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Nonlinear associations between blood lead in children, age of child, and quantity of soil lead in metropolitan New Orleans

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ABSTRACT

Previous studies identified a curvilinear association between aggregated blood lead (BL) and soil lead (SL) data in New Orleans census tracts. In this study we investigate the relationships between SL (mg/kg), age of child, and BL (μg/dL) of 55,551 children in 280 census tracts in metropolitan New Orleans, 2000 to 2005. Analyses include random effects regression models predicting BL levels of children (μg/dL) and random effects logistic regression models predicting the odds of BL in children exceeding 15, 10, 7, 5, and 3 μg/dL as a function of age and SL exposure. Economic benefits of SL reduction scenarios are estimated. A unit raise in median $SL^{0.5}$ significantly increases the BL level in children ($b = 0.214$ $p = <0.01$), and a unit change in $Age^{0.5}$ significantly increases child BL ($b = 0.401$, $p = <0.01$). A unit change in $Age^{0.5}$ increases the odds of a child BL exceeding 10 μg/dL by a multiplicative factor of 1.23 (95% CI 1.21 to 1.25), and a unit (mg/kg) addition of SL increases the odds of child BL > 10 μg/dL by a factor of 1.13 (95% CI 1.12 to 1.14). Extrapolating from regression results, we find that a shift in SL regulatory standard from 400 to 100 mg/kg provides each child with an economic benefit ranging from $4710 to $12,624 ($US 2000). Children's BL is a curvilinear function of both age and level of exposure to neighborhood SL. Therefore, a change in SL regulatory standard from 400 to 100 mg/kg provides children with substantial economic benefit.

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1. Introduction

Dose–response relationships between lead-contaminated soil (SL) and child exposure are expressed in the scientific literature as expected change in blood Pb (BL) as a function of unit change in SL, with observed SL values typically scaled to standard units of SL of 1000 or 100 mg/kg (Reagan and Silbergeld, 1989; Madhavan et al., 1989; Xintaras, 1992). Mielke et al. (1997, 1999, 2007) previously reported strong non-linear associations between SL and children's BL response and a high variance explaining mathematical model of the relationship as: $BL = 2.038 + 0.172 \times (SL)^{0.5}$ (Mielke et al., 2007). The non-linear association operates at the neighborhood scale—the observed relationship is between census tract median SL and median census tract BL of sampled children in metropolitan New Orleans (Mielke et al., 2007). The principal objective of this study is to downscale the analysis to the individual child level.

In our study, we conduct a series of random-effects regression models investigating non-linear relationships between children's age, level of exposure to SL, and BL response of children in metropolitan New Orleans. Also, we extend the existing literature by rendering a series of random-effects logistic regression models showing how unit changes in both children's age and level of exposure to SL significantly push children over various thresholds of μg/dL. With regression equations, and following Grosse et al. (2002), we calculate the expected economic benefits of shifting the current US SL regulatory standard from 400 mg/kg to 100 mg/kg.

Our investigation is organized into five sections. First, we briefly describe the research setting noting important industrial characteristics that constitute New Orleans as a highly lead-contaminated urban area. Second, we describe data collection efforts, with particular attention paid to soil sampling and analytic procedures. Third, we describe statistical logic. Fourth, we present GIS, descriptive, and regression results. Finally, we reconfigure statistical results to comment on the expected economic benefits of soil lead remediation.

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1.1. Pre-Katrina New Orleans

New Orleans is positioned on the Delta of the Mississippi River. The sediments from the Mississippi River are the parent materials of the alluvial soils of metropolitan New Orleans which contain only trace amounts of Pb (Mielke et al., 2000). Despite the high quality of parent soils, the city of New Orleans, as of 2000, had ten census tracts with a median SL of ≥1000 mg/kg (Mielke et al., 2006). Inner city children, regardless of whether or not they live in public or private housing, have alarmingly high rates (up to 29%) of BL≥10 μg/dL (Rabito et al., 2003). Urban soils in New Orleans integrate all dust sources of Pb, including lead-based paint (either deteriorated or haphazardly removed by power sanding, sand blasting, etc.), lead additives by gasoline emissions, and incinerator or industrial Pb emissions that have accumulated in the environment (Mielke, 1999, 2005; Mielke et al., 2010). Soils then, are both a sink and a source of Pb dust in New Orleans, rendering the city of New Orleans as a highly Pb contaminated urban agglomeration.

2. Materials and methods

2.1. Soil lead (SL) data

The SL dataset was assembled from samples collected on the top 2.5 cm of the soil surface within residential census tracts of metropolitan New Orleans (Mielke et al., 2005). Where possible, 19 samples per census tract were collected as described previously (Mielke et al., 2005). The soil samples were stratified by 1990 Census Tracts ($n=286$). Census tracts are a sensible geo-statistical unit for approximating neighborhood soil lead conditions, given their relative uniformity in population size and demographic data richness. Sampling was conducted by using U.S. Census Bureau maps as a guide (U.S. Census Tracts and Block Number Areas, 1993).

Overall, the soil survey resulted in 5467 surface samples collected from 286 census tracts (Mielke et al., 2005). The extraction procedure involved room temperature leachate methods using 1 M nitric acid (HNO_3), a scheme that correlates well with total methods (Mielke et al., 1983; U.S. EPA, 1996). The method has the advantage of more closely resembling physiologic conditions compared with extraction methods using boiling and concentrated HNO_3.

