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The burden of disease from pediatric lead exposure at hazardous waste sites in 7 Asian countries

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ABSTRACT

Identification and systematic assessment of hazardous wastes sites in low and middle-income countries has lagged. Hazardous waste problems are especially severe in lower income Asian countries where environmental regulations are non-existent, nonspecific or poorly enforced. In these countries extensive unregulated industrial development has created waste sites in densely populated urban areas. These sites appear to pose significant risks to public health, and especially to the health of children.

To assess potential health risks from chemical contamination at hazardous waste sites in Asia, we assessed 679 sites. A total of 169 sites in 7 countries were classified as contaminated by lead. Eighty-two of these sites contained lead at levels high enough to produce elevated blood lead levels in surrounding populations.

To estimate the burden of pediatric lead poisoning associated with exposure to lead in soil and water at these 82 lead-contaminated sites, we used standard toxicokinetic models that relate levels of lead in soil and water to blood lead levels in children. We calculated blood lead levels, and we quantified losses of intelligence (reductions in IQ scores) that were attributable to lead exposure at these sites.

We found that 189,725 children in the 7 countries are at risk of diminished intelligence as a consequence of exposure to elevated levels of lead in water and soil at hazardous waste sites. Depending on choice of model, these decrements ranged from 4.94 to 14.96 IQ points. Given the restricted scope of this survey and the conservative estimation procedures employed, this number is almost certainly an underestimate of the full burden of disease.

Exposure to toxic chemicals from hazardous waste sites is an important and heretofore insufficiently examined contributor to the Global Burden of Disease.

1. Introduction

Lead is an ancient toxin, and lead poisoning has been recognized since antiquity. The signs and symptoms of acute, high-dose lead toxicity, including abdominal pain, vomiting, encephalopathy and death, were first recorded by the physician Nikander in the second century BC (Landrigan, 1990). For many centuries, lead poisoning was thought to be exclusively a disease of workers exposed occupationally.

The first cases of childhood lead poisoning were described in the early decades of the twentieth century. These initial cases were seen among children in Australia who had ingested chips of lead-based house paint and swallowed dust contaminated by deterioration of this paint. Their exposure reflected the great increase in introduction of lead into consumer products and the environment that began in the late nineteenth and early twentieth centuries and continues to this day. The children in Australia suffered acute toxicity with coma, convulsions and death. At that time childhood lead poisoning was thought to be an acute, self-limited disease from which a child either died or recovered.

In more recent years, medical understanding of the toxicity of lead has greatly expanded. It is now understood that lead can cause toxic injury to the brain and nervous system even at very low levels of exposure that cause no overt signs and symptoms. Moreover, it is now understood that this injury is permanent and irreversible and that there is no current form of medical treatment that can reverse the brain injury caused by lead once this injury has occurred. Brain injury caused by lead results in decreased intelligence (loss of IQ

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points), shortening of attention span, disorders of executive function, increased risk of dyslexia and other learning disabilities, and diminished economic and social productivity over the entire lifespan of an affected person. Both prenatal and early childhood lead exposure can result in demonstrable alterations in attention, behavior and IQ (Bellinger et al., 1987; Hu et al., 2006; Schwartz, 1994). Lead is also toxic to the cardiovascular system and causes increased risk of hypertension, heart disease and stroke. The International Agency for Research on Cancer has declared lead to be a possible human carcinogen (WHO, 2006).

Due to its numerous impacts on human health, especially its well-established, permanent, detrimental effects on neurodevelopment in children, efforts have been made to characterize and to quantify the effects of lead exposure on human health. Mild mental retardation and cardiovascular outcomes resulting from lead exposure have been estimated to account for nearly 1% of the entire global burden of disease (Fewtrell et al., 2004). However, this analysis excluded exposures occurring at toxic waste sites, because there were insufficient data to quantify exposures from this source. Because of this lack of data, the exposure of children to lead from toxic waste sites has not to date been quantified on a global scale.

