

# Racial/Ethnic Differences in Childhood Blood Lead Levels Among Children <72 Months of Age in the United States: a Systematic Review of the Literature

Brandi M. White<sup>1</sup> · Heather Shaw Bonilha<sup>2,3</sup> · Charles Ellis Jr.<sup>4</sup>

Received: 17 December 2014 / Revised: 1 April 2015 / Accepted: 27 April 2015 / Published online: 15 May 2015  
© W. Montague Cobb-NMA Health Institute 2015

**Abstract** Childhood lead poisoning is a serious public health problem with long-term adverse effects. *Healthy People 2020*'s environmental health objective aims to reduce childhood blood lead levels; however, efforts may be hindered by potential racial/ethnic differences. Recent recommendations have lowered the blood lead reference level. This review examined racial/ethnic differences in blood lead levels among children under 6 years of age. We completed a search of PubMed, CINAHL, and PsycINFO databases for published works from 2002 to 2012. We identified studies that reported blood lead levels and the race/ethnicity of at least two groups. Ten studies met inclusion criteria for the review. Blood lead levels were most frequently reported for black, white, and Hispanic children. Six studies examined levels between blacks, whites, and Hispanics and two between blacks and whites. Studies reporting mean lead levels among black, whites, and Hispanics found that blacks had the highest mean blood lead level. Additionally, studies reporting blood lead ranges found that black

children were more likely to have elevated levels. Studies suggest that black children have higher blood lead levels compared to other racial/ethnic groups. Future studies are warranted to obtain ample sample sizes for several racial/ethnic groups to further examine differences in lead levels.

**Keywords** Childhood blood lead levels · Environmental public health · Racial and ethnic disparities

## Introduction

In the USA, pediatric environmental diseases cost an estimated \$80 billion in 2008 with childhood lead poisoning constituting over half of those estimated costs [1]. Consequently, failure to prevent or reduce childhood lead exposures not only increases the likelihood of chronic disease but also threatens the quality of the life of a child and adds a heavy financial burden to our health care system [1].

Toxic exposures during early childhood can have a profound effect on overall development. Among these exposures is lead, a neurotoxin with long-term deleterious effects [2, 3]. Lead most often affects the central nervous system and results in lead poisoning, an acute or chronic toxic condition caused by the absorption of lead into the body by skin contact, ingestion, or inhalation [2, 3]. Children are susceptible to lead because their body structures are still developing [4, 5]. Exposures among children under the age of six are especially concerning given the physical, cognitive, and behavioral development that occurs during that period.

Some researchers have concluded that there is no safe level of lead exposure for children [6]. Elevated childhood blood lead levels (BLLs) are associated with reductions in cognitive function as lead affects the portion of the brain that controls cognitive function [7]. As a result, affected children

✉ Brandi M. White  
whitbm@musc.edu

<sup>1</sup> Department of Health Sciences & Research, College of Health Professions, Medical University of South Carolina (MUSC), 45 Courtenay Drive, SW 974, Charleston, SC 29425, USA

<sup>2</sup> Department of Health Sciences & Research, College of Health Professions, MUSC, 77 President Street, MSC 700, Charleston, SC 29425, USA

<sup>3</sup> Department of Otolaryngology-Head and Neck Surgery, College of Medicine, MUSC, 77 President Street, MSC 700, Charleston, SC 29425, USA

<sup>4</sup> Communication Equity and Outcomes Laboratory, Department of Communication Sciences and Disorders, College of Allied Health Sciences, East Carolina University, 3310H Health Sciences Building, MS 668, Greenville, NC, USA

experience reductions in cognitive skills associated with learning and application of knowledge. Examples of reduction in cognitive performance have been demonstrated in low cognitive test scores and below age/grade level reading and arithmetic scores [7]. Unfortunately, impairments resulting from lead are irreversible and remain present throughout one's life.

Sources of lead exposure among children include lead-based paint, lead particles in dust/soil, drinking water, and consumer goods [8, 9]. The most common source for children is lead-based paint. Particles of paint containing lead in homes built before 1978 pose a major risk to young children [4]. Although lead-based paint was banned in 1978, it remains in some older homes [4, 5].

Policies have been implemented at the national level to mitigate lead exposure [10, 11]. In the 1970s, manufacturers began to phase out lead in gasoline, residential paint, food, and drink cans [12]. As a result, there was a sharp decline in overall childhood BLLs in the USA [11]. Subsequently, lead poisoning prevention funding declined, which in turn could reduce the number of available screening and abatement programs [13]. However, evidence suggests that many children still experience detectable and elevated BLLs [4, 9, 10]. A secondary concern among those studying and monitoring childhood BLLs is that minority children may be at a higher risk for lead poisoning resulting from substandard housing conditions and/or cultural factors [10, 11, 14, 15]. There is a concern that a racial/ethnic disparity exists despite the *Healthy People 2020* environmental health objective to eliminate the number of children under age of 6 years with elevated BLLs and reduce the overall mean BLL to 1.4  $\mu\text{g}/\text{dL}$  [16]. The objective calls for an increase in the number of children screened who live in substandard housing. In response to the objective, public health agencies need to determine if certain racial/ethnic groups should be targeted to reduce lead exposure.

