Human exposure and risk associated with trace element concentrations in indoor dust from Australian homes

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Abstract

This study examines residential indoor dust from 224 homes in Sydney, Australia for trace element concentrations measured using portable X-ray Fluorescence (pXRF) and their potential risk of harm. Samples were collected as part of a citizen science program involving public participation via collection and submission of vacuum dust samples for analysis of their As, Cr, Cu, Mn, Ni, Pb and Zn concentrations. The upper 95% confidence level of the mean values for 224 samples (sieved to <250μm) were 20.2μg/kg As, 99.8μg/kg Cr, 298μg/kg Cu, 247μg/kg Mn, 56.7μg/kg Ni, 364μg/kg Pb and 2437μg/kg Zn. The spatial patterns and variations of the metals indicate high homogeneity across Sydney, but with noticeably higher Pb values in the older areas of the city. Potential hazard levels were assessed using United States Environmental Protection Agency's (US EPA) carcinogenic, non-carcinogenic and Integrated Exposure Uptake Biokinetic (IEUBK) model human health risk assessment tools for children and adults. US EPA hazard indexes (HI) for Cr and Pb were higher than the safe level of 1.0 for children. HI > 1 suggests potential non-carcinogenic health effects. Carcinogenic risks were estimated for As, Cr and Pb whose carcinogenic slope factors (CSF) were available. Only the risk factor for Cr exceeded the US EPA's carcinogenic threshold (1×10⁻⁶) for children. Children aged 1–2 years had the highest predicted mean child blood lead (PbB) of 4.6μg/dL, with 19.2% potentially having PbB exceeding 5μg/dL and 5.80% exceeding 10μg/dL. The Cr and Pb levels measured in indoor dust therefore pose potentially significant adverse health risks to children.

Introduction

Residential indoor dust is customarily considered to be more of a nuisance than a hazard (Lioy et al., 2002). Contemporary advances in the field of exposure science and environmental contaminant studies have involved detailed analysis of residential indoor dust with respect to exposure assessment and human health risk analysis (Whitehead et al., 2011). Recent studies (Kurt-Karakus, 2012; Rasmussen et al., 2013; Whitehead et al., 2014, 2015; Yoshinaga et al., 2014) have demonstrated that human exposure to indoor contaminants is an emerging area of health concern, especially due to the fact that people spend up to 90% of their time indoors (Klepeis et al., 2001; Latif et al., 2011). The home environment can be a source of passive or active exposure to environmental contaminants such as persistent organic contaminants, trace metals, allergens and tobacco smoke (Whitehead et al., 2011). In particular, children are more vulnerable to both acute and chronic environmental contaminant exposures due to their size and early development stage (ATSDR, 2012; Chance, 2001; Lioy et al., 2002).

Researchers have looked to indoor dust for detecting human exposure to a variety of organic, chemical, physical, biological and radiological contaminants (Bornhag et al., 2005; Lioy et al., 2002; Mitro et al., 2016; Pelley, 2017; Rasmussen et al., 2013; Whitehead et al., 2011; Wilford et al., 2005; Van den Eede et al., 2011). Indoor dust is a composite of particulate matter derived from a range of indoor and outdoor sources, which can act as both a sink and transport medium for persistent contaminants such as toxic metals (Deng et al., 2014; Spurgeon et al., 2011; Whitehead et al., 2011). Given that indoor dust can accumulate persistent environmental contaminants over extended time periods, it has potential to be used for retrospective exposure assessment. Moreover, because indoor dust sampling is less invasive than collecting biological samples, such as blood (Whitehead et al., 2011), it can be used as a proxy for estimating human exposures for...
contaminants in indoor environments.

Over the past few decades, many researchers have explored the advantages of using indoor dust for environmental contaminant research (Kurt-Karakus, 2012; Lisiewicz et al., 2000; Rasmussen et al., 2001, 2008, 2013; Rasmussen, 2004; Whitehead et al., 2012, 2013, 2014, 2015; Yoshinaga et al., 2014). These studies have linked human exposure to dust-borne metals by ingestion, inhalation and dermal absorption (Glorennec et al., 2012; Kurt-Karakus, 2012; Rasmussen et al., 2013). Given the toxicity of many metals, their persistence and non-degradability, they have the potential to accumulate in tissues and internal organs of the human body (Zheng et al., 2010). Elevated concentrations of contaminants in the body can affect the central nervous system and act as co-factors, initiators, or promoters of other diseases (Faiz et al., 2009).

Children are typically more susceptible to the impacts of acute and chronic environmental contaminant exposures (ATSDR, 2012; Chance, 2001) because they are exposed to indoor dust accumulated on floors, in soft furnishings and on other hard surfaces. Further, infants and children typically engage in more hand-to-mouth activity than other age groups, which might involve the placement of toys contaminated with dust in their mouth or the consumption of food with contaminated fingers (Darus et al., 2012; Goudarzi et al., 2014). A few studies have explicitly considered human contact with metals in indoor dust in the Australian context (e.g. Chattopadhyay et al., 2003; Gulson et al., 2014; Laidlaw et al., 2014; Simon et al., 2007). However, most human exposure assessment estimates have been based on exposure to soil and outdoor dust (e.g. Dong et al., 2015; Mackay et al., 2013; Rouillon et al., 2017). Indoor dust is thus a common but often overlooked exposure pathway for metals in Australian homes. This paucity of knowledge is the catalyst for this research into trace metal contamination in residential indoor dust, which seeks to better understand the potential hazards that have arisen due to anthropogenic activities.

To address this knowledge gap, this study utilizes Australian data collected as part of a global citizen science program called ‘DustSafe’. The program invites residents to send in their residential vacuum dust for analysis to the program partners and to complete a short questionnaire about their home environment (DustSafe, 2019). Vacuum bag dust received by the DustSafe program is analysed for the following metals and metalloids (hereafter called trace metals): As, Cr, Cu, Mn, Ni, Pb and Zn. Participants are provided a summary report on their results to enable them to make more informed decisions about limiting exposure to toxic metals in their indoor home environment.

This study presents the results of the analysis of indoor dust for their trace metal concentrations from 224 residential dwellings in Sydney, Australia. The study addresses the following questions:

(a) What are the trace metal components of residential indoor dust?
(b) Which trace metals are of greatest concern?
(c) Are there any potential health risks from exposure to the levels of trace metals measured in indoor dust from the Sydney area?
(d) What are the likely impacts of dust Pb concentrations to child PbB levels?

2. Methods and approach

2.1. Engagement with community

The DustSafe program was advertised via the Australian media and through platforms developed for the VegeSafe program, which also utilizes a citizen science approach to assess trace metals in residential soils (VegeSafe, 2019). VegeSafe participants were informed of the new program and a posting was placed on the VegeSafe Facebook portal (https://www.facebook.com/MQVegeSafe/). Interested participants registered online, completed an online consent form and household questionnaire and mailed their residential vacuum dust in a sealed and labelled Ziploc® bag to Macquarie University for XRF analysis.