To begin to address this gap in knowledge, we calculated the burden of disease in children under the age of 4 years associated with exposure to lead at hazardous waste sites in 7 Asian countries (Bangladesh, India, Indonesia, Kazakhstan, Pakistan, Philippines and Thailand). This work was a component of a larger project investigating the global burden of disease caused by toxic chemicals at waste sites in low- and middle-income countries. This paper presents data on the burden of pediatric disease associated with lead exposure at hazardous waste sites and considers the substantial benefit that may result from remediation of waste sites worldwide.

2. Methodology

2.1. The blacksmith institute inventory of polluted sites

Blacksmith Institute is a New York City-based non-governmental organization with expertise in identifying and arranging for the remediation of contaminated sites in low- and middle-income countries (http://blacksmithinstitute.org). From March 2010 to July 2011, with Regional Technical Assistance support from the Asian Development Bank, Blacksmith Institute carried out a regional inventory of contaminated environmental hot spots in 15 countries in Asia. The goal of this inventory was to identify and assess contaminated sites where human health was at risk. The inventory focused predominantly on sites where heavy metals were present and a pathway of human exposure was identified.

This inventory built on previous inventory efforts in the region, funded in part by the European Commission, the United Nations Industrial Development Organization, and Green Cross Switzerland (Ericson et al., 2012). The inclusion of countries in the inventory was based on several criteria including income, governance, and level of industrial development.

Sites were prioritized for assessment based on a number of criteria. Those with industrial chemical, rather than bacterial, contamination formed the focus of the inventory. Additionally, non-point source issues or very small-scale, disparate artisanal industries were excluded. The well-known problems of urban air quality and human sewage effluents were not assessed as part of the inventory.

Assessments were conducted by country-based specialists engaged and trained by Blacksmith Institute. These specialists typically held masters-level or doctoral degrees in one of the physical sciences and often were experienced in environmental site assessment (Ericson et al., 2012). In certain cases, government experts were also involved in the data collection process. International consultants were engaged in the training and coordination of the national consultants as well as in Quality Control and Assurance efforts.

A total of 428 sites were assessed through this program. An additional 251 assessments had been carried out in the preceding year with support from other sources. This analysis, which was based on all 679 sites evaluated through these programs from 2009 to 2011, is included here. The majority of assessments (498) were conducted in three countries: India, Indonesia and the Philippines (Fig. 1). The Regional Technical Assistance Program did not provide funding for site assessments in People's Republic of China. Therefore, assessments and data from this country are not included in this analysis.

Data from all assessments were entered directly into a secure online database. The use of such a database allowed for central review and comparison of all site assessments across the fifteen countries. Environmental health site assessment specialists were employed to ensure data integrity and quality as well for onsite review and validation of selected sites.

2.2. The site assessment protocol

Blacksmith Institute utilized a simplified risk assessment protocol known as the Initial Site Screening protocol in carrying out the inventory. This protocol was designed by several of the authors as well as by Blacksmith's Technical Advisory Board. It is designed to rapidly acquire key details of a polluted site during a field visit. Specifically, the Initial Site Screening identifies a Key Pollutant; a credible human exposure pathway; and general information about the size and demographics of the population at risk. We have described the methodology of the Initial Site Screening and overall project in further detail previously (Ericson et al., 2012).

Lead, chromium, arsenic and pesticides were the key pollutants most commonly identified in the initial site screening. Lead was the key pollutant in 169 sites, making it the most frequently identified chemical hazard in the region. Sites with lead also expose the largest fraction of affected population. Among a total population of 27,549,000 for all sites in the region across a range of contaminants, 5,596,900, or approximately 20%, were at risk of exposure from sites where lead was identified as the key pollutant. Given this high percentage, the initial burden of disease calculation for childhood exposures was based solely on lead.

The Initial Site Screening protocol includes a Hazard Ranking System to allow for comparison between different types of hazards. The Hazard Ranking System was first developed by the MITRE Corporation for the US Environmental Protection Agency's (EPA) Comprehensive Environmental Response, Compensation, and Liability Act program; commonly referred to as the "Superfund" program (Kushner, 1986; NRC, 1994). The Hazard Ranking System is a semi-quantitative assessment of the potential human health impact for chemical agent in landfills or contaminated environments. The metric evaluates three site characteristics; (1) likelihood that a site has released hazardous chemicals in the environment; (2) the toxicity and quantity of waste; and (3) the size of the population affected. Four pathways are scored under the Hazard Ranking System; groundwater, surface water, soil exposure and air exposure. Scores are then calculated for the pathways using a root-mean-square equation to determine the overall site score. Under the US EPA Superfund Program, a Hazard Ranking Score of >28.5 places a waste site on the National Priorities List where dedicated funds are available to ensure proper environmental remediation.