Therefore, this systematic review sought to (1) examine articles reporting BLLs among children under age of 6 years and (2) determine if racial/ethnic differences exist in BLLs. A systematic review offers a preferential focus of the entire body of evidence rather than individual studies and is particularly informative when examining the contribution of factors such as race/ethnicity to a specific outcome [17]. The identification of subpopulations with disproportionately higher BLLs will offer evidence to facilitate targeted screening programs. If needed, this would improve the efficiency of screening programs by allowing for more tailored efforts to specific racial/ethnic groups experiencing detectable and/or elevated BLLs.

## Methods

### Search Strategy

We used the PRISMA guidelines to guide the design, implementation, and reporting of this systematic review, and to

ensure its quality (<http://www.prisma-statement.org/>). We searched the PubMed, CINAHL, and PsycINFO databases using the following (MESH) terms: blood lead poisoning, lead poisoning and toxicity, lead poisoning and adverse effects, blood lead, child, and United States. Two reviewers (BW, CE) assessed titles and abstracts to determine if full-text review was warranted; full-text articles were reviewed to determine final eligibility. We then reviewed the reference sections of the full-text articles to identify additional articles to include in the analysis. All studies that included BLLs ( $\mu\text{g}/\text{dL}$ ) were considered including surveillance reports.

The final inclusion criteria were US studies of children under age of 6 years, with race/ethnicity reported, written in English. The review was limited to studies published from 2002 to 2012. Blood lead levels were chosen as the metric since lead concentrations are most often measured by taking a blood sample. Studies were limited to those conducted in the USA. US studies that mentioned refugees in the titles were excluded. Studies conducted in other countries and US studies of refugees were not reviewed because exposures in other countries may be different than in the USA and might skew findings.

This study did not require review from an ethics review board because existing data was used.

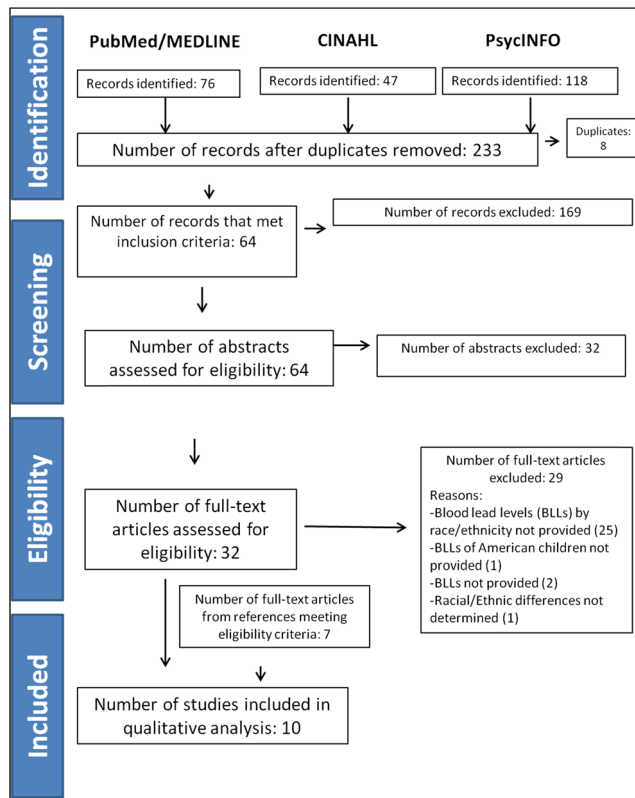
### Data Abstraction and Quality Assessment

Data from articles included in the final analysis were independently abstracted by two reviewers (BW, CE). Study elements abstracted were primary author, study design, data source, years of data collection, age range, race/ethnicity, and baseline mean BLLs or BLL ranges. Study quality was assessed by two reviewers (BW, CE) using an adapted version of the Quality Assessment Tool for Quantitative Studies [18, 19]. Each article was rated on the following components: (1) the overall study design and its appropriateness; (2) the collection and analysis of BLLs, and its validity and reliability; (3) the statistical analyses of lead levels; and (4) whether the sample was representative of the general population.

## Results

### Study Selection

Initially, 241 studies were identified through a preliminary search and 233 studies remained after duplicates were removed. Ten studies met inclusion and exclusion criteria and were included in the final analysis. Six articles reported mean childhood blood levels, one of which was a CDC surveillance report. Four articles reported blood levels by range; one of these articles was also a surveillance report from the CDC. Eight studies reported geometric means and two reported the arithmetic means (Fig. 1).