The extraction protocol requires mixing 0.4 g of dry and sieved (#10 USGS—2 mm) soils with 1 M HNO_3 and agitated at slow speed on an Eberbach® shaker for 2 h at room temperature (~22 °C). The extract is then centrifuged (10 min at 1600×g) and filtered using Fisherbrand® P4 paper. The extract is stored in 20 mL polypropylene scintillation vials until analyzed. A Spectro Analytical Instruments CIROS® CCD Inductively Coupled Plasma Atomic Emission Spectrometer (ICP-AES) is used to analyze the metals in each sample. The ICP-AES is calibrated with NIST traceable standards, and a laboratory reference, at a rate of 1 per 15 samples, is analyzed during each run. Internal laboratory references included one low SL sample from New Orleans City Park and one high SL sample from the junction of Elysian Fields and Interstate 10 within the inner city of New Orleans. Duplicate extractions are included for every 15 samples. The final SL database is the result of all samples collected in each census tract of metropolitan New Orleans (Mielke et al., 2005). The neighborhood soil lead data are summarized as the median value of samples per census tract expressed in mg/kg units.

2.2. Blood lead (BL) and age data

By law the Louisiana childhood lead poisoning prevention program (LACLPPP) specifies: "1. Administration of a risk assessment questionnaire at every well baby visit; 2. Use a blood lead test to screen all children at ages 12 months and at 24 months or at any time from ages 36 months to 72 months, if they have not been previously screened; 3. Blood lead levels >15 pgldL obtained from finger stick samples will be confirmed using a venous blood sample (Louisiana LPPP rules, 2008)." Mandatory case reporting (defined as BL >15 μg/dL) is required by health care providers (Louisiana LPPP rules, 2008). Details of the childhood blood lead surveillance system (CBLSS) are given in a report which notes that for the combined dataset of both public health lab data and private lab data about 3% of the blood samples were venous specimens, about 66% were capillary specimens, and specimen type unknown for the remaining 31% of the blood samples (Louisiana CBLSS, 2004). The CBLSS datasets were obtained through a formal application and request to the LACLPPP.

Each BL was geo-coded and matched to the corresponding 1990 census tract boundary. BL values for 55,551 children are analyzed. BL values are expressed in μg/dL units. In addition to BL levels, CBLSS data contain information on the age of the child when the blood sample was taken. *Age* is measured in months. Residential tenure data from the 2000 Population and Housing Census (item PCT49) show that 85.42% of the population in Orleans Parish had the exact same address or resided in the same parish from 1995 to 2000, supporting a logic that age may be conceived as adequate (though imperfect) surrogate for length of exposure. Descriptive statistics on SL, BL, and age in months are presented in Table 1.

2.3. Modeling procedures

We execute *random effects models* of blood lead levels, allowing each neighborhood or census tract to have its own intercept and yielding a weighted average of *between* and *within* neighborhood effects. The model for the blood lead y_{ij} of child i in neighborhood j is specified as:

$$y_{ij} = \beta_1 + \beta_2 x_{2ij} + \ldots + \beta_p x_{pij} + \varepsilon_{ij} \quad (1)$$

where, x_{2ij} through x_{pij} are parameters and ε_{ij} is a residual.

The random effects model divides the residual term into two components—a neighborhood-specific error component (ζ_j), which is constant across blood lead levels, and a child-specific error component (ϵ_{ij}) which varies between children and neighborhood:

$$\varepsilon_{ij} = \zeta_j + \epsilon_{ij}. \quad (2)$$

Table 1
Percentile values of neighborhood soil lead, child age, and blood lead levels.

Percentile	Soil lead (SL)	Age (months)	Blood lead (BL)
$P_{0.01}$	12.60	7	0.40
$P_{0.05}$	18.30	10	1.00
$P_{0.10}$	25.80	12	2.00
$P_{0.20}$	37.00	13	3.00
$P_{0.30}$	46.40	16	3.00
$P_{0.40}$	69.60	24	3.20
$P_{0.50}$	107.20	26	4.00
$P_{0.60}$	200.50	33	5.00
$P_{0.70}$	285.20	40	6.00
$P_{0.80}$	469.50	48	7.90
$P_{0.90}$	608.30	54	11.00
$P_{0.95}$	882.00	60	15.10
$P_{0.99}$	1164.00	69	26.45
Mean	249.42	30.22	5.68
Std. dev.	290.90	16.68	5.16
Min	6.2	6	0
Max	1789	72	117
N	55,551	55,551	55,551

Substituting for ε_{ij} into regression model (1), we obtain the random intercept model with parameters:

$$y_{ij} = \beta_1 + \beta_2 x_{2ij} + \ldots + \beta_p x_{pij} + \zeta_j + \epsilon_{ij} = (\beta_1 + \zeta_j) + \beta_2 x_{2ij} + \ldots + \beta_p x_{pij} + \epsilon_{ij}. \quad (3)$$

The neighborhood-specific error component ζ_j can be thought of as the combined effects of omitted neighborhood characteristics or unobserved heterogeneity (Rabe-Hesketh and Skrondal, 2008). Letting $x_{ij} = (x_{2ij},\ldots, x_{pij})'$ be a vector of observed parameters, exogeneity assumptions of the random intercept model are $E(\zeta_j|x_{ij}) = 0$, and $E(\epsilon_{ij}|x_{ij}, \zeta_j) = 0$, from which it follows that $E(\epsilon_{ij}|x_{ij}) = 0$. Both ζ_j and ϵ_{ij} are assumed uncorrelated with observed parameters. Analysis of residual terms shows no endogeneity for parameters analyzed. Residual variances of full models closely approximate Gaussian. This procedure is useful in the absence of good data estimating exterior paint lead and interior dust lead that influence BL outcomes in children.