The modified hazard ranking system used in the Initial Site Screening largely mimics the US EPA process (i.e., assesses pollution, pathway and population) but assigns an index ranging from 1 to 10 to each site, with a higher integer indicating a higher severity of public health risk. The revised system relies primarily on a single key pollutant as opposed to US EPA's, which sums the risk from all contaminants present. For this analysis, we used lead as the key pollutant.

Other information collected as part of each Initial Site Screening includes: sampling data; global positioning system coordinates; photographs; maps; stakeholder and ownership information; information on type, source, and quantity of the contaminant; basic topographical details; and a narrative description. Where credible sampling data could not be identified from either government sources or a peer-reviewed journal, environmental samples were collected in the field and analyzed using either laboratory analysis or an INNOV-X Alpha X-Ray Fluorescence instrument. This handheld direct reading, elemental heavy metals analyzer quickly and accurately measures lead in soil and sediment with a lower limit of detection of less than 50 ug/g. When the X-ray fluorescence instrument was unavailable and no historical data were available, field staff collected representative composite samples (US EPA Method SW 846-7000B) and sent them to a regional laboratory with either experience or accreditation in environmental analyses in air, soil or water. The analytical method used was EPA Method 7421 Furnace Atomic Absorption Spectrophotometry analysis or equivalent.

For the purposes of the inventory, the affected population is considered to be the estimated number of people coming into contact with the key pollutant via the dominant exposure pathway. While the dominant pathway was generally apparent—either lead in soil or lead in drinking water—determining the size and demographics of the exposed population was a tedious process that required field staff to quantify the extent of contamination and number of people exposed. Information on population estimates were drawn from a number of sources, including census data, overhead photographic maps, and interviews with government and community members and field surveys.

2.3. Exposure assessment

The health impact of lead exposure at each site was calculated through a two-step process. The initial step involved translating environmental lead levels from the Toxic Sites Identification Program database into expected population-level blood lead levels (BLL). This was accomplished utilizing the US EPA's Integrated Exposure Uptake Biokinetic Model (IEUBK) for Lead in Children (IEUBKwin v1.1 build 11). The IEUBK is a computer application designed to predict a distribution

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of blood lead levels in children between 6 months and 7 years of age based on environmental lead levels (EPA, 1994; White et al., 1998). The IEUBK was initially developed by the US EPA for estimating the effect of emissions from lead smelters and was used to evaluate remediation activities at sites involved in the Superfund program. More recently, this modeling tool has been revised with expert input and used in a variety of settings, including research.

The IEUBK model uses standard biokinetic absorption factors as the default, but also allows users to edit values for absorption of lead from air, soil, water and dietary sources as well as quantities of water ingested by age group, volumes of inhaled air, ands GI/Blood transfer in response to local circumstances. In additional maternal blood lead level can be entered.

The pharmacokinetic uptake values are built into the IEUBK model and applied accordingly. As children grow, however, gastrointestinal uptake, urinary excretion and personal factors such as hand to mouth activity are altered. This pattern of decremental blood lead levels per unit soil lead continues throughout the years and translates to the observed finding that similar lead exposures in children versus adults produce quite different blood lead levels.

For this analysis, default values were used with the exception of the value for ingested dust. The standard value was increased to 400 ug/day to reflect increased absorption of lead from dust and soil from dirt roads and earthen floors in low income countries (Harris and Harper, 2004). Once all the data are entered, the model determines the distribution of blood lead levels at specified ages. For this analysis, all inputs were set to zero (lead in air, lead in diet, maternal blood lead etc.) prior to inputting soil and water data.

2.4. Assessment of health risks

The second part of the risk assessment process involved estimating the impact of elevated blood lead levels from environmental exposures blood on children's cognitive function, specifically intelligence quotient (IQ).

