lead. Agency for Toxic Substances & Disease Registry, Atlanta

Australian Government (2009) National phase out of leaded petrol. Department of Sustainability, Environment, Water, Population and Communities, Australian Government. http://www.environment.gov.au/atmosphere/airquality/publications/qa.html. Accessed 13 December 2012

Australian Government (2012) Lead alert facts: lead in house paint. Department of Sustainability, Environment, Water, Population and Communities, Australian Government. http://www.environment.gov.au/atmosphere/airquality/publications/housepaint.html. Accessed 13 December 2012

Batterman S, Su F-C, Jia C, Naidoo RN, Robins T, Naik I (2011) Manganese and lead in children's blood and airborne particulate matter in Durban, South Africa. Sci Total Environ 409(6):1058–1068. doi:10.1016/j.scitotenv.2010.12.017

Bierkens J, Smolders R, Van Holderbeke M, Cornelis C (2011) Predicting blood lead levels from current and past environmental data in Europe. Sci Total Environ 409(23):5101–5110. doi:10.1016/j.scitotenv.2011.08.034

BOM (2012) Climate Statistics for Australian locations. Bureau of Meteorology. http://www.bom.gov.au. Accessed 7 May 2012

Bryant RG, (2013) Recent advances in our understanding of dust source emission processes. Progress in Physical Geography 37: 397–421

Bruce S, Noller B, Matanitobua V, Ng JC (2007) In-vitro physiologically based extraction test (PBET) and bioaccessibility of arsenic and lead from various mine waste materials. J Toxicol Environ Health: Part A 70(19):1700–1711

Carrizales L, Razo I, Tellez-Hernandez JI, Torres-Nerio R, Torres A, Batres LE, Cubillas AC, Diaz-Barriga F (2006) Exposure to arsenic and lead of children living near a copper-smelter in San Luis Potosi, Mexico: importance of soil contamination for exposure of children. Environ Res 101(1):1–10. doi:10.1016/j.envres.2005.07.010

Davies DJA, Thornton I, Watt JM, Culbard EB, Harvey PG, Delves HT, Sherlock JC, Smart GA, Thomas JFA, Quinn MJ (1990) Lead intake and blood lead in two-year-old U.K. urban children. Sci Total Environ 90:13–29

Dong ZM, Hu JY (2012) Development of lead source-specific exposure standards based on aggregate exposure assessment: Bayesian Inversion from biomonitoring information to multipathway exposure. Environ Sci Technol 46(2):1144–1152. doi:10.1021/es202800z

Donovan J (1996) Lead in Australian children: report on the national survey of lead in children. Australian Institute of Health and. Welfare, Canberra

Ekström M, McTainsh GH, Chappell A (2004) Australian dust storms: temporal trends and relationships with synoptic pressure distributions (1960–99). Int J Climatol 24(12):1581–1599. doi:10.1002/joc.1072

enHealth (2004) Environmental health risk assessment—guideline for assessing human health risks from environmental hazards. The Environmental Health Committee (enHealth)

enHealth (2012) Environmental health risk assessment. Guidelines for assessing human health risks from environmental hazards. The Environmental Health Committee (enHealth)

enHealth (2012a) Australian exposure factor guidance handbook. The Environmental Health Committee (enHealth)

enHealth (2012b) Environmental health risk assessment. Guidelines for assessing human health risks from environmental hazards. enHealth

FSANZ (2003) The 20th Australian total diet survey. Food Standards Australia New Zealand

Gabric AJ, Cropp RA, McTainsh GH, Johnston BM, Butler H, Tilbrook B, Keywood M (2010) Australian dust storms in 2002–2003 and their impact on Southern Ocean biogeochemistry. Glob Biogeochem Cycles 24(2), GB2005. doi:10.1029/2009GB003541

Glorennec P, Declercq C (2007) Performance of several decision support tools for determining the need for systematic screening of childhood lead poisoning around industrial sites. Eur J Public Health 17(1):47–52. doi:10.1093/eurpub/ckl091

Hogan K, Marcus A, Smith R, White P (1998) Integrated exposure uptake biokinetic model for lead in children: empirical comparisons with epidemiologic data. Environ Health Perspect 106(Suppl 6)

ICMM (2007) Metals environmental risk assessment guidance. International Council of Mining & Metals

JECFA (2011) Safety evaluation of certain food additives and contaminants. Joint FAO/WHO Expert Committee on Food Additives

Jusko T, Henderson C, Lanphear B, Cory-Slechta D, Parsons P, Canfield R (2008) Blood lead concentrations<10 mu g/dL and child intelligence at 6 years of age. Environ Health Perspect 116:243–248