On completion of analysis for the following elements: As, Cr, Cu, Mn, Ni, Pb and Zn, a summary report was emailed to each participant. The elements As, Cr, and Pb were selected for analysis because they are among the trace metals of greatest concern due to their high degree of toxicity (Tchounwou et al., 2012). Copper, Mn, Ni and Zn were also selected due to their high prevalence in indoor dust as identified in previous research in Australia, Turkey and Canada (Chattopadhyay et al., 2003; Kurt-Karakus, 2012; Rasmussen et al., 2001). As part of the report, summary options for remedial intervention were provided to participants to reduce or eliminate future exposure to trace metals where necessary. As part of the summary report, other dust reference values (Rasmussen et al., 2013) and Australian soil guidelines for residential homes (NEPC, 2013) were also provided to participants to allow them to assess, interpret and understand their results in a wider context.

The DustSafe program has ethics approval, Macquarie University Ethics reference number 5201700430.

2.2. Sampling and data collection

The collection of dust in previous studies has relied on vacuuming a known floor area for a specific time duration (Harrad et al., 2008; Rasmussen et al., 2013), which allows for the calculation of loading values (μg/m²). However, for the citizen science program it was determined that asking participants to measure and vacuum only a known limited area would be a significant hurdle to their involvement. Moreover, it would likely introduce too many errors into the sample collection component of the study. As part of the submission, participants are asked to complete a 2–3 minute survey that collects household information (metadata). The information collected covers a range of relevant factors that could potentially influence the exposure of occupants to metals present in their homes. The data collected included the age range of occupants, their occupations, hobbies, smoking habits, type of fuel used indoors for cooking and/or heating, flooring characteristics, vacuuming frequency and type of vacuum cleaner used. In addition, further information potentially relevant to dust production at or nearby the property was also collected including household renovation history, the presence of a waste incinerator, proximity to industry, land use history and the approximate age of the property and primary construction materials.

2.3. Sample treatment/processing

On receipt of the samples at Macquarie University, the Ziploc® bags containing the dust samples were opened and air-dried (minimum 24 h). After air drying, manual removal of pet and human hair and large particles was undertaken before samples were sieved through a stainless steel 250μm sieve with nylon mesh, using a sieve shaker (Octagon 200CL).

2.4. Sample analysis

Sieved dust samples were analysed for trace element concentration using a portable Olympus Premium Delta Innow-X X-ray fluorescence (pXRF) analyser fitted with a 50 kV, 4 W Ta anode X-ray tube and a silicon drift detector. Samples and certified reference materials (CRMs) were measured at 60 s per X-ray beam (180 sec total measurement time) using propriety soil mode with dust calibration created by the project team (Table 1). The instrument reproducibility (QA/QC) was validated using the following CRMs: NIST 2583 (indoor dust), NIST 2584 (indoor dust), NIST SRM 2709a (San Joaquin soil), NIST 2710a SRM (Montana I soil), NIST SRM 2711a (Montana II soil). Other certified reference materials were added for instrument calibration as follows: NIST 2586 (trace elements in soil containing lead from paint), NIST 2587 (trace elements in soil containing lead from paint), NIST 1646a (estuarine sediment), RM 8704 (Buffalo river sediment), CNRC PACS-2 (Marine...
Table 1
Summary calibration data for Pb. *Sample results based on mean of three repeat analyses. Recovery was calculated as a percentage of the CRM (certified reference material) reference value (pXRF value/reference value × 100). Factory data was obtained using the pXRF factory supplied dust calibration mode.

<table>
<thead>
<tr>
<th>CRM</th>
<th>CRM value (mg/kg)</th>
<th>Factory value (pXRF soil mode) (mg/kg)*</th>
<th>Recovery - factory setting recovery (%)</th>
<th>pXRF dust calibration data (mg/kg)*</th>
<th>Dust calibration recovery (%)</th>
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</thead>
<tbody>
<tr>
<td>NIST 2709a</td>
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<td>15.7</td>
<td>90.8</td>
<td>15.2</td>
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<tr>
<td>NIST 2710a</td>
<td>5520</td>
<td>5510</td>
<td>99.8</td>
<td>5735</td>
<td>103.9</td>
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<tr>
<td>NIST 2711a</td>
<td>1400</td>
<td>1387</td>
<td>99.1</td>
<td>1396</td>
<td>99.7</td>
</tr>
<tr>
<td>NIST 2583</td>
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<td>72.2</td>
<td>84.1</td>
<td>77.5</td>
<td>90.2</td>
</tr>
<tr>
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<td>9761</td>
<td>9516</td>
<td>97.5</td>
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<td>430</td>
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<tr>
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<td>3226</td>
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<tr>
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<td>10.1</td>
<td>86.3</td>
<td>12.1</td>
<td>103.4</td>
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<td>RM 8704</td>
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<td>CNRC MESS-2</td>
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<td>22.6</td>
<td>103.2</td>
<td>21.9</td>
<td>100.0</td>
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<tr>
<td>CNRC BCSS-1</td>
<td>22.7</td>
<td>22.7</td>
<td>100.0</td>
<td>23.7</td>
<td>104.4</td>
</tr>
</tbody>
</table>

2.5. Statistical analysis

Descriptive statistics, boxplots and linear regression analysis of trace metal concentrations were compiled using Origin* 2016 and R environment software*. The spatial distribution and of trace element with respect to Sydney central business district (CBD) were determined using ArcGIS version 10.5.1. Metadata were analysed with Microsoft Excel 2016 version with the statistical add-in package Analyze-It. Trace metal concentrations have not been weighted geographically nor corrected for water content (typically 1–3%, Rasmussen et al., 2011). For purposes of statistical analysis, results reporting below the instrument limit of detection (LOD) have been assigned a value of 0.5 the LOD (Supplementary Table S1).

2.6. Health risk assessment model

The US EPA human health risk assessment method was adopted in this study. The US EPA model (2002) was designed to assess the nature and probability of adverse health effects to humans who may be exposed to trace metals in the environment. The US EPA model is designed to address the likely adverse health outcomes from exposure, the probabilities of people experiencing the adverse health effects and whether the current exposure levels pose a health risk to humans.