To estimate the likelihood of a child's blood lead exceeding 15, 10, 7, 5, and 3 μg/dL, adjusting for the combined effect of omitted neighborhood-specific covariates that may cause children to be more or less prone to threshold exceedance, we include a census tract-specific random intercept (ζ_j) in our binary prediction equation

$$\text{logit}\left\{Pr(y_{ij} = 1|x_{ij}, \zeta_j)\right\} = \beta_1 + \beta_2 x_{2ij} + \ldots + \beta_p x_{pij} + \zeta_j \quad (4)$$

with $\zeta_j|x_{ij} \sim N(0, \psi)$ and t independent across census tracts j, given a random-intercept logistic regression. We complete the model specification by assuming that, given $\pi_{ij} \equiv \Pr(y_{ij}|x_{ij}, \zeta_j)$, y_{ij} are independently distributed as

$$y_{ij}|\pi_{ij} \sim binomial\left(1, \pi_{ij}\right). \quad (5)$$

In the next section we detail GIS, descriptive, and regression results examining blood lead levels in children as a function of length and level of exposure to soil lead.

3. Results

We begin with geographic analysis. Fig. 1 shows the joint distribution of median BL and median SL at the census tract scale. This aggregate-level display of data corroborates the basic intuition and logic of our analysis—SL conditions are significantly correlated with BL outcomes in children. The distribution of median neighborhood BL levels in children is divided into 4 intervals, with smaller blue-shaded dots indicating relatively lower median BL levels, and larger red-shaded dots indicating relatively higher median BL levels. Each dot is encircled. The size of each circle corresponds to an interval value reflecting the median level of SL observed for each neighborhood, with larger circles reflecting relatively higher median SL values. The geographic correspondence between median SL and median BL is apparent.

Thus, regarding SL collection and extraction, the protocol is sensitive to the differences between various communities and the specific arrangement of houses and streets, and the results are consistent with the pattern of children's response to quantities of Pb dust. The perimeter of the building has larger amounts of Pb, but this is empirically true regardless of whether the building is painted wood siding or brick and stucco siding (Mielke et al., 2010). In perspective of the city, an important issue is that in outlying communities yards are larger, and children are more likely playing away from buildings within these residential communities; within inner city communities, lots are smaller with houses packed together and close to the street, and children are more likely playing within or near the drip-line because the drip-lines comingle with the street side in these residential communities.

Table 2 reports mean BL levels of children at various points in the joint distribution of child age and level of exposure to SL. Table 2 refines and extends observations made regarding Map 1 by inclusion of child age data and more percentile divisions of the distributions of BL and SL. At the 10th percentile of both length (12 months) and level (26 mg/kg) of SL exposure (near the left-hand corner of the table), we observe an average BL of 3.135 μg/dL (SD = 2.189, $N = 460$). Moving down the principal diagonal from $P_{0.01}|P_{0.01}$ to $P_{0.99}|P_{0.99}$ we observe a steady increase in the mean BL of children. A closer inspection of cell values in Table 2 shows that BL levels in children are a nonlinear function of both age and level of exposure to SL. This age-based result is consistent with established literature finding that BL levels in children (with stable residence) rise through age 24 months and flatten thereafter (Baghurst et al., 1992). Normally children's BL is expected to peak at around 18 to 24 months and then decrease. In New Orleans, BL continues to increase with age beyond 24 months; this finding was also observed by Rabito (1998). The non-linear age effect is partially determined by environmental exposure and the mild climate of New Orleans, in combination with developmental behavioral (i.e., dirt eating) and bio-kinetic factors (i.e., calcium metabolism).

Fig. 2 illustrates the nonlinear effects of child age and level of SL exposure on BL. Level of SL exposure is on the x-axis, age of child is on the y-axis, and mean BL (μg/dL) is plotted in the space. The size of each sphere corresponds with the observed mean level of BL. Note the behavior of mean BL at the 75th and 99th percentiles of child age, while moving across the horizontal axis from the 1st to the 99th percentile on level of SL—nonlinearity consistent with theory and established literature is apparent. A similar (but not as striking) pattern in mean BL is observable if we fix attention on the 75th and 99th percentiles of level of SL exposure, and move vertically along the axis of child age. Both Table 2 and Fig. 1 confirm the fact that both age and level of exposure to SL drive observed BL in children.

Table 3 reports random effects generalized least squares regression coefficients predicting BL in children. Age and SL variables are square root transformed. Transformation of predictor variables is consistent with previous literature (Mielke et al., 2007) and best approximates the behavior of data presented in Table 2 and Fig. 2. We render a series of models to observe robustness of coefficients. Across all models specified, both the square root of $\text{Age}^{0.5}$ and the square root of exposure level ($\text{Soil lead}^{0.5}$) are positively and significantly associated with BL, adjusting for year (date) of BL observation. For example, in Model 1 of Table 3, we find that a unit change in $\text{Age}^{0.5}$ increases BL levels in children by 0.401 ($p = <0.001$), and a unit increase in Soil $\text{lead}^{0.5}$ increases child BL by 0.214 ($p = <0.001$). Between-census tract variance explained by our full model (in column 1) is strong ($R^2 = 0.746$).