Knight AW, McTainsh GH, Simpson RW (1995) Sediment loads in an Australian dust storm: implications for present and past dust processes. CATENA 24(3):195–213. doi:10.1016/0341-8162(95)00026-O

Kolossa-Gehring M, Becker K, Conrad A, Lüdecke A, Riedel S, Seiwert M, Schulz C, Szewzyk R (2007) German Environmental Survey for Children (GerES IV)—first results. Int J Hyg Environ Health 210(5):535–540. doi:10.1016/j.ijheh.2007.07.018

Lanphear BP, Dietrich K, Auinger P, Cox C (2000) Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents. Public Health Report

Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, Canfield RL, Dietrich KN, Bornschein R, Greene T, Rothenberg SJ, Needleman HL, Schnaas L, Wasserman G, Graziano J, Roberts R (2005) Low-level environmental lead exposure and children's intellectual function: aAn international pooled analysis. Environ Health Perspect 113:894–899

Lesjak M (2011) Blood lead trends in children aged less than 5 years in Broken Hill 2011. Broken Hill University Department of Rural Health and NSW Government

Lewandowski TA (2009) Modelling chemical exposures in risk assessment. In: Simeonov LI, Hassanien MA (eds) Exposure and risk assessment of chemical pollution—contemporary methodology. NATO Science for Peace and Security Series C-Environmental Security. pp 155–164. doi:10.1007/978-90-481-2335-3_10

Leys JF, Heidenreich SK, Strong CL, McTainsh GH, Quigley S (2011) PM_{10} concentrations and mass transport during "Red Dawn"–Sydney 23 September 2009. Aeolian Res 3(3):327–342. doi:10.1016/j.aeolia.2011.06.003

Lyle DM, Phillips AR, Balding WA, Burke H, Stokes D, Corbett S, Hall J (2006) Dealing with lead in Broken Hill—trends in blood lead levels in young children 1991–2003. Sci Total Environ 359(1–3):111–119. doi:10.1016/j.scitotenv.2005.04.022

McMichael A, Baghurst P, Robertson E, Vimpani G, Wigg N (1985) The Port Pirie cohort study—blood lead concentrations in early childhood. Med J Aust 143(11):499–503

McTainsh G, Chan Y-c, McGowan H, Leys J, Tews K (2005) The 23rd October 2002 dust storm in eastern Australia: characteristics and meteorological conditions. Atmos Environ 39(7):1227–1236. doi:10.1016/j.atmosenv.2004.10.016

McTainsh GH, Burgess R, Pitblado JR (1989) Aridity, drought and dust storms in Australia (1960–1984). J Arid Environ 16:11–22

Mitchell RM, Campbell SK, Qin Y (2010) Recent increase in aerosol loading over the Australian arid zone. Atmos Chem Phys 10(4):1689–1699

NEPC (1999) Schedule B(7a) Health-based investigation levels. National Environment Protection Council

Ng JC, Juhasz A, Smith E, Naidu R (2013) Assessing the bioavailability and bioaccessibility of metals and metalloids. Environ Sci Pollut Res Epub ahead of print. doi:10.1007/s11356-013-1820-9

NHMRC (2009) NHMRC public statement: blood lead levels. Lead exposure and health effects in Australia. National Health and Medical Research Council

NPI (2012) National Pollution Inventory. Department of Sustainability, Environment, Water, Population and Communication, Australian Government. http://www.npi.gov.au. Accessed 8 May 2012

Oulhote Y, Tertre AL, Etchevers A, Bot BL, Lucas J-P, Mandin C, Strat YL, Lanphear B, Glorennec P (2013) Implications of different residential lead standards on children's blood lead levels in France: predictions based on a national cross-sectional survey. International Journal of Hygiene and Environmental Health. In Press (0). doi: 10.1016/j.ijheh.2013.02.007

Pizzol M, Thomsen M, Andersen MS (2010) Long-term human exposure to lead from different media and intake pathways. Sci Total Environ 408(22):5478–5488. doi:10.1016/j.scitotenv.2010.07.077

Radhi M (2010) Physical and Chemical Properties of Australian Continental Aerosols. Doctor of Philosophy, UNIVERSITY OF NEW SOUTH WEALS, Sydney

Radhi M, Box MA, Box GP, Mitchell RM, Cohen DD, Stelcer E, Keywood MD (2010) Size-resolved mass and chemical properties of dust aerosols from Australia's Lake Eyre Basin. Atmos Environ 44(29):3519–3528. doi:10.1016/j.atmosenv.2010.06.016