2.6.1. Chronic daily intake (CDI)

The potential non-carcinogenic health risk due to metal exposure from indoor dust through inhalation, dermal contact and ingestion pathways were calculated using US EPA (2002) model. The Australian Exposure Factor Guidance Document (2011) recommends the use of daily dust/soil ingestion rates of 50 mg/d (day) for children (1–2 years) and 25 mg/d for adults in exposure assessment. The chronic daily intake (CDI) for ingestion, inhalation and dermal exposure routes was estimated from the following Eqs. (1)–(7):

\[
\text{CDI}_{\text{Ingestion}} = C \times \frac{\text{IngR} \times EF \times ED}{\text{BW} \times \text{AT}} \times \text{CF}
\]

(1)

\[
\text{CDI}_{\text{Inhalation}} = C \times \frac{\text{InhR} \times EF \times ET \times ED}{\text{PEF} \times \text{BW} \times \text{AT}}
\]

(2)

\[
\text{CDI}_{\text{Dermal}} = C \times \frac{\text{SA} \times \text{SL} \times \text{ARS} \times EF \times ED}{\text{BW} \times \text{AT}} \times \text{CF}
\]

(3)

Table 2
Input values of variables used for human health risk assessment.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Unit</th>
<th>Definition</th>
<th>Childa</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>mg/kg</td>
<td>Trace metal concentration</td>
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<td>0.001</td>
</tr>
<tr>
<td>ABSd</td>
<td>-</td>
<td>Dermal absorption factor</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>AF/SL</td>
<td>mg/cm²</td>
<td>Soil to skin adherence factor</td>
<td>11</td>
<td>78</td>
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<tr>
<td>BW</td>
<td>Kg</td>
<td>Body weight</td>
<td>2</td>
<td>30</td>
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<tr>
<td>ED</td>
<td>Year</td>
<td>Exposure duration</td>
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<td>350</td>
</tr>
<tr>
<td>EF</td>
<td>d/year</td>
<td>Exposure frequency</td>
<td>17.8</td>
<td>20</td>
</tr>
<tr>
<td>ET</td>
<td>h/d</td>
<td>Exposure time</td>
<td>50</td>
<td>25</td>
</tr>
<tr>
<td>IngR</td>
<td>mg/d</td>
<td>Soil ingestion rate</td>
<td>9.0</td>
<td>15</td>
</tr>
<tr>
<td>InhR</td>
<td>m³/day</td>
<td>Inhalation rate</td>
<td>1600</td>
<td>6700</td>
</tr>
<tr>
<td>CF</td>
<td>kg/mg</td>
<td>Conversion factor</td>
<td>1 × 10⁻⁶</td>
<td>USEPA (1997, 2002)</td>
</tr>
<tr>
<td>DFSd</td>
<td>mg/year/kg/d</td>
<td>Soil dermal contact factor-age-adjusted</td>
<td>115</td>
<td>USEPA (1997, 2002)</td>
</tr>
<tr>
<td>IR</td>
<td>mg/year/kg/d</td>
<td>Soil ingestion rate age-adjusted</td>
<td>82</td>
<td>USEPA (1997, 2002)</td>
</tr>
<tr>
<td>LT</td>
<td>Year</td>
<td>Lifespan</td>
<td>1.36 × 10⁸</td>
<td>USEPA (1997, 2002)</td>
</tr>
</tbody>
</table>

a A child of 1 to 2 years of age.
The definition and values of the variables used in Eqs. (2) to (4) are provided in Table 2. The carcinogenic risk from metals in household dust was calculated using the following equation:

\[
\text{CDI}_{\text{ingestion-ca}} = C \times \frac{IR \times EF}{AT_{\text{ca}}} \times CF \tag{4}
\]

where

\[
IR = \frac{ED_{\text{dust}} \times Ingr_{\text{dust}}}{BW_{\text{child}}} + \left(ED_{\text{dust}} - ED_{\text{dust}}\right) \times Ingr_{\text{dust}} \tag{5}
\]

\[
\text{CDI}_{\text{inhalation-ca}} = C \times \frac{EF \times ET \times ED}{PEF \times 24 \times AT_{\text{ca}}} \times 10^3 \tag{6}
\]

\[
\text{CDI}_{\text{dermal-ca}} = C \times \frac{ABS \times EF \times DFS_{\text{dadj}}}{AT_{\text{ca}}} \times CF \tag{7}
\]

where \(\text{CDI}_{\text{ingestion-ca}}, \text{CDI}_{\text{inhalation-ca}}, \text{CDI}_{\text{dermal-ca}}\) are the carcinogenic (ca) chronic daily intake or dose contacted through oral ingestion (mg/kg/d), inhalation (mg/m³ for non-cancer and µg/m³ for cancer) and dermal contact with dust particles (mg/kg/d), respectively. The definition and values of the variables used in Eqs. (4) to (7) are provided in Table 2.

The trace metal concentration (C), when combined with the values for the exposure factors as listed in Table 2 (The Australian Exposure Factor Guidance Document, 2011; USEPA, 1997, 2002), is assumed to provide an approximation of the ‘reasonable maximum exposure’ (De Miguel et al., 2007; USEPA, 1989). This value is representative of the 95% upper confidence limit (95% UCL) of the mean concentration (C). Calculated results of C 95% UCL of all metals is provided in Supplementary Table S2.

2.6.2. Risk characterisation

Potential non-carcinogenic and carcinogenic risks for children and adults were determined using the USEPA Methods (US EPA, 2005a, 2005b, 2005c, 2011a, 2011b). Non-cancer health effects were estimated using the hazard quotient (HQ) for ingestion, inhalation and dermal routes (Eq. (8)). Hazard indexes (HI), for each potentially toxic element of concern is estimated as the summation of hazard quotients taking into account all routes of exposure (US EPA, 2011b) (Eq. (9)).

The reference dose (RfD) and reference concentration (RfC) (for inhalation of potentially toxic elements in air) are estimates of daily exposures to the human population that are likely to be without appreciable risks of adverse health effects during a lifetime (US EPA, 2011a). The reference dose (RfD) is drawn from literature sources and is detailed in Supplementary Table S1.

\[
HQ = \frac{CDI}{RfD} \tag{8}
\]

\[
HI = \sum HQ = HQ_{\text{ingestion}} + HQ_{\text{inhalation}} + HQ_{\text{dermal}} \tag{9}
\]

Excess lifetime cancer risk is calculated using the following published approaches (Eqs. (10)–(11)) (De Miguel et al., 2007; USEPA, 1997):

\[
\text{Risk} = CDI_{\text{ca}} \times CSF \tag{10}
\]

\[
\text{Total Cancer Risk} = \sum_{k=1}^{4} CDI_k \times CSF \tag{11}
\]

where RfD (Eq. (9)) is the reference dose, and CSF is the chronic slope factor.