To make regression results more meaningful, we graphically display point estimates from Table 3. Fig. 3 shows the predicted level of BL for unit changes in observed levels of SL, while fixing child age at the sample mean of 30.22 months. In general, statistically controlling for the known effects of age, the fractional polynomials intersecting the space behave as the aggregate BL and SL curves reported in Mielke et al. (2007).

Fig. 4 shows the predicted level of BL in children for unit changes in age. SL exposure level is set at three different regulatory standards, including the United States' 400 mg/kg, Norway's general standard of 100 mg/kg, and Norway's playground standard of 60 mg/kg (Ottesen et al., 2008). The height of the exposure length–BL curve at US SL regulatory setting is notable.

Next, we estimate the likelihood of a child's BL exceeding 15, 10, 7, 5, and 3 μg/dL as a function of length and level of SL exposure, adjusting for the combined effect of omitted neighborhood-specific covariates that may cause children to be more or less prone to threshold exceedance. Table 4 reports random effects logistic regression coefficients predicting threshold exceedance of BL levels in children (μg/dL). For ease of interpretation, Table 5 reports odds

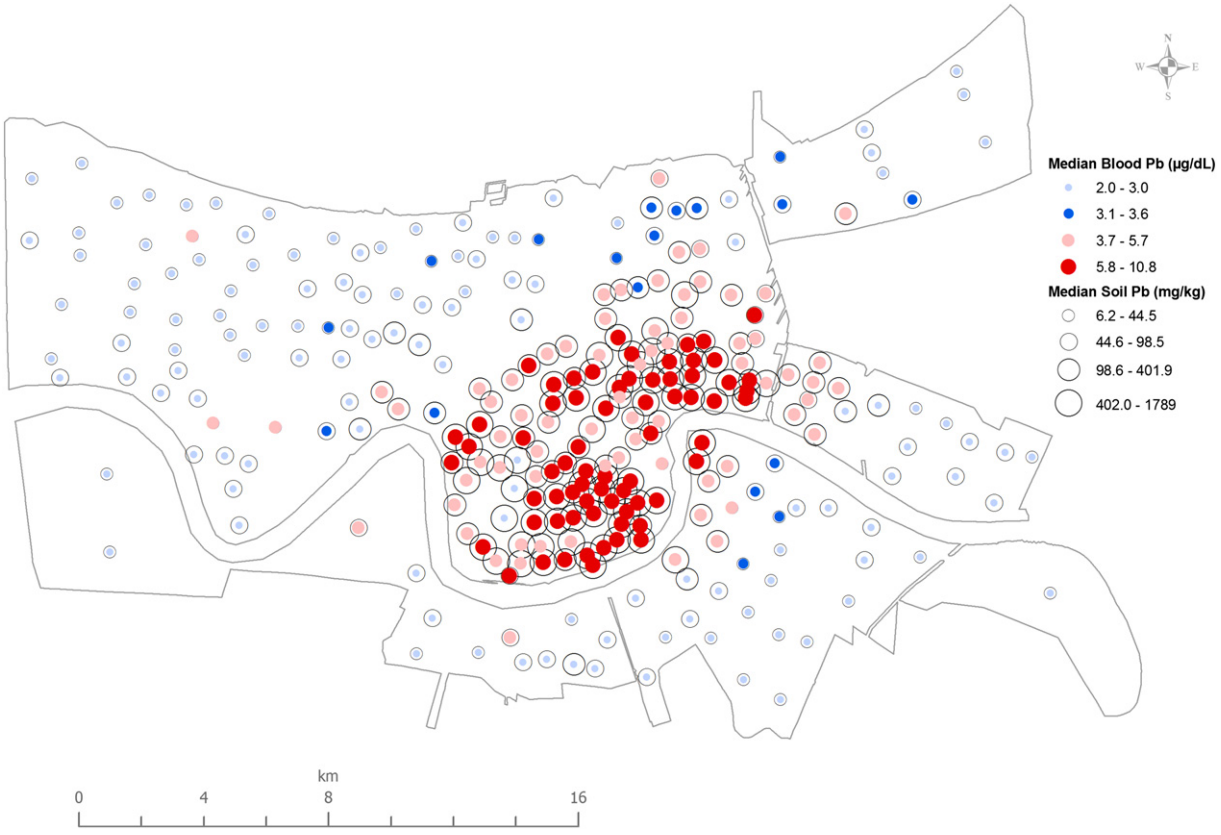

Fig. 1. The spatial distribution of median blood Pb and median soil Pb for census tracts in metropolitan New Orleans.

ratios derived by transformation of coefficients reported in Table 4. In Table 5, column 1, we observe, all things held equal, that the odds of a child's BL exceeding 15 μg/dL increase by a multiplicative factor of 1.199 (95% CI, 1.169 to 1.230) for every unit added to our age parameter. Similarly, with a unit increase in Pb to neighborhood soil, we observe an increase in the odds of a child recording a BL level of 15 μg/dL higher by a factor of 1.138 (95% CI, 1.125 to 1.151). The patterning of odds ratios for both age and level of SL exposure across

Table 2
Average observed blood lead levels at percentile values of level of soil lead exposure and age of child.