To provide a range of estimates for these calculations, we used the results of two meta-analyses—Schwartz (1994), Lanphear et al. (2005). The Schwartz meta-analysis involved 8 studies investigating over 2700 children from a wide range of socioeconomic classes, with blood lead levels mostly in the teens to lower twenties (Schwartz, 1994). This meta-analysis found that an increase in blood lead level from 10 to 20 μg/dL resulted in a decrease of 2.57 IQ points, with a standard error of 0.41 (95% Confidence Interval, 1.77–3.37). A linear model was found to best predict the impact in this range. Therefore, the projected IQ loss in the blood lead level range from 10 μg/dL to 20 μg/dL can be calculated by multiplying the blood lead level by 0.257.

To investigate the effect of lower blood lead level (i.e., below 10) on IQ, Lanphear et al. analyzed multiple prospective studies involving a total of 1333 children (Lanphear et al., 2005). Taking into account multiple variables, such as birth weight, maternal IQ and education, and the quality of stimulation in the home environment as measured by the Home Observation for Measurement of the Environment Inventory score, a log-linear model was found to best predict the impact on IQ. The equation of this model is: IQ loss = beta * ln (concurrent blood Pb/cutpoint), with a beta of −2.70 (95% Confidence Interval, −3.74 to −1.66) and cutpoint of 1.0 μg/dL. By substituting the estimated blood lead level for each specific site in the Toxic Sites Identification Program database generated by the IEUBK model into these two equations, the decrement in IQ resulting from this level of lead exposure was predicted. For example, a site in India had a soil lead level of 2062 ppm, which corresponded to a blood lead level of 16.5 μg/dL. Entering this blood lead level into the Lanphear and Schwartz formulas resulted in a decrement of 7.6 and 4.2 IQ points, respectively.

In addition to the estimated population at risk of exposure at each site, information regarding age distribution of the population living around each site was obtained from the US Census Bureau's International Data Base. Incorporation of these data enabled calculation of the number of children at risk at each site. Due to specific, overlapping age ranges used by the IEUBK model and the International Data Base, this analysis was restricted to children 0–48 months old. These sources enabled the researchers to estimate the number of children in this age range who are exposed to the type of environmental lead at each site.

3. Results

3.1. Site identification

A total of 428 sites were assessed through the Regional Technical Assistance Program funded by the Asian Developmental Bank, and an additional 251 assessments were carried out in the preceding year with support from other sources. In this analysis we present data from all 679 of the sites assessed from 2009 to 2011 (Table 1).

The majority of the 679 assessments were conducted in three countries: India (51%), Indonesia (11%), and the Philippines (11%). This was expected given the large population size of these countries relative to the others.

Table 1
Environmental site assessments by country.

	2009	2010	2011	Total
Bangladesh*	9	4	12	**25**
Cambodia	2	2	3	**7**
Georgia	0	4	0	**4**
India*	126	213	8	**347**
Indonesia*	32	43	3	**78**
Kazakhstan*	6	5	0	**11**
Kyrgyzstan	1	0	5	**6**
Mongolia	1	2	1	**4**
Nepal	22	12	10	**44**
Pakistan*	12	20	10	**42**
Philippines*	31	25	17	**73**
Sri Lanka	0	3	1	**4**
Tajikistan	1	0	8	**9**
Thailand*	2	2	2	**6**
Vietnam	6	2	11	**19**
Totals	251	337	91	**679****

* Countries used in determining Pediatric Lead Exposure (the 7 Asian countries).
** 428 site assessments were carried out with support from the Regional Technical Assistance Program of the Asian Developmental Bank.

3.2. Environmental lead levels

One hundred sixty nine sites with lead levels above international standards were identified through the inventory. Various environmental media, including soil, air, water, blood and food were tested at each site to determine potential for human exposure. Of the 169 sites assessed, 82 had soil or water levels above current standards.

Soil lead levels were evaluated for 57 sites with the results reported in micrograms per gram (ug/g or ppm). As a reference, the worldwide health-based standard for lead in soil is 400 ug/g in areas where children play or frequent (US ATSDR, 2007). Water lead levels were elevated for 25 sites. Each of these sites had drinking water with lead levels above 150 ug/L. These levels are well above standards for drinking water established by the World Health Organization (10 ug/L) and the US EPA (15 ug/L) (US EPA, 2011; WHO, 2011). These data were entered into the IEUBK model and the predicted blood lead level resulting from such exposures at each site was calculated.