**Fig. 1** Flow diagram of study selection process

**Descriptive Characteristics**

Seven of the ten articles included in the final review used a cross-sectional study design [20–26]. Five of the ten articles analyzed childhood BLLs from the National Health and Nutrition Examination Survey (NHANES) [20, 21, 23–25]. Several articles provided childhood blood lead data over several time periods ranging from 1988 to 2004 [21, 23, 24, 27–29]. Five of the ten articles analyzed data for a specific community [22, 26], city [28, 29], or state [27]. Age ranges of childhood BLLs were from 0 to 72 months.

Racial/ethnic categories reported varied across articles. All studies in this review reported childhood BLLs for at least two racial/ethnic categories. All studies included data for non-Hispanic black children. One article reported lead levels for six different racial/ethnic groups. Four of the ten articles assessed whether there were statistically significant differences between the racial/ethnic categories in lead levels. Finally, one article examined racial-ethnic differences for one time period (1999–2004), using statistical methods.

**Childhood Blood Lead Levels—Means**

See Tables 1 and 2 for the mean BLL for each article. Six of the ten articles reported mean childhood lead

levels, one of which was a surveillance report (Table 2). Several articles provided the mean childhood lead level; however, not all studies determined whether statistically significant differences existed between racial/ethnic groups. The arithmetic means of BLLs by racial/ethnic groups for select studies that provided this information across all age groups (Tables 1 and 2) were 2.28 µg/dL for white/Caucasian children, 4.55 µg/dL for black children, and 4.07 for Hispanic and Mexican American children.

In an examination of children residing at a US military installation in 1991, Stroop and colleagues found racial/ethnic differences [26]. White children under 1 year had the highest lead level compared to black and Hispanic children (2.3, 2.0, and 1.2 µg/dL, respectively). Black children 1 to 2.9 years and 3 to 5.9 years had the highest lead levels (2.3 and 2.1 µg/dL, respectively) compared to white (2.1 and 1.9 µg/dL) and Hispanic children (2.2 and 1.6 µg/dL) for these age groups. This study was limited in that it focused only on children living on a military base, a subpopulation that faces unique exposures compared to the general population. Leighton and colleagues obtained baseline lead levels from children 10 to 72 months in New York City from 1994 to 1996 [28]. Hispanic children had significantly higher BLLs ( $p < 0.01$ ; 25.4 µg/dL) compared to black children (23.7 µg/dL) and other races (Asian, white, and race unknown; 24.5 µg/dL). It is important to note that children included in this study initially had elevated BLLs.

Jones and colleagues reported childhood lead levels for multiple years (1988–1991 and 1991–1994) using NHANES data among children 1 to 5 years of age but did not report whether there were statistically significant differences [23]. For each time period, black children had the higher BLL when compared to Mexican-American and white children (1988–1991, 5.5, 3.9, and 3.1 µg/dL, respectively; 1991–1994, 4.3, 3.1, and 2.3 µg/dL, respectively). Another study [25] used NHANES data from 1988 to 1994 and 1999–2004 for children 1 to 5 years of age. They found racial/ethnic differences with black children having the highest lead level for both time periods (4.18 and 2.27 µg/dL, respectively), followed by Mexican-American children (3.10 and 1.68 µg/dL) and white children (2.80 and 1.65 µg/dL) [25]. While NHANES provides continuous data on childhood BLLs, because it is a national survey, it may not be able to capture regional differences.

Some reports were designed to statistically measure racial/ethnic differences in mean BLLs. Joseph and colleagues examined racial/differences in BLLs between white and black children 1 to 3 years old while examining the association of lead levels and asthma risk from 1995 to 1998 [27]. Black

**Table 1** Racial/ethnic differences in mean childhood blood lead levels

Primary author	Study design	Data source	Location	Sample size	Year	Race/ethnicity	Age range	Blood lead level <sup>a</sup> (µg/dL)	Significance	Quality score		
Stroop <sup>26</sup>	CS	Military base	Fort Devens, Massachusetts	646	1991	Black	<1 years	2.0	Not calculated	Fair		
						Hispanic		1.2				
						White		2.3				
						Black	1–2.9 years	2.3				
						Hispanic		2.2				
						White		2.1				
						Black	3–5.9 years	2.1				
						Hispanic		1.6				
						White		1.9				
						Black	10–72 months	23.7			Not calculated	Fair
Hispanic		25.4										
Leighton <sup>28</sup>	RCO	Registry	New York City	221	1994–1996	Other		24.5	Not calculated	Fair		
						Black	1–3 years	5.5				
Joseph <sup>27</sup>	PCO	Organization	SE Michigan	4634	1995–1998	Caucasian		3.2	p<0.01	Good		
						Black, NH	1–5 years	5.2				
Jones <sup>23</sup>	CS	NHANES	National	2232	1988–1991	Mexican-American		3.9	Not calculated	Fair		
						White, NH		3.1				
						Black, NH	1–5 years	4.3				
						Mexican-American		3.1				
						White, NH		2.3				
						Black, NH	1–5 years	2.8			p<0.05 (MA and white, NH)	
						Mexican-American		1.9				p<0.05 (black, NH)
						White, NH		1.7			p<0.05 (black, NH)	
						Black, NH	1–5 years	2.80				Not calculated
						Mexican-American		4.18				
Scott <sup>25</sup>	CS	NHANES	National	3517	1988–1994	Mexican-American		3.10	Not calculated	Fair		
						White, NH		1.65				
						Black, NH	1–5 years	2.27				
						Mexican-American		1.68				