2.7. Integrated Exposure Uptake Biokinetic (IEUBK) model child blood lead (PbB) predictions

The IEUBK win1.1 Build 11 was used to predict the blood lead (PbB) levels from the indoor dust sample dataset. The US EPA's IEUBK model is used to predict the risk of elevated PbB for a hypothetical child under the age of seven (US EPA, 2007). This model is a four-step process that mathematically and statistically links environmental Pb exposure to the PbB level for a population of children. The model also estimates the probability that the hypothetical child will have a PbB level greater than or equal to the level associated with adverse health effects (US EPA, 2007). The default IEUBK mean Pb percentage bioavailability of 30% was used. Additionally, the assumed dust/soil mean percentage bioavailability of 50% for Australia was also applied to the IEUBK model to calculate the Australian-specific data. The other input variables modified here are outdoor soil lead concentration, ingestion rate for soil & dust (HIL A), ventilation rate and dietary lead intake (HIL A, HIL B and HIL C). Other variables include the water consumption rate, Pb concentration in drinking water together with 50% bioavailability of Pb in soil and dust, all sourced from Schedule B7 (Appendix C) of the 2013 amendment of the Australian National Environment Protection (Assessment of Site Contamination) Measure 1999 (NEPM, 2013). An age range of 0 to 84 months was used in the modelling.

2.8. Spatial analysis

ArcGIS (version 10.5.1) was used to map the trace metal distributions in indoor dust across the Sydney metropolitan area. The spatial distribution of the US EPA non-carcinogenic hazard indexes (HI) and the modelled child PbB concentrations across Sydney's local government areas (LGA) were displayed using ArcGIS.

3. Results

Trace metal analyses of the 224 residential indoor dust samples are presented below by detailing concentrations in the sampled homes (Section 3.1) along with their spatial distribution (Section 3.2).

3.1. Trace metals levels in indoor dust

A summary of the trace metal concentrations in indoor dust on a dry weight (mg/kg) basis in Sydney is presented in Fig. 1 and Supplementary Table S1. Zinc showed the highest upper 95% UCL of the mean concentration (2437 mg/kg) followed by Pb (364 mg/kg), Cu (298 mg/kg), Mn (247 mg/kg), Cr (99.8 mg/kg), Ni (56.7 mg/kg), and As (20.2 mg/kg). The upper 95% UCL concentration for Pb is above the Australian guideline level of 300 mg/kg for soil Pb levels for residential facilities with garden and accessible soil (NEP, 2013) (Table 3). The upper 95% UCL for all other elements were below the NEP (2013) requirement (Tables S1 & S2). The mean concentrations of trace metals in vacuum dust measured in this study fall within the range of values reported in literature (Table 3). In terms of comparison to global geogenic background values (Callender, 2003), median Zn values were 17 times greater, while Cu and Pb concentrations were approximately 10 and 5 times higher. Arsenic, Cr and Ni also returned median concentrations above global geogenic background values. By contrast, the median concentration of Mn at 194 mg/kg is markedly lower than natural background values of 850 mg/kg. When compared to New South Wales (NSW) background values (Olszowy et al., 1995), median Zn values were 34 times greater, while Cu and Cr concentrations were approximately 14.8 and 5.92 times higher. Lead, Ni and As were 5.09, 4.5 and 2.25 times higher than NSW background values. The median concentration of Mn at 189 mg/kg is however lower than NSW background values of 233 mg/kg. Other variables include the water consumption rate, Pb concentration in drinking water together with 50% bioavailability of Pb in soil and dust, all sourced from Schedule B7 (Appendix C) of the 2013 amendment of the Australian National Environment Protection (Assessment of Site Contamination) Measure 1999 (NEPM, 2013). An age range of 0 to 84 months was used in the modelling.
samples were sieved to <250 μm to mirror the corresponding size fraction measured in the indoor dust samples. The mean concentration for all soil samples relating to an individual property was used to compare to indoor dust at the same location. Indoor vacuum dust metal concentrations were found to be more contaminated than co-located residential garden soils (Supplementary Table S3). The trace metal concentration ratio (CR) values were estimated as the concentration of a particular trace metal observed in indoor dust (mg/kg) divided by the concentration observed in co-located residential garden soil, [C]id/[C]rs (Fergusson and Kim, 1991). The median CR values (n = 90) for each sample were >1.0. (Supplementary Table S6). The CR > 1 suggests that these trace metals have an additional internally generated source other than from outdoor environment (Rasmussen, 2004; Rasmussen et al., 2018; Yaghi and Abdul-Wahab, 2004). For instance, elevated Pb concentrations are often considered to be internally generated due to the presence of lead paint in the house (Fergusson and Kim, 1991). The CR values also indicate that indoor dust trace metal levels were 1.08 to 3.35 times higher than co-located residential garden soil levels (Supplementary Table S4).

Based on linear regression analysis, 70% (r² = 0.70, p < 0.00001) of the concentration of Mn in indoor dust could be explained by the concentration of Mn in residential garden soil. The values for other trace metals were lower: Pb, 35% (r² = 0.35, p < 0.00001); Zn, 28% (r² = 0.28, p < 0.00001); As, 8% (r² = 0.08, p = 0.006); Ni, 4% (r² = 0.04); Cu, 3% (r² = 0.03, p = 0.089) and Cr, 0.4% (r² = 0.004, p = 0.576). Supplementary Fig. S1 provides a linear regression analysis of indoor dust and residential garden soil trace metal concentration.

The arithmetic mean concentration of trace metals were compared to previous studies conducted on indoor dust (Table 3). The arithmetic mean values for Cr and Pb from this study compare well with a similar but smaller study (n = 82) conducted in Sydney (Chattopadhyay et al., 2003). The concentrations of Cu, Mn, Ni and Zn obtained for this study were higher than those obtained by Chattopadhyay et al. (2003). Chattopadhyay et al. (2003) did not report As concentrations. The results for As and Cu from this study are within the range of those reported from the Canadian indoor dust survey (Rasmussen et al., 2013). Sydney indoor dusts contained higher concentrations of Pb and Zn compared to those reported in the Canadian study. Canadian indoor Ni values were greater than those in Sydney: 63 mg/kg vs 51 mg/kg, respectively (Table 3; Supplementary Fig. S2). The levels of the trace metals from this study were higher than those obtained in Istanbul (Kurt-Karakus, 2012), yet within the limits of those obtained in the UK (Turner and Simmonds, 2006) and Egypt (Khoder et al., 2009). Higher mean values were obtained for trace metals in China (Zheng et al., 2013).

Perhaps of most significance is the fact that the mean concentration of Pb in dust (299 mg/kg) is equivalent to the Australian guideline level of 300 mg/kg for standard residential soils (Australia NEPC, 2013). Of the 224 residences samples, 51 (22.8%) contained dust Pb concentrations >300 mg/kg. All the other trace metals on average were below corresponding regulatory soil guideline values.