Data from these 82 sites formed the basis for determining the burden of disease with respect to lead poisoning. Of note both the soil and drinking water standards were promulgated when the blood lead level of concern was 10 μg/dL. Given evidence of adverse effects of lead below 10 μg/dL and no established safe threshold, the US Centers for Disease Control recently eliminated the "level of concern" and established a new reference level of 5 μg/dL. This reference level, which is based on the distribution of BLL in the US, is meant to identify lead-exposed children and trigger exposure reduction actions (US CDC, 2012). As government standards regarding soil and water lead levels are updated to reflect the focus on decreasing BLLs to as low as possible, future estimates of the number of children at risk will almost certainly increase (Table 2).

3.3. Population at risk

Data on the size of the affected population at each hazardous waste site did not include a break-down of the population by age. Therefore to estimate the number of children at risk at each site, we obtained information from the US Census Bureau's International

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Table 2
Lead contaminated sites and environmental levels.

Country*	Contaminated sites discovered		Range of environmental lead levels	
	Lead in soil	Lead in water	Lead in soil ($\geq$ 400 ug/g)	Lead in water ($\geq$ 150 ug/l)
Bangladesh	0	1	–	190
India	7	21	400–6909	152–135,000
Indonesia	23	1	425–83,000	840
Kazakhstan	2	0	3399–9296	–
Pakistan	2	0	404–2505	–
Philippines	22	2	721–692981	320–488
Thailand	1	0	143,097	–
TOTAL	57	25	400–692,981	190–135,000

* A total of 210 initial site screenings in these 7 Asian countries with 169 having lead present. 83 of those had levels about current soil and water standards and used in this analysis.

Table 3
Childhood population living near identified lead contaminated sites.

Country	Total population (all ages)	Children at risk (0–4 yr)
Bangladesh	268,900	30,900
India	496,000	50,084
Indonesia	111,300	10,141
Kazakhstan	20,000	1,906
Pakistan	8,000	958
Philippines	776,400	95,217
Thailand	8,000	519
Total	**1,688,600**	**189,725**

Data Base on the per cent of the population of each country in the range of 0–4 years and applied this fraction to the population living near sites contaminated with lead. Through this procedure, we computed that 189,725 children were at risk of lead poisoning at the 82 sites included in this analysis (Table 3).

3.4. Blood lead levels

Relationships between soil lead levels, water lead levels, and blood lead levels were calculated using the Integrated Environmental Uptake and Biokinetic Model (IEUBK) as was described in the methods section. Although IEUBK was designed for integration of lead exposure from multiple sources, for this analysis we set absorption values for all lead sources other than soil and water at zero so that the attributable blood lead levels solely reflected soil/dust or water exposures. Sites with soil lead levels below 400 ug/g lead were not included in the analysis. Sites with soil levels above 30,000 ug/g defaulted to values of 30,000 ppm in the analysis. Similarly sites with water lead values below 144 ug/L were not included. Sites with drinking water lead values above 6030 ug/L defaulted to 6030 ug/L in the analysis.

Table 4 presents estimated blood lead levels in 0–4 year-old children living near hazardous waste sites in 7 Asian countries in relation to soil lead levels. The range extends from a soil lead level of 400 mg/kg producing BLLs of 5.1 ug/dL in 0–4 year old children and 3.3 ug/dL in 5–9 years old children to a soil lead level of 20,000 mg/kg producing BLLs of 63.4 ug/dL and 50.1 ug/dL.

3.5. Loss of intelligence

Table 5 shows the calculated decrements in cognitive function (IQ score) in 0–4 year-old children in relation to a range of blood lead levels. For the lower levels of BLL, such as 2 ug/dL, Schwartz produces a more conservative estimate of the loss of IQ points (0.51) compared to Lanphear (1.87). For BLLs at the higher end, such as 60 ug/dL, the Lanphear estimate (11.05) is lower than the Schwartz estimate (15.42).