Ricci PF (2006) Environmental and health risk assessment and management: principles and practices. Springer, London

Ruby MV, Davis A, Link TE, Schoof R, Chaney RL, Freeman GB, Bergstrom P (1993) Development of an in-vitro screening-test to evaluate the in-vivo bioaccessibility of ingested mine-waste lead. Environ Sci Technol 27:2870–2877

Ruby MV, Davis A, Schoof R, Eberle S, Sellstone CM (1996) Estimation of lead and arsenic bioavailability using a physiologically based extraction test. Environ Sci Technol 30(2):422–430

Schroijen C, Baeyens W, Schoeters G, Den Hond E, Koppen G, Bruckers L, Nelen V, Van De Mieroop E, Bilau M, Covaci A, Keune H, Loots I, Kleinjans J, Dhooge W, Van Larebeke N (2008) Internal exposure to pollutants measured in blood and urine of Flemish adolescents in function of area of residence. Chemosphere 71(7):1317–1325

Schuhmacher M, Bellés M, Rico A, Domingo JL, Corbella J (1996) Impact of reduction of lead in gasoline on the blood and hair lead levels in the population of Tarragona Province, Spain, 1990–1995. Sci Total Environ 184(3):203–209. doi:10.1016/0048-9697(96)05102-9

Shao Y, Leys JF, McTainsh GH, Tews K (2007) Numerical simulation of the October 2002 dust event in Australia. J Geophys Res: Atmospheres 112(D8), D08207. doi:10.1029/2006jd007767

Smolders R, Alimonti A, Cerna M, Den Hond E, Kristiansen J, Palkovicova L, Ranft U, Selden AI, Telisman S, Schoeters G (2010) Availability and comparability of human biomonitoring data across Europe: a case-study on blood-lead levels. Sci Total Environ 408(6):1437–1445. doi:10.1016/j.scitotenv.2009.11.025

Sobanska S, Ricq N, Laboudigue A, Guillermo R, Bremard C, Laureyns J, Merlin JC, Wignacourt JP (1999) Microchemical investigations of dust emitted by a lead smelter. Environ Sci Technol 33(9):1334–1339. doi:10.1021/es9805270

Statistics Canada (2013) Blood lead concentrations in Canadians, 2009 to 2011

Strömberg U, Lundh T, Skerfving S (2008) Yearly measurements of blood lead in Swedish children since 1978: the declining trend continues in the petrol-lead-free period 1995–2007. Environ Res 107(3):332–335. doi:10.1016/j.envres.2008.03.007

Tristan E, Demetriades A, Ramsey MH, Rosenbaum MS, Stavrakis P, Thornton I, Vassiliades E, Vergou K (2000) Spatially resolved hazard and exposure assessments: an example of lead in soil at Lavrion, Greece. Environ Res 82(1):33–45. doi:10.1006/enrs.1999.3997

U.S. CDC (2013) Fourth National Report on human exposure to environmental chemicals—updated tables, March 2013

U.S. EPA (1994) Method 200.2—samplepreparation procedure for spectrochemical determination of total recoverable elements. U.S. EPA

U.S. EPA (2007a) AIRTrends 1995 summary—PM_{10}. U.S. EPA. http://www.epa.gov/air/airtrends/aqtrnd95/pm10.html. Accessed 21 July 2011

U.S. EPA (2007b) User's guide for the integrated exposure uptake biokinetic model for lead in children (IEUBK) Windows. U.S. EPA

U.S. EPA (2008) OSWER 9285.7-81. Guidance for the sampling and analysis of lead in indoor residential dust for use in the integrated exposure uptake biokinetic (IEUBK) model. The Office of Solid Waste and Emergency Response

U.S. EPA (2008) OSWER. Office of Soild Waste and Emergency Response. http://www2.epa.gov/aboutepa/about-office-solid-waste-andemergency-response-oswer

U.S. EPA (2010) Integrated Exposure Uptake Biokinetic Model for Lead in Children. U.S. EPA

U.S. EPA (2011) Exposure factors handbook: 2011 Edition. U.S. EPA

WHO (2011) Evaluation of certain food additives and contaminants: seventy-third report of the Joint FAO/WHO Expert Committee on Food Additives. World Health Organization

Wu W-T, Tsai P-J, Yang Y-H, Yang C-Y, Cheng K-F, Wu T-N (2011) Health impacts associated with the implementation of a national petrol-lead phase-out program (PLPOP): evidence from Taiwan between 1981 and 2007. Sci Total Environ 409(5):863–867. doi:10.1016/j.scitotenv.2010.11.024