	Level $P_{0.01}$	Level $P_{0.05}$	Level $P_{0.10}$	Level $P_{0.25}$	Level $P_{0.50}$	Level $P_{0.75}$	Level $P_{0.90}$	Level $P_{0.95}$	Level $P_{0.99}$
Age $P_{0.01}$	2.033	2.233	2.364	2.559	2.572	2.931	3.036	2.998	3.996
	(1.674)	(1.695)	(0.962)	(3.851)	(1.667)	(2.376)	(2.680)	(2.648)	(3.884)
	3	24	31	92	156	309	216	87	103
Age $P_{0.05}$	2.60	2.774	2.508	2.995	3.391	3.878	4.406	4.649	5.129
	(1.595)	(1.407)	(1.286)	(1.774)	(2.024)	(3.065)	(3.974)	(3.604)	(3.890)
	15	58	51	221	453	647	414	150	136
Age $P_{0.10}$	3.078	3.111	3.135	3.240	3.332	4.70	5.402	5.987	6.865
	(3.061)	(2.047)	(2.189)	(1.904)	(2.20)	(3.842)	(4.222)	(5.837)	(5.381)
	128	404	460	1328	2165	1281	742	190	217
Age $P_{0.25}$	2.751	3.064	3.575	3.512	3.447	5.026	5.704	6.475	5.935
	(1.545)	(1.90)	(5.429)	(2.764)	(2.301)	(4.309)	(4.474)	(5.307)	(3.721)
	68	192	196	643	1044	876	480	166	148
Age $P_{0.50}$	3.625	3.731	3.991	4.258	4.422	6.883	8.887	10.01	10.34
	(2.256)	(2.519)	(2.859)	(3.105)	(3.582)	(5.293)	(7.212)	(8.665)	(7.216)
	186	615	769	2362	3654	3273	1917	636	659
Age $P_{0.75}$	3.534	3.802	3.940	4.370	4.375	7.486	8.980	9.563	11.151
	(2.441)	(2.845)	(3.018)	(3.047)	(2.938)	(5.923)	(6.835)	(6.739)	(7.90)
	139	508	593	1971	3118	4040	2374	805	779
Age $P_{0.90}$	3.429	3.716	3.829	4.078	4.108	6.752	8.930	8.625	10.20
	(2.498)	(2.805)	(1.947)	(2.793)	(2.757)	(4.918)	(7.001)	(7.442)	(7.924)
	69	298	305	1088	1760	2231	1263	431	412
Age $P_{0.95}$	3.178	4.062	3.521	4.162	4.167	6.903	8.473	9.111	10.20
	(1.54)	(3.566)	(2.364)	(2.675)	(3.484)	(5.01)	(6.393)	(6.687)	(7.14)
	23	98	96	351	645	800	453	152	183
Age $P_{0.99}$	3.408	4.705	3.575	4.074	3.996	6.598	7.587	9.431	10.25
	(2.085)	(10.388)	(2.087)	(2.827)	(2.885)	(5.459)	(5.538)	(6.395)	(6.690)
	37	104	167	376	716	597	373	120	111

Note: Standard deviation reported in parenthesis with cell count underneath.

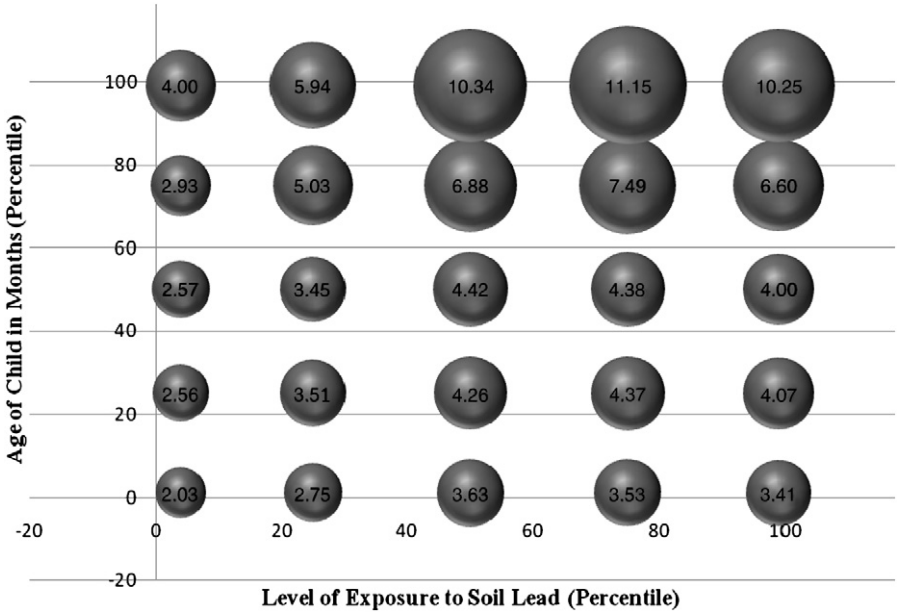

Fig. 2. Mean blood lead by level and length of exposure to soil lead percentiles.

threshold models executed reveals that Age$^{0.5}$ is incrementally more important at lower thresholds of μg/dL, while Soil lead$^{0.5}$ is incrementally more important at higher thresholds of μg/dL. This cross-patterning of odds ratios for child age and level of exposure is logically expectable.

4. Discussion and conclusion

Gould's (2009) recent work applies the same type of analysis, but more broadly for all US children under 7 years of age versus our focus on the New Orleans cohort. The present paper's analyses are more specific, explicitly modeling the link between SL and BL while emphasizing nonlinearities, with the narrower regional focus allowing for more precise estimates. Nevertheless, our estimates are broadly in line with Gould, with both papers underscoring the considerable returns of reducing environmental lead hazards.