Table 4
Expected blood lead levels in relation to soil lead levels (IEUBK).

Soil lead levels (mg/kg)	Predicted blood lead levels (ug/dl) 0–4 years old	Predicted blood lead levels (ug/dl) 5–9 years old
400	5.1	3.3
1000	10.2	6.7
1200	11.2	7.7
1600	14.2	9.7
2000	16.5	11.5
4000	25.4	19.1
6000	32.1	25.0
8000	37.7	29.9
10,000	42.7	34.2
20,000	63.4	50.1

Table 5
Relationship between blood lead level and cognitive (IQ) changes.

Blood lead levels (ug/dl)	IQ deficits in 0–4 years old (points)	
	Lanphea	Schwartz
2	−1.87	−0.51
4	−3.74	−1.03
6	−4.83	−1.54
8	−5.61	−2.06
10	−6.21	−2.57
15	−7.31	−3.86
20	−8.08	−5.14
30	−9.18	−7.71
40	−9.96	−10.28
50	−10.56	−12.85
60	−11.05	−15.42

The final computation involves determination of the number of children with anticipated cognitive (IQ) decrements at each site, at each soil lead level, in each country investigated. Table 6 shows the blood lead levels in each country, from 5.1 ug/dL at the low end to 100 ug/dL at the upper end. India (5.1–100 ug/dL), Indonesia (5.1–81.4 ug/dL), and the Philippines (8.6–81.4 ug/dL) exhibit wide ranges of BLLs due to the corresponding range of environmental lead levels at the sites in these countries. Table 7 includes four different IQ decrement estimates for each country—the low and high estimates from both the Schwartz and Lanphear formulas. For the region, the lower estimated number of IQ points lost is 4.94, while the upper estimate is 14.96 points (Table 7).

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Table 6
Range of blood lead levels and children at risk for 7 asian countries.

	Low BLL	High BLL	Total child population at risk
Bangladesh	12.05	12.1	30,900
India	5.1	100.0	50,084
Indonesia	5.1	81.4	10,141
Kazakhstan	23.0	40.8	1906
Pakistan	5.1	19.5	958
Philippines	8.6	81.4	95,217
Thailand	81.4	81.4	519

Table 7
Impact of lead contaminated site on the cognitive (IQ) changes in children living near sites contaminated with lead.

	IQ decrement associated with low BLL		IQ decrement associated with high BLL		Population at risk (children)
	Lanphear	Schwartz	Lanphear	Schwartz	
Bangladesh	−6.7	−3.1	−6.7	−3.2	30,900
India	−4.4	−1.1	−12.4	−25.7*	50,084
Indonesia	−4.4	−1.1	−11.9	−20.1*	10,141
Kazakhstan	−8.5	−5.9	−9.5	−10.5	1,906
Pakistan	−4.4	−1.1	−8.0	−5.0	958
Philippines	−5.8	−2.2	−11.9	−20.1*	95,217
Thailand	−11.9	−20.1*	−11.9	−20.1*	519
Region Average	**−6.59**	**−4.94**	**−10.32**	**−14.96**	**Total: 189,725**

* Estimated IQ decrements from BLL at the upper range (i.e., BLL 80–100) must be interpreted with caution, as there is uncertainty in the models at the upper extreme.

4. Discussion

The main finding of this analysis is that an estimated 245,949 children between the ages of 0 and 4 years are at risk of exposure to environmental sources of lead at hazardous waste sites in 7 Asian countries. These levels of exposure are high enough to cause acute toxicity and also to produce chronic effects, such as decreased IQ. In the Philippines, for example, the estimated blood lead level resulting from exposure to toxic waste sites in more than 150,000 children ranges from 8.6 ug/dL to 81.4 g/dL. The blood lead levels on the upper end of this spectrum can cause the signs and symptoms of acute lead poisoning, including effects on the hematological and renal systems, and they approach the levels associated with seizures and coma. Levels at the lower end of this range can result in neurocognitive effects.