### 3.2. Health risk assessment

The results for human health risk assessment due to exposure to trace metals in indoor dust are presented in the Supplementary Information (Table S6). The non-carcinogenic hazard indexes (HI) ranged from 2.66 × 10⁻² (Ni) to 1.57 (Cr) for children (Supplementary Table S6). The HI for Pb was 1.03, also greater than unity (i.e. > 1.0). Generally, where the HI of a particular trace metals is above unity, the greater the level of concern (Asante-Duah, 2017; Gržetić and Ghariani, 2008). The estimated HI of all other trace metals were below unity for children with the HI estimates of all trace metals for adults also being less than unity. In terms of assessing the lifetime carcinogenic risk associated with total metal concentrations, slope factors were only available for As, Cr, and Pb (US EPA., 2011a). For children, the estimated total lifetime carcinogenic risks were 4.39 × 10⁻⁵, 1.18 × 10⁻⁴ and 4.28 × 10⁻⁶ for As, Cr and Pb, respectively (Supplementary Table S6) with ingestion posing the greatest risk followed by inhalation and dermal pathways. For adults, the levels were 1.05 × 10⁻⁴, 9.46 × 10⁻⁴, and 7.37 × 10⁻⁶ for As, Cr and Pb, respectively with the ingestion pathway posing the greatest carcinogenic risk followed by ingestion and dermal pathways respectively. The upper 95% UCL of the mean concentration of total trace metal concentration was utilised for the health risk assessment even though only certain forms of some trace metals are considered carcinogenic (e.g. CrVI).

### 3.3. Child blood lead (PbB) predictions

For this study, child PbB were predicted by assuming two bioavailability scenarios: the default US EPA IEUBK bioavailability of 30% and the Australian NEPM default bioavailability of 50% for soil in Australia (NEPM, 2013). The mean PbB estimates of 4.0, 4.6, 4.3, 4.0, 3.3, 2.8 and 2.5 μg/dL were predicted for children aged 0.5–1, 1–2, 2–3, 3–4, 4–5, 5–6, and 6–7 years, respectively (Supplementary Table S8) assuming IEUBK default bioavailability of 30%. Mean PbB were generally higher at 3.3, 4.9, 4.8, 4.2, 4.0, 3.8 and 3.6 for the respective age categories considering the second modelling scenario using the Australia NEPM Pb bioavailability of 50% (Fig. 2 & Supplementary Table S8). The highest mean PbB concentrations were estimated for children aged 1–2 years and 2–3 years for both modelling scenarios (4.6 and 4.3 μg/dL for US EPA IEUBK default bioavailability of 30% with 4.9 and 4.8 μg/dL for Australia NEPM bioavailability of 50%). The spatial distribution of PbB levels are presented in Section 4.2.
Table 3
Comparison of the Sydney indoor vacuum dust arithmetic mean trace metals concentration with global distribution of trace metals in indoor dust. The range of metal concentrations (minimum to maximum) where available are also presented.

<table>
<thead>
<tr>
<th>Location</th>
<th>Sample type</th>
<th>n</th>
<th>Trace metal levels (mg/kg)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>As</td>
<td>Cd</td>
</tr>
<tr>
<td>Sydney, Australia</td>
<td>House vacuum dust</td>
<td>224</td>
<td>17.6</td>
<td>n.a</td>
</tr>
<tr>
<td>Britain</td>
<td>House vacuum dust</td>
<td>52355</td>
<td>&lt;0.1-80.4</td>
<td>n.a</td>
</tr>
<tr>
<td>Ottawa, Canada</td>
<td>House vacuum dust</td>
<td>48</td>
<td>7.3</td>
<td>6.46</td>
</tr>
<tr>
<td>Canada</td>
<td>House vacuum dust</td>
<td>1025</td>
<td>117</td>
<td>279</td>
</tr>
<tr>
<td>Sydney, Australia</td>
<td>House vacuum dust</td>
<td>82 n.a</td>
<td>0.3-109</td>
<td>4.9-425</td>
</tr>
<tr>
<td>Warsaw, Poland</td>
<td>House vacuum dust</td>
<td>27 n.a</td>
<td>93</td>
<td>141</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>32-63μm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>63-125μm</td>
<td></td>
</tr>
<tr>
<td>Istanbul, Turkey</td>
<td>House vacuum dust</td>
<td>31</td>
<td>0.80</td>
<td>5.0</td>
</tr>
<tr>
<td>UK*</td>
<td>House vacuum dust</td>
<td>n.a</td>
<td>1.2</td>
<td>0.4-20</td>
</tr>
<tr>
<td>Japan</td>
<td>House vacuum dust</td>
<td>100</td>
<td>1.02</td>
<td>4.69</td>
</tr>
<tr>
<td>USA</td>
<td>House vacuum dust</td>
<td>6.3</td>
<td>0.6-4.9</td>
<td>0.18-5.62</td>
</tr>
<tr>
<td>Hong Kong</td>
<td>House vacuum dust</td>
<td>n.a</td>
<td>22-38.5</td>
<td>n.a</td>
</tr>
<tr>
<td>Cairo, Egypt</td>
<td>House vacuum dust</td>
<td>n.a</td>
<td>33.1</td>
<td>12-67</td>
</tr>
<tr>
<td>Guangzhou, China</td>
<td>House vacuum dust</td>
<td>n.a</td>
<td>59.0</td>
<td>1712</td>
</tr>
<tr>
<td>*Guideline values</td>
<td>Outdoor soil</td>
<td>100</td>
<td>20</td>
<td>–</td>
</tr>
</tbody>
</table>

n.a. = not available; *Residential A=with garden/accessible soil.
Section 4.3. Section 4.4 provides details of intervention practices provided to participants on how to reduce exposure to trace metals and their potential non-carcinogenic hazards and carcinogenic risks.

3.4. Spatial distribution of trace metals

Indoor dust Pb and Cr concentrations were the only two elements that returned a Hazard Index >1.0. Spatial mapping showed Pb concentrations were generally more elevated in the centre and inner west parts of Sydney (Fig. 3a). The spatial distribution of indoor Pb dust concentrations are similar to surface soil Pb values (Birch et al., 2011; Rouillon et al., 2017), which are considered to reflect the higher consumption of leaded petrol in the older, more populated inner city areas of Sydney (Kristensen, 2015). Lead isotopic composition measurements of deposited aerosols, European honey bees and honey from Sydney showed that former Pb petrol depositions are remobilised as dust, causing contamination of bees and their honey (Zhou et al., 2018). Fig. 3b and Table S5 show that dust Pb concentrations increased with house age (r = 0.510, p < 0.001). With respect to Cr concentrations, there was no systematic spatial distribution with respect to distance from Sydney’s CBD as is evident with Pb (Fig. 3a; Supplementary Fig. S4). The spatial distribution of As, Cu, Mn, Ni and Zn are provided in Supplementary Fig. S3. None of these trace metals exhibited a spatial relationship with distance from Sydney CBD (Fig. 4; Supplementary Fig. S4).

4. Discussion

The potential human health risks from exposure to trace metals in indoor dust are considered in this section. The spatial distribution of trace metals and their potential non-carcinogenic hazards and carcinogenic risks are discussed in Section 4.1 and 4.2, respectively. The predicted PbB concentrations for children of up to 84 months are evaluated in Section 4.3. Section 4.4 provides details of intervention practices provided to participants on how to reduce exposure to trace metals in indoor dust.