Several studies have evaluated the BL response of children to multiple sources of Pb. In a pooled analysis of 12 epidemiologic studies Lanphear et al. (1998) report that lead-contaminated house dust is the primary source of variation in children's BL. Lanphear et al. also observe that exterior lead (measured by a soil sample around the foundation or perimeter of the child's residence or in the play area of the child or by an exterior dust sample) significantly increases BL levels in children and significantly increases the probability of a child's BL level exceeding 10 μg/dL. Similarly, Succop et al. (1998) reanalyzed data from 1855 children (72 months or age or less) from 11 studies stretching over 15 years. Using a structural equation modeling technique, Succop et al. find that proximate variables of BL exposure involving interior floor dust lead and hand-wipe dust Pb were the most important predictors of child BL. However, of particular relevance to our manuscript and as reported in their composite structural equation pathway model of BL exposure, SL appears to be the primary underlying source of variation in both interior floor dust lead and hand-wipe dust Pb, doubling the explanatory power of interior paint Pb, for example.

Before we consider the policy implications of results, we briefly recapitulate research motivation and basic findings. We sought to extend the research reporting a positive non-linear relationship between median neighborhood SL and median neighborhood BL in children. We extend this line of research by moving the analysis to the individual level, and by incorporating age of child and year of observation controls. We conducted a series of random-effects regression models showing significant non-linear relationships between child age, level of exposure to SL, and BL in children. Also, we extend existing literature by rendering a series of random-effects logistic regression models showing how unit changes in both length of time and level of exposure significantly push children over various policy relevant and physiologically meaningful thresholds of μg/dL. Across all specifications and modeling procedures we find that child age and quantity of exposure to neighborhood SL significantly increase observed BL levels in children.

These results naturally present important questions regarding explicit benefits of reducing children's exposure to environmental lead sources. Quantitative findings provide a fulcrum for estimating such benefits by highlighting a key transmission mechanism from soil concentrations to individuals' blood lead levels. These BL benchmarks can then be translated into effects on a child's neurodevelopment through previous research, which in turn have implications for that person's eventual lifetime earnings potential discounted to a single

Table 3
Random effects generalized least squares regression coefficients predicting blood lead levels in children (μg/dL).

	Model 1 bPb	Model 2 bPb	Model 3 bPb	Model 4 bPb	Model 5 bPb
Age$^{0.5}$	0.401***		0.396***		0.403***
	(0.013)		(0.013)		(0.013)
Soil lead$^{0.5}$	0.214***	0.220***	0.217***	0.222***	
	(0.007)	(0.007)	(0.007)	(0.007)	
Year	−0.110***	−0.090***			−0.115***
	(0.013)	(0.014)			(0.014)
Constant	0.821***	2.795***	0.567***	2.566***	3.695***
	(0.135)	(0.123)	(0.133)	(0.120)	(0.126)
Wald X^2	1971.33	964.22**	1886.76*	887.47	997.68
R^2 between	0.746	0.7353	0.7429	0.7322	0.3323
N	55,551	55,551	55,551	55,551	55,551
Census tracts	280	280	280	280	280

Standard errors in parentheses.
*** $p<0.01$.
** $p<0.05$.
* $p<0.1$

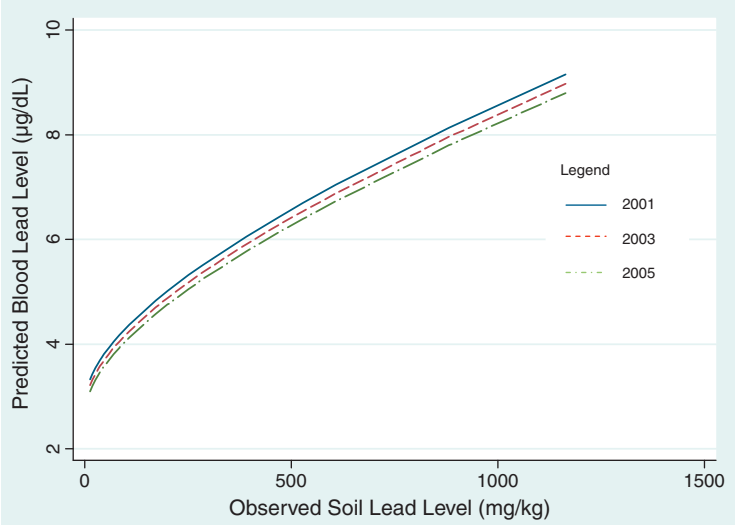

Fig. 3. Predicted (estimates retrieved from the full random effects generalized least squares model with age fixed at the sample mean of 30.22 months) blood lead in children (µg/dL) as a function of observed soil lead (mg/kg) by year.

present value figure. We leverage Grosse et al. (2002), who themselves build upon Schwartz's (1994) seminal benefit–cost template.

With coefficients from our fully saturated random-effects model in Table 3, we consider the economic benefits of three SL reduction scenarios. Table 6 summarizes the steps of our extrapolation. Grosse et al. (2002) provide a sensible extrapolation procedure based on a 5-step causal model moving from lead exposure to expected earnings (i.e., net present value of earnings). Our analysis allows for more explicit consideration of the first step in Grosse et al.'s (2002: p. 564) causal model—*lead in the environment*. By first back-stepping the extrapolation and then moving forward with Grosse et al. (2002), we calculate the expected economic returns of shifting neighborhood SL conditions.