The quality score measures the following: (1) the overall study design and its appropriateness; (2) the collection and analysis of FBLLs, and its validity and reliability; (3) the statistical analyses of lead levels; and (4) whether the sample was representative of the general population

CC case-control study, CS cross-sectional study, MA Mexican-American, NH not hispanic, PCO prospective cohort study, RCO retrospective cohort study

<sup>a</sup> Geometric mean reported for all studies except Joseph et al<sup>27</sup> which reports the arithmetic mean

**Table 2** Racial/ethnic differences in mean childhood blood lead levels, multi-year surveillance report

Primary author	Location	Sample size	Year	Race/ethnicity	Age range	Blood lead level <sup>a</sup> (µg/dL)	Significance	Quality score		
MMWR <sup>21</sup>	National	13,472	1991–1994	White, NH	≥1 year	2.2	$p < 0.05$ (black, NH and Mex Am)	Fair		
				Black, NH		2.8	$p < 0.05$ (white, NH)			
				Mexican-American		2.4	$p < 0.05$ (white, NH)			
				2392	1991–1994	White, NH	1–5 years		2.3	$p < 0.05$ (black, NH and Mex Am)
		Black, NH				4.3	$p < 0.05$ (white, NH and Mex Am)			
		Mexican-American				3.1	$p < 0.05$ (white, NH and Black, NH)			
				16,825	1999–2002	White, NH	≥1 year		1.5	$p < 0.05$ (black, NH)
		Black, NH				1.8	$p < 0.05$ (white, NH)			
		Mexican-American				1.6	ns			
			1160	1999–2002	White, NH	1–5 years	1.8		$p < 0.05$ (black, NH)	
	Black, NH				2.8	$p < 0.05$ (white, NH and Mex Am)				
	Mexican-American				1.9	$p < 0.05$ (black, NH)				

The quality score measures the following: (1) the overall study design and its appropriateness; (2) the collection and analysis of BLLs, and its validity and reliability; (3) the statistical analyses of lead levels; and (4) whether the sample was representative of the general population

Mex Am Mexican-American, MMWR Morbidity and Mortality Weekly Report, NH non-Hispanic, ns not significant

<sup>a</sup> Geometric mean reported

children had significantly higher BLLs compared to white children (5.5 and 3.2 µg/dL, respectively) ( $p < 0.01$ ). This study had a small sample of white children, making it difficult to accurately measure differences across racial groups. Although Jones and colleagues did not calculate statistical differences between racial/ethnic categories for all time periods, they did use statistical methods to examine differences across racial/ethnic groups between 1999 and 2004 for children 1 to 5 years of age [23]. They found that the lead level for black children (2.8 µg/dL) was significantly higher compared to that for whites (1.7 µg/dL;  $p < .001$ ) and Mexican-Americans (1.9 µg/dL;  $p < .0001$ ). A multivariable logistic regression model also indicated that the odds of having a BLL  $\geq 10$  µg/dL was over two times higher for black children compared to that for white children ( $p < .0001$ ).

The CDC's Morbidity and Mortality Weekly Report (MMWR) also found statistically significant racial/ethnic differences across multiple years (1991–1994 and 1999–2002) for children  $\geq 1$  year and 1 to 5 years [21]. Compared to white and Mexican-American children, black children had significantly higher BLLs for both time periods and age groups ( $p < 0.05$ ). From 1991 to 1994, black children  $\geq 1$  year had a mean lead level of 2.8 µg/dL compared to 2.4 µg/dL for Mexican-American children and 2.2 µg/dL for white children. This study used NHANES data.

### Childhood Blood Lead Levels—Ranges

Four of the ten articles provided childhood BLLs by range rather than mean BLLs. Raymond and colleagues was the only article designed to examine whether statistically

significant differences existed from 2001 to 2004 [29]. They found that black children, aged 3 to 4 years, had significantly more cases of elevated BLLs  $\geq 10$  µg/dL compared to white children (49.8 and 39.6 %, respectively;  $p = 0.02$ ). Bernard and McGeehin analyzed NHANES data from 1988 to 1994 and found racial/ethnic differences in childhood lead level ranges (children 1–5 years) among whites and blacks, and Mexican-Americans [20]. Black children had the highest percentage of elevated lead levels  $\geq 10$  µg/dL compared to whites and Mexican-American children (14.5, 5.5, and 4.3 %, respectively). Dignam and colleagues obtained BLLs from children 1 to 5 years living in two Chicago communities in 2001 [22]. They also found that black children aged 1 to 5 years had the highest percentage of lead levels  $\geq 10$  µg/dL compared to white children and children of other races (29.5, 0.0, and 0.7 %, respectively); 98 % of the sample was black. Finally, the surveillance report by Meyer and colleagues found racial/ethnic differences in elevated lead levels ( $\geq 10$  µg/dL) among children  $< 72$  months (under 6 years of age) from 1997 to 2001 [24]. Of children with an elevated lead level, 60 % were black children, 16 % were Hispanic, and 17 % were white children (See Tables 3 and 4 for BLL ranges).