4.1. Potential sources of trace metals in indoor dust

Mapping and distance decay analysis of metal concentrations can be used as an initial step for identification and delineation of contaminated areas or zones that can cause potential adverse human health outcomes (Atkinson, 2009; Dayani and Mohammad, 2010). In this study, spatial mapping, distance from the CBD and analysis of metadata were undertaken to provide insight into the possible factors influencing concentrations of trace metals in indoor dust.

The average age of sampled homes in this study was 60 years (n = 219), with 20.6% being ≥100 years. As noted above, older homes are significantly more Pb-contaminated than new homes (Table S5), but further analysis of the participant metadata offered an opportunity to establish the likely causal factors for this relationship. The participant metadata shows that older houses that have undergone extensive renovations in recent years returned the highest concentrations of Pb (95% UCL = 588 mg/kg) compared to newer buildings (< 50 years old) (Pb, 95% UCL = 182 mg/kg). Moreover, buildings aged ≥50 years (n = 11) that have not been recently renovated that reported instances of internal peeling paint had higher average Pb concentrations (Pb (95% UCL = 1732 mg/kg) than homes of the same age (n = 10) that reported no peeling paint (Pb (95% UCL = 1348 mg/kg). In addition to Pb-based paint sources, the accumulation of automobile-related petrol Pb depositions (Kelepertzis et al., 2016; Kristensen, 2015) is another likely contributor to elevated Pb concentrations in household dusts and soils.

Several of the indoor dust trace metals (As, Mn, Pb, Zn) were significantly associated with residential soil metal values from the same location (Fig. 5). This suggests, that exterior sources of these contaminants are contributing to trace metal loads measured inside homes. Contaminants released into the atmosphere from industry, city traffic, top soil remobilisation and building materials (Kumpiene et al., 2011) have the potential to be either deposited directly into homes or tracked-in on footwear (Hunt et al., 2006).

Although indoor trace metal values were consistently higher than corresponding outdoor values (Fig. 5; Supplementary Fig. S1) not all elements were significantly associated. Chromium, Cu and Ni concentrations in indoor dusts were not significantly associated with levels measured in adjoining residential garden soils, indicating that indoors sources may be more important. Potential sources might include contaminants being produced during house renovation, the use of consumer products or different cooking fuels (Kulshrestha et al., 2014). For example, elevated Cr concentrations in indoor dust (Madany et al., 1994; Stern et al., 2010) have been attributed to the use of wood stains between 1910 and 1970 (Stern et al., 2010). The presence of As and Ni in indoor indoors may have a variety of sources including cigarette smoke, fuel consumption, and chemicals used in aerosol sprays (Fishbein, 1998). In addition, consumer products including detergents, paints, pigments, rubber, plastic, house appliances, and computers can also act as sources of significant enrichment of trace metals in indoor dust (Davies et al., 1985).

4.2. Estimation of daily intake of trace metals from indoor dust and potential health risk analysis

The estimated carcinogenic risks and non-carcinogenic HI (Hazard Index) due to trace metals in indoor dust were calculated using the upper 95% UCL mean concentration of all trace metals (Supplementary Table S1). Hazard quotients (HQ) were determined for each exposure route: inhalation, ingestion and dermal contact with HI being the summation of all three exposure routes. As noted, Hazard Index (HI) values less than or equal to unity (HI ≤ 1) are typically assumed as acceptable (Asante-Duah, 2017). The greater the HI value above unity, the greater is the probability of the onset of adverse non-carcinogenic toxicity effects (Asante-Duah, 2017). The HI values for trace metals in indoor dust, in a decreasing order of risk for children, were: Cr > Pb > Mn > As > Zn > Cu > Ni (Supplementary Table S6). The HI values for Cr and Pb were more than unity for children (HI > 1), suggesting potential non-carcinogenic health implications from exposure to these two elements in indoor dust. Previous studies of trace metals in indoor dust have been related to health risks, with increased concentrations associated with greater health risk. For example, Lambear et al. (1996) established a significant correlation between Pb loading rates in surface dust and childhood blood lead (PbB) levels.

Fig. 2. Boxplot of predicted PbB (based on the NEPM bioavailability of 50%) for a hypothetical child exposed to lead levels in indoor dust. Boxplots show the quartile, median, and third quartile. Squares represent the mean concentration. (Outliers excluded)
Using a modelling scenario, Gulson et al. (2014) detected strong associations between PbB and Pb concentrations measured in indoor dust and soil, particularly those in dust fall accumulation. There was however no significant association between blood Mn with any of the predictors studied (Gulson et al., 2014). Gulson et al. (2013) established statistically significant correlations ($r = 0.53, p = 0.20–0.75$) between Pb concentrations in indoor dust and the age of houses. The spatial distribution of indoor dust samples is shown in Fig. 3a, with the correlation in 3b. The local government areas of Sydney are shown in 3c.
PbB isotopic composition ratios and those measured in corresponding indoor dust wipes. Gulson and Taylor (2017) also calculated that PbB would increase by 1.5 μg/dL with an increase of 100 μg/m²/30d of Pb in dust fall, measured via accumulation in petri dishes. Thornton et al. (1990) in a detailed survey in Birmingham showed that dust Pb loading in the home environment is a significant predictor of PbB levels in 2-year old children (Thornton et al., 1990). For adults, the calculated HI values for all trace metals in this study were < 1, indicating concentration levels did not present a significant toxicity risk. The HI indicators for all trace metals for children were 1 to 3 orders higher than those for adults. This is not surprising, given higher dust ingestion rates for children as well as their smaller body mass. The Australian Exposure Factor Guidance Document (2011) estimates the average dust/soil ingestion rate for children at 50 mg/d and 25 mg/d for adults. Considering that children have lower body mass than adults, the ingestion hazard quotient for children is consequently higher. Another factor accounting for the higher calculated HI indicator for children is that they have a higher dermal absorption factor of 0.03 compared to 0.01 for adults, according to the US EPA estimates (USEPA, 1997, 2002) (Table 2).

Hazard indexes for Cr and Pb HI were >1 for children residing in the centre and inner west parts of Sydney. Both Cr and Pb are systemic toxicants that can induce a range of carcinogenic, neurotoxic, hepatotoxic, genotoxic and mutagenic diseases, even at low levels of exposure (Jaishankar et al., 2014; Tchounwou et al., 2012). In addition to carcinogenic effects, Pb, for example, has been demonstrated at low-levels to cause neurological damage, decreased cognitive function, increased blood pressure, cardio-vascular disease; and at higher levels seizures, coma and even death (ATSDR, 2007; Lanphear et al., 2018; NTP, 2016; Reuben et al., 2017). Children are particularly susceptible to Pb exposure, with effects typically expressing as diminished educational outcomes, widening socio-economic achievement gap and inequalities that span across generations (Evans et al., 2015; Gould, 2009; McIain et al., 2013; Miranda et al., 2009; NCHH, 2015; Reuben et al., 2017; Zhang et al., 2013).