These numbers are almost certainly an underestimate of the true magnitude of the problem of environmental exposure to lead in Asia because the project was limited to sites evaluated by Blacksmith Institute collaborators and because this analysis did not include effects other than loss of IQ points (e.g., anemia, gastrointestinal effects, death). In addition, we limited the analysis to sites with environmental lead levels above established standards, excluding sites with soil and water lead levels below these standards. Given accumulating evidence of the effects of lead below 10 μg/dL, exposure to lead at sites below these standards may still adversely impact IQ. Also China, the largest country in the region, was not included in the analysis. If remediation of these sites does not occur, then a new cohort of children will be born each year that will be exposed to these levels of environmental lead, resulting in adverse health effects and limiting neurodevelopment.

4.1. Economic impact

Pediatric lead poisoning has a tremendous economic impact in addition to its adverse impacts on human health and quality of life. This impact is quantifiable in terms of increased healthcare costs, increased need for additional educational resources, and lost productivity due to decreases in IQ. A 2002 study by Landrigan et al., 2002 estimated that the annual costs of childhood lead poisoning in the US are $43.3 billion. An updated analysis by Trasande and Liu (2011) that includes new information on the costs of lead poisoning at low doses increased this estimate to $50.9 billion per year for the US

4.2. Limitations of approach

The Blacksmith Institute inventory is an important first step that begins to make progress in addressing the paucity of scientific data on environmental exposure to industrial toxicants in low and middle income countries. Soil and water data collected through the inventory do not reflect the level of assessment common during the review of contaminated sites in wealthier nations. Limited resources for laboratory analysis, access issues, and time available for visits and review all affected the quality of data collected. As a result, a single sample is used to draw conclusions about oftentimes large areas. This limitation is in part mitigated by the focus on sampling a credible human exposure pathway, though there are clear deficits to this approach.

Population estimates are determined based on an array of sources. Detailed census data is often difficult to come by; the resulting approach reflects what is currently known about a given site. This is dealt with here by qualifying estimates as within an order of magnitude. Similarly, demographic information was drawn from national rather than provincial or locality census data. Large countries like India, or disparate countries like the Philippines, have significant differences in demographic trends between regions. This approach ignores those differences to facilitate calculations. The number of sites utilized here (82) represents fewer than half of the total number of sites in the inventory (169). The inventory itself is very limited in its coverage and likely represents fewer than a third of all such sites in the region, with disparities in coverage between and within countries.

The IEUBK model is based on the behavior of US children. It has not been adapted to account for the behavior of the children in the countries reviewed here. Anecdotal evidence indicates the children in the region covered by the Regional Technical Assistance Program are much more likely to come into contact with contaminants present in their environment than children in the US. Contributing factors include time spent outside, lack of footwear, dusty conditions and lack of treated water options. In addition, the calculations involved in projecting the BLL included only one pathway of exposure to lead (soil or drinking water). Thus at a site that had elevations of lead levels in both soil and drinking water, only one pathway was considered and additional absorption from food or from vertical transmission from mother to child in-utero were excluded. As a result we believe the numbers presented here are likely an underestimate of the scale and scope of the problem.

An additional limitation in this analysis that probably leads to further underestimation of the full extent of the problem of lead exposure from hazardous waste sites is that even though lead has been documented in the environment through these sampling techniques, a complete pathway of exposure was not documented at each site. Previous studies and the IEUBK model allow for the projection of blood lead level based on environmental levels, but these are estimates and not true blood lead levels. Furthermore, the estimated IQ decrements associated with blood lead levels at the upper levels are associated with greater uncertainty (US EPA, 2007). None of the papers included in the Lanphear BP, et al. meta-analyses included individuals with a peak blood lead level