The first and second scenarios are statistical exercises, showing the expected return in net present value of earnings if neighborhood SL levels were reduced from the maximum to the minimum and from the 75th percentile to the 25th percentile in median SL value observed in our data. The third scenario imagines the economic benefits of the United States becoming Norway. More specifically, we calculate the net present value in earnings gained by children in New Orleans if the SL regulatory standard of the United States (400 mg/kg) were changed to the SL regulatory standard of Norway (100 mg/kg).

We begin first with the polar benchmarks of SL fixed at their respective observed minimum (6.2 mg/kg) and maximum (1789 mg/kg) values. The net resulting difference in $\Delta BL_1 = 8.53$ µg/dL. This value of 8.53 µg/dL is derived by, all things held equal, taking the difference in the expected value of average child BL under maximum and minimum SL conditions: $E(BL_{max}) - E(BL_{min}) = 11.77 - 3.24 = 8.53$. Second, using the same procedure, we construct the 75th/25th quartile comparison, with respective values of 390.7 mg/kg and 41 mg/kg, with a resulting $\Delta BL_2 = 2.86$. Finally, in a policy-specific comparison, we consider the effect of tightening US standards to those of Norway, resulting in a $\Delta BL_3 = 2.14$.

For conservative estimates of such reductions in BL levels on earnings, we exploit the IQ–BL function from Grosse et al. (2002), with a slope of 0.257, along with the earnings %–IQ slope of 2.00. Finally, we incorporate the expected present value of lifetime earnings of a 2-year old in 2000 $US of $723,300, based on a discount rate of 3% (Gold et al., 1996) and labor productivity gains of 1% per year which are consistent with historical trends.

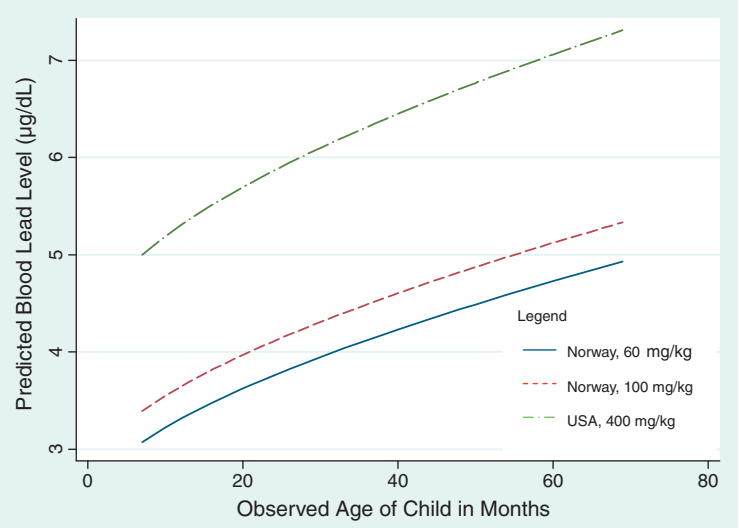

Fig. 4. Predicted (estimates retrieved from the full random effects generalized least squares model with year fixed at the sample mean of 2.13 years past 2000) blood lead in children (µg/dL) as a function of age of child by soil lead standard.

Table 4
Random effects logistic regression coefficients predicting threshold exceedance of blood lead levels in children (μg/dL).

	Model 1 μg/dL>15	Model 2 μg/dL>10	Model 3 μg/dL>7	Model 4 μg/dL>5	Model 5 μg/dL>3
$Age^{0.5}$	0.181***	0.207***	0.208***	0.210***	0.224***
	(0.0130)	(0.00908)	(0.00707)	(0.00621)	(0.00767)
Soil $lead^{0.5}$	0.129***	0.124***	0.103***	0.0903***	0.0550***
	(0.00592)	(0.00523)	(0.00398)	(0.00347)	(0.00380)
Year	−0.0803***	−0.0986***	−0.0568***	−0.0269***	−0.0344***
	(0.0135)	(0.00944)	(0.00735)	(0.00648)	(0.00779)
Constant	−6.143***	−5.099***	−3.759***	−2.714***	−0.229***
	(0.138)	(0.108)	(0.0791)	(0.0673)	(0.0716)
Wald X^2	721.13	1179.88**	1573.89*	1811.93	1087.42
Log likelihood	−9873.248	−17,609.407	−26,716.427	−33,074.096	−25,170.908
N	55,551	55,551	55,551	55,551	55,551
Census tracts	280	280	280	280	280

Standard errors in parentheses.
*** $p<0.01$.
** $p<0.05$.
* $p<0.1$

Extrapolating out from this syllogism, the gain produced by reducing blood lead levels by a standardized 1 μg/dL would thus be $3716. Our ΔBL_i reductions across our respective three *i* scenarios would thus respectively yield benefits per child of $31,699 for min/max, $10,628 for 75th/25th percentile, and $7952 for the US/Norway contrast. With a metro New Orleans cohort size of 23,000 born in 2000, total cumulative benefits would be $729.1 million for min/max, $244.4 million for 75th/25th, and $182.9 million for bringing Norwegian policies to the shores of Louisiana.