The exposure routes considered for estimation of carcinogenic risks were ingestion, inhalation and dermal exposure to indoor dust for both children and adults (Supplementary Table S6). The carcinogenic risks were estimated for As, Cr and Pb because they are either known or are potential carcinogens and their carcinogenic slope factors were available (Supplementary Table S7). According to the US EPA (1996), the acceptable range of carcinogenic risk across all exposures routes is $1 \times 10^{-6}$ to $1 \times 10^{-4}$ (Jia et al., 2018). Total cancer risk can be regarded negligible for values lower than $1 \times 10^{-6}$ and values above $1 \times 10^{-4}$ are significant (Hu et al., 2011; Jia et al., 2018; US EPA, 1996). Thus, with the exception of Cr (children: $1.18 \times 10^{-4}$; adults: $9.46 \times 10^{-5}$) the total carcinogenic risks calculated based on indoor dust trace metal levels analysed for this study were within the

![Fig. 3.](continued)

![Fig. 4.](continued)
acceptable range for children. The ingestion exposure pathway contributed the highest risk to the overall carcinogenic risk assessment for children, followed by dermal and inhalation pathways, respectively. Similar results were obtained from a study of dust in Istanbul, Turkey, by Kurt-Karakus (2012). The risk of inhalation from resuspended As particles of $6.55 \times 10^{-10}$ is negligible compared to the ingestion and dermal routes of exposure for children. Although the sources of potentially toxic elements in indoor dust are not always known (Chattopadhyay et al., 2003), they are typically derived from a combination indoor and outdoor sources (Abdul-Wahab and Yaghi, 2004; Abdul-Wahab, 2006; Jaradat et al., 2004; Latif et al., 2009).

Trace metal concentrations in the smaller size fractions of indoor dust may be more than 3 orders of magnitude compared to those measured in bulk dust (Gulson et al., 1995; Rasmussen et al., 2001; Rasmussen, 2004), posing higher inhalation risks. Therefore, using a smaller dust size fraction rather than the $\leq 250 \mu m$ might result in a higher trace metal concentrations and subsequently higher risk associated with inhalation. Moreover, trace metal toxicity values used to calculate inhalation risks are derived from studies of occupational exposure to fumes, powdered or chemical forms of the elements (De Miguel et al., 2007). Therefore, using $\leq 250 \mu m$ is likely to result in conservative or lower estimates of inhalation exposure.

The US EPA non-carcinogenic and carcinogenic risk assessment model used in this study assumes a 100% bioavailability of all potentially toxic elements. Most toxicity data are also obtained from more bioavailable soluble salt solutions (Lottermoser, 2002). The use of soluble salt derived toxicity data can therefore lead to an overestimation of risk (Freeman et al., 1995; Lottermoser, 2002). Further, an assumption of 100% bioavailability for solid matrices that comprise dust is conservative in regard to protection of human health. The risk estimates calculated in this study for As and Pb were within the acceptable range of $1 \times 10^{-6}$ to $1 \times 10^{-4}$ (Jia et al., 2018; US EPA, 1996) for children. Contribution from other sources to the total exposure for residents would include dietary intake, inhalation of outdoor urban aerosols, occupational exposures and exposure to playground and garden soils, which have not been included in the analyses. The composite of the trace metal data indicate exposure to potentially toxic elements from indoor dust alone should lead to minimal adverse carcinogenic health effects. Nevertheless, it is worth noting that for health risk estimates, exposure factors and toxicity data are characteristically uncertain (De Miguel et al., 2007).

The risk estimates in this study are based on 95% UCL of the mean of potentially toxic elements. The geographical distribution of HI values for Pb concentrations in indoor vacuum dust decrease toward outlying areas in comparison to inner city areas where older buildings are more common (Fig. 6). Concentrations of As and Pb increase with building age ($r = 0.20$, $p = 0.003$; $r = 0.51$, $p < 0.001$) (Supplementary Table S5). It is anticipated that older inner city households would experience higher risks due to higher concentrations of potentially toxic elements, assuming a linear exposure scenario. The spatial distribution of HI values for As, Cr, Cu, Mn, Ni, and Zn are presented in Supplementary Fig. S5.

### 4.3. IEUBK Model PbB estimates

Childhood PbB levels were also predicted by assuming two
bioaccessibility scenarios: the default US EPA IEUBK bioavailability of 30% and the Australian NEPM default bioavailability of 50% for soil in Australia (NEPM, 2013). The detailed estimated PbB level data are presented in Fig. 6 and Supplementary Table S8. According to Laidlaw et al. (2017), reliance on high bioavailability values such as the NEPM default bioavailability of 50% may overestimate PbB levels. Thus, using the US EPA IEUBK default value, which is similar to the absolute bioavailable Pb values used by Laidlaw et al. (2017) for soil, would likely lead to a more conservative and realistic estimate of PbB exposures.

Predicted average PbB levels using mean Pb concentration and 30% US EPA default bioavailability (Supplementary Table S8) were generally lower than the NHMRC investigation level of 5 μg/dL (NHMRC, 2015). For this modelling scenario, we applied the US EPA default IEUBK total outdoor soil Pb concentration, daily soil/dust ingestion rate, child water consumption rate, child inhalation rate, ventilation rate, outdoor air Pb concentration and dietary Pb intake for 0.5–1 year, 1–2 years, 2–3 years, 3–4 years, 4–5 years, 5–6 years and 6–7 years age categories for estimating the PbB concentrations.

Gulson et al. (2018) have indicated the possibility of using alternative dust exposure measurements to provide reliable estimates of PbB in urban settings. The common application of ‘default dust’ values
based on outdoor soil site-specific values, results in predicted PbB ~22% (range 0 to 52%) higher than those based on soil values versus values from petri dish dust data sets (Gulson et al., 2018). We considered a second modelling scenario, where we estimated PbB using Australian specific air Pb levels, water Pb levels, dietary Pb intake, child water consumption rate, child inhalation rate, ventilation rate, and the Australian default soil Pb bioavailability of 50% (NEPM, 2013). The highest mean PbB concentrations were estimated for children aged 1–2 years and 2–3 years for both modelling scenarios (4.6 and 4.3 μg/dL for US EPA IEUBK default model 4.9 and 4.8 μg/dL for Australia specific data; Fig. 2).