### Discussion

This systematic review suggests that racial/ethnic differences exist in childhood BLLs. Overall, black children had the highest lead levels when reporting mean BLLs and lead level ranges. Understanding potential racial-ethnic differences that exist in childhood levels is especially pertinent because of the



**Table 3** Racial/Ethnic differences in childhood blood lead levels by range (within each race/ethnicity)

Primary author	Study design	Data source	Location	Sample size	Year	Race/ethnicity	Age range	Blood lead levels <sup>a</sup> (µg/dL), %		Significance	Quality score
								<10	≥10		
Bernard <sup>20</sup>	CS	NHANES	National	4624	1988–1994	White, NH Black, NH	1–5 years	96.0	4.0	Not calculated	Fair
								85.0	15.0		
Dignam <sup>22</sup>	CS	Community	North Carolina, Vermont	539	2001	White Black	1–5 years	95.0	5.0	Not calculated	Poor
								0.6	0.0		
Raymond <sup>29</sup>	RCC	Hospital	Cleveland, Ohio	262	2001–2004	Black Other	3–4 years	68.3	29.5	<i>p</i> =0.02	Fair
								0.9	0.7		
								50.2	49.8		
								60.4	39.6		

Totals may not exactly match 100 % because of rounding. The quality score measures the following: (1) the overall study design and its appropriateness; (2) the collection and analysis of BLLs, and its validity and reliability; (3) the statistical analyses of lead levels; and (4) whether the sample was representative of the general population

CS cross-sectional study, NH not Hispanic, RCC retrospective case-control study

<sup>a</sup> Geometric mean reported for all studies except Dignam et al<sup>22</sup> which reports the arithmetic mean

new public health reference threshold for lead poisoning. In 2012, the CDC concurred with recommendations from an advisory council to report children with a blood lead level of 5 µg/dL, lower than the previous value of 10 µg/dL [30–32]. However, the American Academy of Pediatrics (AAP) and several researchers note that there is no safe level of lead, especially for children under 6 years of age [32, 33]. The number of children exceeding the new blood lead reference level of 5 µg/dL, especially among ethnic minorities, is particularly alarming because of the adverse health impacts of lead exposure.

### Causes of Racial/Ethnic Differences

One suggested cause for racial/ethnic differences in childhood blood levels is that there are different levels of exposure for different racial/ethnic groups [10]. That is, “social inequities” can exist that place ethnic minority children at a higher risk for lead exposure. Risk factors for childhood BLLs include living below the federal poverty line and the location of their residence [4, 10, 11]. Children living in poverty are more likely to live in substandard housing that is in turn at a higher risk for having lead-based paint that is deteriorating. In addition, they are more likely to be exposed to lead-contaminated dirt near home and at school and to live in close proximity to industrial facilities emitting lead contaminants [9, 10]. Differences in childhood BLLs also exist along income levels; on average, low-income children have the highest lead levels [10, 11]. However, even when economic status is taken into consideration, differences across racial/ethnic groups are still found. For instance, low-income black children have higher BLLs compared to low-income white and Hispanic children [10].

The impact of lead exposure in older homes is substantial. Homes built prior to 1978, when lead regulations began to be implemented, are at an increased risk of having lead-based paint. Lead-based paint can peel off and fall to the ground and these “paint chips or flakes” can become ingested by young children who spend most of their time crawling on the ground. This route of lead exposure is the most common for young children and likely is differentially more common among individuals from low-income background and certain racial/ethnic groups. The CDC’s 2011 Health Disparities and Inequalities Report found that racial/ethnic differences exist among children living in housing units that have peeling paint [34]. According to the report, black children (3.5 %) were more likely to live in homes with peeling paint compared to Hispanic (2.4 %) and white (1.9 %) children.