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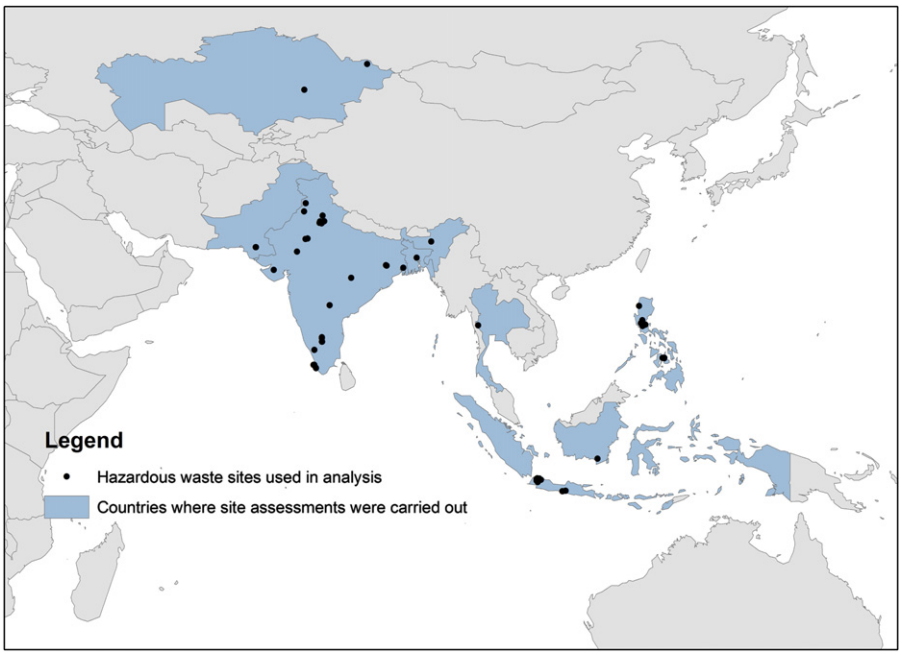

Fig. 1. Countries and hazardous waste sites evaluated.

above 61.5, so results from this meta-analyses are extrapolated to the sites with blood lead level of 81.4 and 100. Even though the BLL-IQ relationship calculated by Schwartz J, 0.257 IQ points lost per 1 unit increase in blood lead level, was associated with the blood lead level range of 10–20 μg/dL, this relationship is often used to project IQ losses beyond the range of 10–20 μg/dL (US EPA, 1998). Regarding the use of IEUBK and lead in water, the model presents age-specific results for each year in a child's life. The results of the first four years of life were averaged here for the purpose of presenting data jointly with levels derived from soil.

While the Lanphear et al. analysis combined multiple prospective studies from several different countries, including the US, Mexico, Yugoslavia, and Australia, none of the included studies were completed in Asian countries. There may be specific, unknown interactions that magnify or reduce the impact of lead on IQ in these populations. For example, Solon et al., 2008 documented a 3.32 IQ point decline with each 1 μg/dL increase in BLL in the Philippines. As the authors highlight, this impact is greater than has been established in more developed countries, even after adjusting for nutritional status. In addition, this paper, like many environmental health studies, evaluates the impact of just one environmental toxicant and does not take into account the potential interactions from different environmental exposures that may mitigate or exacerbate the impact of lead on neuro-cognition.

4.3. Further research

Further research is required to better understand the societal and economic risks posed by pediatric environmental lead exposure in low and middle income countries. The above cited economic impact analyses rely on data and projections from the US and are likely not transferrable to developing countries. This Regional Technical Assistance and the larger Toxic Sites Identification Program provide an important starting point for further analysis, though more identification and assessment work is necessary. With the inclusion of site assessments from People's Republic of China, the impact would most likely be greater.

There is a need to also examine other contaminants at a similar level. Mercury, Chromium, and Cadmium, for instance, compose significant portions of the database. Each has well-documented health effects. It is necessary to develop an appropriate analysis to understand the scale and scope of health effects resulting from exposure to these contaminants and others (Fig. 1).

5. Conclusion

As a first step toward analyzing the burden of disease associated with exposure to toxic waste sites in low and middle income countries, this paper highlights the profound impact that exposure to lead from unregulated hazardous waste sites can have on children's health. Even in this examination of a limited number of sites in only 7 countries, an estimated 189,725 children are exposed to levels of lead in soil and water sufficiently high to produce decreases in IQ. As shown by previous analyses, lead exposure can adversely affect a society in multiple ways, such as by increasing the number of children with mild mental retardation; decreasing the productivity and earnings potential of affected children; increasing demand for educational supports; and increasing healthcare costs. While further research is needed to clarify the exposure and impact of industrial toxicants from waste sites, collaborative efforts should be pursued now to ensure remediation of these sites and a resulting decrease in childhood exposure to lead.

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