The mean Pb concentration of 299 mg/kg measured in residential indoor vacuum dust samples collected for this study is equivalent to the Australian Residential A (Supplementary Table S4) guideline for soil Pb concentrations in residential gardens (NEPC, 2013) of 300 mg/kg. Therefore, the estimated PbB for a hypothetical child were approximately the same for both mean Pb concentration (299 mg/kg) and for the NEPC regulatory limit (300 mg/kg). Approximately 22% of all sampled households have Pb concentration >300 mg/kg, most of which are located in the inner city area (Fig. 3a). The predicted PbB concentration using the mean Pb concentration of 299 mg/kg was approximately 5 μg/dL for a hypothetical child of 1–3 years (Fig. 2). This means that the PbB of children from these 22% self-selected sampled households who are between the ages of 1 to 3 years are likely to present with a PbB > 5 μg/dL.

Based on distance decay analysis of Pb concentrations (Fig. 4), we calculated that 14.3% of children within the age group of 1–2 years and 2–3 years within a 10 km radius from the Central Business District (CBD) of Sydney are likely to have PbB exceeding 5 μg/dL. The probability of a hypothetical child within this 10 km radius having PbB exceeding 5 μg/dL in the other age categories are 8.1% for a 0.5–1 year old child, 13% for 3–4 years and 4–5 years and 12.5% and 11.1% for a child aged 5–6 years and 6–7 years, respectively. The percentage of sampled homes that may have a child of 1–2 years and 2–3 years with modelled PbB exceeding 5 μg/dL at 10–20 km distance from the CBD falls to 7.1% (Fig. 7). At 20–30 km distance from the CBD, this further reduces to 1.79%. This follows the wider trend of inner city populations falls to 7.1% (Fig. 7). At 20–30 km distance from the CBD, this further reduces to 1.79%. This follows the wider trend of inner city population being at a greater risk of exposure to Pb from indoor dust. The predicted PbB for children between the ages of 0.5–1 year, 3–4 years, 4–5 years, 5–6 years and 6–7 years exposed to indoor dust based on Australian specific data were lower but follow a similar trend (Supplementary Fig. S6).

Previous research has indicated that the adverse effects of lead on a child’s intelligence quotient (IQ) exist even at low concentrations, i.e. < 5 μg/dL (Budtz-Jørgensen et al., 2013; NTP, 2016; Reuben et al., 2017). Miranda et al. (2007) found a discernible adverse impact of PbB levels on end-of-grade academic assessment with childhood PbB levels at 2 μg/dL. In addition, other studies have also indicated the adverse effects of lead exposures are proportionately greater at low levels (Canfield et al., 2003; Earl et al., 2016; Lanphear et al., 2005; Rocha and Trujillo, 2019).

4.4. Limiting human exposure

Most trace metal exposures are preventable. As part of the outreach for this citizen science based research, one of the overarching aims was to provide feedback to participants so they could make informed decisions about what options are available where their household dust has elevated trace metals levels. Participants were provided information about the pathways by which trace metals can be tracked into their indoor environments and how they can be reduced (see Report 1 in Supplementary Information for an example of a sample report provided to DustSafe participants).

Turner (2015) and Hunt et al. (2006) estimated that the first 5 steps that residents take inside their front door mobilises about 85% of the outdoor contaminants found inside the home. Remobilisation of tracked-in soil by human movement across hard flooring surfaces increases its redistribution within homes (Hunt et al., 2006). This process leads to widespread contamination of the indoor environment. Closing doors and windows on windy days or during dust storms is a simple procedure a home owner can implement to limit the migration of airborne contaminants into homes. To further reduce the tracking of potentially toxic elements from outdoors into the indoor environment residents are advised to adopt a suite of mitigation strategies (Fig. 8).

The clean-up of dust/soil using approaches such as wet wiping methods appear to be more effective than dry wiping methods for reducing exposure to trace metals in the indoor environment (Hunt et al., 2006).

4.5. Uncertainties and limitations

Several uncertainties affect the health risk assessment output, including actual exposure duration, the ingestion and inhalation rates, and the metal concentration in dust (Yuswir et al., 2015). The comparison of our results with those derived from other studies must also consider differences in sample collection and treatment between studies, including analysis of different particle size fractions.

Although CrVI has been determined elsewhere as the carcinogenic species of Cr, for human health assessment it is assumed that all the Cr present is in the form of CrVI. Although this precautionary approach was applied, it corresponds with other health risk assessments of potentially toxic elements in indoor dust (Kurt-Karakus, 2012). If the estimated risk for the total Cr is within safe or acceptable limits, this would therefore mean the lesser fraction of CrVII would equally be within safe limits and the need for chemical speciation of the Cr components could be avoided (Maseki et al., 2017). Where the risk is exceeded, it may be relevant to investigate the presence of CrVI.

5. Conclusions

This study provides an analysis of selected trace metals (As, Cr, Cu, Mn, Ni, Pb and Zn) in 224 indoor dust samples from Sydney homes and their potential for causing chronic health effects in both adults and children. Zinc was the dominant trace metal detected in indoor dust. The upper 95% UCL of mean concentration of Zn was 2437 mg/kg followed by Pb, Cu, Mn, Cr, Ni and As with values of 364, 298, 247, 99.8, 56.7 and 20.2 mg/kg, respectively. Among all trace metals, only Pb concentrations in 22.3% of dust samples were above the Australian Residential A (Table S4) guideline for Pb of 300 mg/kg. Most of the indoor samples with trace metal concentrations above the regulatory requirement of 300 mg/kg were from inner-city areas. Lead concentrations decreased with distance away from the CBD. Human health risk assessment showed that HI values for Cr and Pb were >1 for children and adults, indicating a potential non-carcinogenic risk. The total carcinogenic risks for As and Pb analysed were within or close to acceptable limits and are unlikely to pose a significant carcinogenic risk for children and adults. The US EPA IEUBK model data indicate that levels of indoor dust Pb concentrations determined in this study could cause child PbB exposures above acceptable levels (>5 μg/dL) in certain locations. Consequently, this may have implications for childhood IQ and educational outcomes.

Declaration of competing interest

The authors declare this study which is part of “The DustSafe: Citizen insights to the composition and risks of household dust” project that was funded by an Australian Government Citizen Science Grant, CSG55984 to M.P. Taylor.

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Fig. 7. Distribution of predicted PbB for a hypothetical child between the ages of (a) 1–2 years and (b) 2–3 years exposed to indoor dust based on Australian specific data.

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STRATEGIES TO REDUCE INDOOR DUST

- ‘No shoes’ policy and keep windows closed.
- Wash hands and face regularly.
- Establish an entry system to capture pollutants and moisture.
- Use wet rag to dust all surfaces.
- Use washable rugs.
- Use vacuum cleaners fitted with HEPA filter.
- Wet mopping instead of dry sweeping.
- Wash dust and soil laden clothes separately.

Fig. 8. Strategies that can be adopted to help limit human exposure to contaminants associated with indoor dust.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2019.105125.

References