### Lead Prevention

Although childhood BLLs have declined because of national and regional lead screening and abatement activities, elevated lead levels remain persistent, especially among minority

**Table 4** Racial/ethnic differences in childhood blood lead levels by range, multi-year surveillance report, children <72 months (of total number of children tested)

Primary author	Location	No. tested	Year	Race/ethnicity	Blood lead levels <sup>a</sup> (µg/dL), percentage			Quality score
					10–19	20–44	≥45	
Meyer <sup>24</sup>	National	1,703,356	1997	White, NH	0.742 %	0.172 %	0.009 %	Fair
				Black, NH	2.834 %	0.847 %	0.045 %	
				NA/AN	0.015 %	0.004 %	0.000 %	
				A/PI	0.065 %	0.024 %	0.003 %	
				Hispanic	0.637 %	0.194 %	0.013 %	
		1,736,908	1998	Other/multiracial	0.071 %	0.020 %	0.002 %	
				White, NH	0.622 %	0.142 %	0.008 %	
				Black, NH	2.430 %	0.654 %	0.035 %	
				NA/AN	0.013 %	0.003 %	0.000 %	
				A/PI	0.059 %	0.021 %	0.002 %	
		1,809,541	1999	Hispanic	0.528 %	0.159 %	0.012 %	
				Other/multiracial	0.054 %	0.012 %	0.001 %	
				White, NH	0.507 %	0.117 %	0.005 %	
				Black, NH	1.811 %	0.458 %	0.023 %	
				NA/AN	0.011 %	0.003 %	0.000 %	
		2,135,932	2000	A/PI	0.046 %	0.017 %	0.002 %	
				Hispanic	0.425 %	0.128 %	0.009 %	
				Other/multiracial	0.045 %	0.010 %	0.001 %	
				White, NH	0.386 %	0.088 %	0.005 %	
				Black, NH	1.287 %	0.310 %	0.017 %	
2,422,298	2001	NA/AN	0.009 %	0.002 %	0.000 %			
		A/PI	0.036 %	0.013 %	0.001 %			
		Hispanic	0.343 %	0.103 %	0.007 %			
		Other/multiracial	0.032 %	0.008 %	0.001 %			
		White, NH	0.292 %	0.064 %	0.004 %			
				Black, NH	0.941 %	0.213 %	0.014 %	
				NA/AN	0.007 %	0.002 %	0.000 %	
				A/PI	0.025 %	0.011 %	0.001 %	
				Hispanic	0.267 %	0.083 %	0.006 %	
				Other/multiracial	0.028 %	0.007 %	0.001 %	

Significance is not calculated. The quality score measures the following: (1) the overall study design and its appropriateness; (2) the collection and analysis of BLLs, and its validity and reliability; (3) the statistical analyses of lead levels; and (4) whether the sample was representative of the general population

A/PI Asian/Pacific Islander, NA/AN Native American/Alaska Native, NH non-Hispanic

<sup>a</sup> Geometric mean reported

children [10]. Screening programs can mitigate childhood lead exposure; however, because of declines in BLLs and federal budget reductions, the availability of screening programs is at risk [10, 13]. The CDC’s recent revision of the blood reference level threshold should spur agencies to provide at-risk communities, especially ethnic minority communities, with sufficient funding sources to protect children from the harms of lead poisoning.

Federal funding is desperately needed for lead abatement programs. Dramatic changes in federal funding has left many agencies with an unclear plan to move forward with programs

to address the new recommended blood lead reference thresholds [13, 35]. Public health agencies need to increase community and parental knowledge of the harms of lead exposure and preventive measures. Past research suggests that there are racial/ethnic differences in parental knowledge of the harms of lead exposure [10, 36]. It is likely that blood lead reduction efforts will be most effective with the use of focused and targeted screening programs. Such programs should be in line with the recently revised CDC guidelines for childhood blood lead poisoning [30, 31]. In addition, geographic difference may have accounted for racial/ethnic differences in lead

levels. These geographic differences are often compounded by socioeconomic differences, which are frequently along racial/ethnic lines [10, 37]. Targeting communities for lead prevention is a much needed strategy to promote health equity because certain areas may have a higher concentration of older homes with contaminated paint, lead-based pipes, and contaminated soil [37, 38].

### Study Limitations

Despite interesting findings, there are several limitations of this systematic review. First, surveillance data used in the study may not be comparable to other studies included in the review, including NHANES data. Surveillance data are collected by state and local childhood lead poisoning prevention programs as part of their blood lead screening efforts. Many programs target children at risk for lead poisoning and, therefore, their findings are not representative of the general population. In contrast, NHANES uses a sampling methodology that results in findings that are more representative of the general population. There was also the potential for redundant sampling because five of the studies included in the review analyzed data from NHANES. Data collection years and the reporting of racial/ethnic categories overlapped in some studies. In addition, no studies reported the sample size for each racial/ethnic group, and significance levels were not calculated for each racial/ethnic group to determine if differences in childhood BLLs existed. The interaction between race/ethnicity and income level was also not considered in the studies included in this review. Additionally, study designs, data sources, localities, and inconsistencies in the reporting of racial/ethnic categories did not allow us to examine the degree to which racial/ethnic disparities existed in BLLs. Although an inclusion criterion was implemented in studies with participants living in the USA, immigrants or refugees may have been included in the samples because they may be at high risk for lead poisoning. Potential publication bias is also a limitation as studies that may have shown no racial/ethnic differences may have not been published. Despite the limitations, the findings of the review provide important formative data with respect to understanding the extent of racial/ethnic differences in childhood lead levels.