Given the explicit policy exercise of our Norwegian standards example, we examine these results in further detail, which should also help clarify the statistical exercises of the first two scenarios. All things held constant (i.e., age of child and year of observation), such a shift in national regulatory standards would reduce expected BL levels in children by 2.14 μg/dL, with a lower bound estimate of 2.00 μg/dL and an upper bound estimate of 2.28 μg/dL. Estimated reductions leverage regression results reported in Table 3, Model 1. Using the noted IQ–BL slopes, earnings–IQ slopes, and net present value of earnings data provided by Grosse et al. (2002: p. 567), we find that a shift in SL standards from 400 to 100 mg/kg provides each child with an economic benefit ranging from $4710 to $12,624. Multiplying estimated benefits to all 23,000 children born annually in New Orleans we arrive at a benefit per cohort estimate of $108.3 to 290.4 million ($US 2000), with the highlighted base case of $182.9 million in Table 6. This aggregate benefit is enjoyed by every subsequent cohort of equal size, providing us with the basis for a payback period estimate. Given the nonlinear properties of the age→BL and SL→BL relationships reported above, the marginal benefits of a policy intervention to minimize the deleterious effects of childhood exposure to SL are best at the front end of dose–response slopes.

While applying place-specific data and methodologies to the estimation of eventual lifetime effects of reducing lead exposure would be optimal, the multiple links between key analytical steps and the lack of regional data for both place-specific numeraires and methodologies necessarily limit the analysis to reliance on broader national proxies. Nevertheless, more recent studies of lead impacts suggest that in fact our analysis provides a lower-bound conservative benchmark for policy consideration. In particular, Rothenberg and Rothenberg (2005) find that a log-linear specification better identifies the lead–IQ relationship than the linear–linear specification proposed by Grosse et al. (2002) which is also used in this paper. As noted by the latter, benefits from lead reductions "using the correct log linear dose–response relationship between BPb and IQ are nearly 2.2 times those estimated using a….linear dose–response relationship for the same decrease in population BPb (Grosse et al., 2002, p1193)." In addition, Salkever (1995) applies superior labor market data to the IQ–earnings relationship described by Schwartz (1994), also leveraged in the present study. Salkever's (1995, p1) conclusions indicate that the eventual earnings benefits derived from IQ improvements due to lead exposure reductions should be upwardly revised by "at least 50%." Finally, the range of studies suggests that the greatest improvements in IQ/earnings impacts are likely towards the bottom of the socioeconomic hierarchy, where the affected populations of New Orleans are likely to fall. The combination of these methodological and data refinements underscores that the present paper's estimates are conservative lower-bounds for cohort earnings improvements from reduced lead exposure, thus particularly useful

Table 5
Random effects logistic regression odds ratios (and 95% CI) predicting threshold exceedance of blood lead levels in children (μg/dL).

	Model 1 μg/dL>15	Model 2 μg/dL>10	Model 3 μg/dL>7	Model 4 μg/dL>5	Model 5 μg/dL>3
$Age^{0.5}$	1.199	1.229	1.231	1.234	1.252
	(1.169 to 1.230)	(1.208 to 1.252)	(1.214 to 1.248)	(1.219 to 1.249)	(1.233 to 1.271)
Soil $lead^{0.5}$	1.138	1.132	1.109	1.095	1.056
	(1.125 to 1.151)	(1.120 to 1.144)	(1.100 to 1.117)	(1.087 to 1.102)	(1.049 to 1.064)
Year	0.923	0.906	0.945	0.973	0.966
	(0.899 to 0.948)	(0.889 to 0.923)	(0.931 to 0.958)	(0.961 to 0.986)	(0.952 to 0.981)
Wald X^2	721.13	1179.88	1573.89	1811.93	1087.42
Log likelihood	−9873.248	−17,609.407	−26,716.427	−33,074.096	−25,170.908
N	55,551	55,551	55,551	55,551	55,551
Census tracts	280	280	280	280	280

Note: Confidence intervals in parentheses.

Table 6
Assumptions and results of the economic benefit of three SL reduction scenarios, in 2000 US dollars.

	Max SL → Min SL			SL $P_{0.75}$ → SL $P_{0.25}$			USA SL → Norway SL		
	Lower bound	Base case	Upper bound	Lower bound	Base case	Upper bound	Lower bound	Base case	Upper bound
Assumptions									
A. Reduction in BL†	7.97	8.53	9.08	2.68	2.86	3.05	2.00	2.14	2.28
B. IQ-BL slope	0.185	0.257	0.323	0.185	0.257	0.323	0.185	0.257	0.323
C. Earnings-IQ slope	1.76	2.00	2.37	1.76	2.00	2.37	1.76	2.00	2.37
D. Present value of earnings of 2-year old	$723,300	$723,300	$723,300	$723,300	$723,300	$723,300	$723,300	$723,300	$723,300
E. Size of 2-year old cohort	23,000	23,000	23,000	23,000	23,000	23,000	23,000	23,000	23,000
Results									
Value of 1 IQ point (C × D)	$12,731	$14,460	$17,142	$12,731	$14,460	$17,142	$12,731	$14,460	$17,142
Gain per 1μg/dL (B × C × D)	$2355	$3716	$5537	$2355	$3716	$5537	$2355	$3716	$5537
Benefit per child (A × B × C × D)	$18,770	$31,699	$50,275	$6311	$10,628	$16,888	$4710	$7952	$12,624
Benefit per cohort (A × B × C × D × E)	$431.7 million	$729.1 million	$1.15 billion	$145.2 million	$244.4 million	$388.4 million	$108.3 million	$182.9 million	$290.4 million

Note: † The expected reduction in BL is derived from the full equation in Table 3, Model 1, with age and year fixed at sample means. In the base case of the max → min scenario, for example, expected BL reduction is calculated as: $E(BL_{max}) - E(BL_{min}) = 11.77 - 3.24 = 8.53$.

for policy considerations such as those that arise from the Norwegian standard.

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