### Conclusion

The findings of this review suggest that racial/ethnic disparities exist in BLLs among children. Future investigations of racial/ethnic differences should obtain ample sample sizes for several racial/ethnic groups to determine differences in childhood BLLs and ascertain the number of children who have lead levels in the potentially harmful range ( $\geq 2$   $\mu\text{g}/\text{dL}$ ), especially since this review found few articles documenting

differences across racial/ethnic groups. These studies should also be combined with environmental assessments to determine the sources of lead. Most importantly, there should be a focus on primary prevention, that is preventing lead exposure, rather than secondary prevention, i.e., preventing further harm after a child is exposed. There are several primary prevention strategies the CDC recommends to prevent childhood lead poisoning, specifically from lead-based paint [39]. They include disseminating lead safety information to parents, engaging policy makers and property managers/owners in high-risk housing units, and expanding housing rehabilitation programs. With concerted efforts across sectors, children at risk for lead poisoning can be targeted and exposure can be prevented.

**Acknowledgments** The first author would like to thank Eric S. Hall, MA, MCE, and Herbert White, Jr., MD, MPH, MS, for providing feedback on the manuscript.

**Ethics Approval and Consent to participate** No animal or human studies were carried out by the authors for this article.

**Conflict of interest** Brandi M. White, Heather S. Bonilha, and Charles Ellis declare that they have no conflict of interest.

**Funding** The authors received no financial support for the research, authorship, and/or publication of this article.

### References

1. Trasande L, Liu L. Reducing the staggering costs of environmental disease in children, estimated at \$76.6 billion in 2008. *Health Aff (Millwood)*. 2011;30(5):863–70.
2. Porter RS, Kaplan JL, editors. Merck manual of diagnosis and therapy. 19th ed. Whitehouse Station: Merck Sharp & Dohme; 2011.
3. Yuan W, Holland SK, Cecil KM, Dietrich KN, Wessel SD, Altaye M, et al. The impact of early childhood lead exposure on brain organization: a functional magnetic resonance imaging study of language function. *Pediatrics*. 2006;118(3):971–7.
4. Landrigan PJ, Carlson JE, Bearer CF, Cranmer JS, Bullard RD, Etzel RA, et al. Children's health and the environment: a new agenda for prevention research. *Environ Health Perspect*. 1998;106 Suppl 3:787–94.
5. Karr C. Addressing environmental contaminants in pediatric practice. *Pediatr Rev*. 2011;32(5):190–200.
6. Barbosa F, Tanus-Santos JE, Gerlach RF, Parsons PJ. A critical review of biomarkers used for monitoring human exposure to lead: advantages, limitations, and future needs. *Environ Health Perspect*. 2005;113(12):1669–74.
7. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations  $<10$   $\mu\text{g}/\text{dL}$  in US children and adolescents. *Public Health Rep*. 2000;115(6):521–9.
8. Koller K, Brown T, Spurgeon A, Levy L. Recent developments in low-level lead exposure and intellectual impairment in children. *Environ Health Perspect*. 2004;112(9):987–94.
9. Levin R, Brown MJ, Kashtock ME, Jacobs DE, Whelan EA, Rodman J, et al. Lead exposures in US children, 2008: implications for prevention. *Environ Health Perspect*. 2008;116(10):1285–93.



10. Dilworth-Bart JE, Moore CF. Mercy mercy me: social injustice and the prevention of environmental pollutant exposures among ethnic minority and poor children. *Child Dev.* 2006;77(2):247–65.
11. Tong S, von Schirnding YE, Prapamontol T. Environmental lead exposure: a public health problem of global dimensions. *Bull World Health Organ.* 2000;78(9):1068–77.
12. Sargent JD, Dalton M, Demidenko E, Simon P, Klein RZ. The association between state housing policy and lead poisoning in children. *Am J Public Health.* 1999;89(11):1690–5.
13. Burns MS, Gerstenberger SL. Implications of the new centers for disease and control and prevention blood lead reference value. *Am J Public Health.* 2014;104(6):e27–33.
14. Brown RW, Longoria T. Multiple risk factors for lead poisoning in Hispanic sub-populations: a review. *J Immigr Minor Health.* 2010;12(5):715–25.
15. Wright RJ. Moving towards making social toxins mainstream in children's environmental health. *Curr Opin Pediatr.* 2009;21(2):222–9.
16. Healthy People 2020: Environmental Health [internet]. U.S. Department of Health and Human Services [updated 2013 Aug 28; cited 2013 Sept 10]. Available from: <http://healthypeople.gov/2020/topicsobjectives2020/objectiveslist.aspx?topicId=12>.
17. Murad MH, Montori VM. Synthesizing evidence: shifting the focus from individual studies to the body of evidence. *JAMA.* 2013;309(21):2217–8.
18. Blumenshine P, Egerter S, Barclay CJ, Cubbin C, Braveman PA. Socioeconomic disparities in adverse birth outcomes: a systematic review. *Am J Prev Med.* 2010;39(3):263–72.
19. Effective Public Health Practice Project. Quality assessment tool for quantitative studies. Ontario, Canada: McMaster University; 2010. Available from: [www.ephpp.ca/PDF/QATool.pdf](http://www.ephpp.ca/PDF/QATool.pdf).
20. Bernard SM, McGeehin MA. Prevalence of blood lead levels  $\geq$  5 micro g/dL among US children 1 to 5 years of age and socioeconomic and demographic factors associated with blood of lead levels 5 to 10 micro g/dL, third national health and nutrition examination survey, 1988–1994. *Pediatrics.* 2003;112(6):1308–13.
21. Centers for Disease Control and Prevention (CDC). Blood lead levels—United States, 1999–2002. *MMWR Morb Mortal Wkly Rep.* 2005;54(20):513–6.
22. Dignam TA, Lojo J, Meyer PA, Norman E, Sayre A, Flanders WD. Reduction of elevated blood lead levels in children in North Carolina and Vermont, 1996–1999. *Environ Health Perspect.* 2008;116(7):981–5.
23. Jones RL, Homa DM, Meyer PA, Brody DJ, Caldwell KL, Pirkle JL, et al. Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years, 1988–2004. *Pediatrics.* 2009;123(3):e376–85.
24. Meyer PA, Pivetz T, Dignam TA, Homa DM, Schoonover J, Brody D. Centers for disease control and prevention. surveillance for elevated blood lead levels among children—United States, 1997–2001. *MMWR Surveill Summ.* 2003;52(10):1–21.
25. Scott LL, Nguyen LM. Geographic region of residence and blood lead levels in US children: results of the National Health and Nutrition Examination Survey. *Int Arch Occup Environ Health.* 2011;84(5):513–22.
26. Stroop DM, Dietrich KN, Hunt AN, Suddendorf LR, Giangiacomo M. Lead-based paint health risk assessment in dependent children living in military housing. *Public Health Rep.* 2002;117(5):446–52.
27. Joseph CL, Havstad S, Ownby DR, Peterson EL, Maliarik M, McCabe Jr MJ, et al. Blood lead level and risk of asthma. *Environ Health Perspect.* 2005;113(7):900–4.
28. Leighton J, Klitzman S, Sedlar S, Matte T, Cohen NL. The effect of lead-based paint hazard remediation on blood lead levels of lead poisoned children in New York City. *Environ Res.* 2003;92(3):182–90.
29. Raymond JS, Anderson R, Feingold M, Homa D, Brown MJ. Risk for elevated blood lead levels in 3- and 4-year-old children. *Matern Child Health J.* 2009;13(1):40–7.
30. CDC. Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention. Atlanta, GA: Advisory Committee on Childhood Lead Poisoning Prevention, U.S. Centers for Disease Control and Prevention (4 Jan 2012). Available: [www.cdc.gov/nceh/lead/acclpp/final\\_document\\_030712.pdf](http://www.cdc.gov/nceh/lead/acclpp/final_document_030712.pdf) [accessed 15 May 2013].
31. CDC. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in “Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention.” Atlanta, GA: U.S. Centers for Disease Control and Prevention. Available: [http://www.cdc.gov/nceh/lead/acclpp/cdc\\_response\\_lead\\_exposure\\_recs.pdf](http://www.cdc.gov/nceh/lead/acclpp/cdc_response_lead_exposure_recs.pdf) [accessed 15 May 2013].
32. Gilbert SG, Weiss B. A rationale for lowering the blood lead action level from 10 to 2  $\mu$ g/dL. *Neurotoxicology.* 2006;27(5):693–701.
33. American Academy of Pediatrics (AAP). AAP Commends CDC for Recognizing That for Children, There is No Safe Level of Lead Exposure. 2012, May 5. Available from: <http://www.aap.org/en-us/about-the-aap/aap-press-room/pages/AAP-Statement-CDC-Revised-Lead-Exposure-Guidelines.aspx>.
34. Raymond J, Wheeler W, Brown MJ; Centers for Disease Control and Prevention (CDC). Inadequate and unhealthy housing, 2007 and 2009. *MMWR Surveill Summ.* 2011;60 Suppl:21–7.
35. Burns MS, Shah LH, Marque ER, Denton SL, Neyland BA, Vereschagin D, Gremse DA.
36. Mehta S, Binns HJ. What do parents know about lead poisoning? the Chicago lead knowledge test. *JAMA Ped.* 1998;152(12):1213–8.
37. Oyana TJ, Margai FM. Geographic analysis of health risks of pediatric lead exposure: a golden opportunity to promote healthy neighborhoods. *Arch Environ Occup Health.* 2007;62(2):93–104.
38. Schlenker TL, Baxmann R, McAvoy P, Bartkowski J, Murphy A. Primary prevention of childhood lead poisoning through community outreach. *WMJ.* 2001;100(8):48–54.
39. Building Blocks for Primary Prevention: Protecting Children from Lead-Based Paint Hazards. Atlanta: CDC, 2005