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Linking Source and Effect: Resuspended Soil Lead, Air Lead, and Children's Blood Lead Levels in Detroit, Michigan

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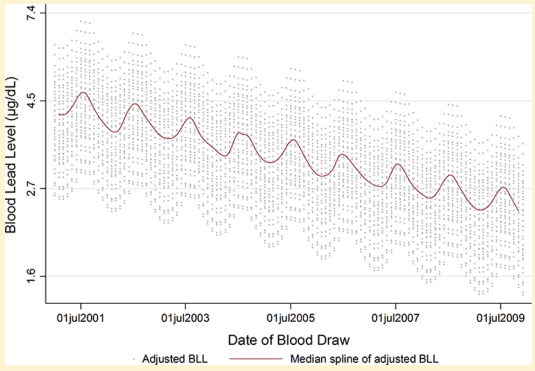
Supporting Information

ABSTRACT: This study evaluates atmospheric concentrations of soil and Pb aerosols, and blood lead levels (BLLs) in 367 839 children (ages 0−10) in Detroit, Michigan from 2001 to 2009 to test a hypothesized soil → air dust → child pathway of contemporary Pb risk. Atmospheric soil and Pb show near-identical seasonal properties that match seasonal variation in children's BLLs. Resuspended soil appears to be a significant underlying source of atmospheric Pb. A 1% increase in the amount of resuspended soil results in a 0.39% increase in the concentration of Pb in the atmosphere (95% CI, 0.28 to 0.50%). In turn, atmospheric Pb significantly explains age-dependent variation in child BLLs. Other things held equal, a change of 0.0069 $\mu g/m^3$ in atmospheric Pb increases BLL of a child 1 year of age by 10%, while approximately 3 times the concentration of Pb in air (0.023 $\mu g/m^3$) is required to induce the same increase in BLL of a child 7 years of age. Similarly, a 0.0069 $\mu g/m^3$ change in air Pb increases the odds of a child <1 year of age having a BLL ≥ 5 $\mu g/dL$ by a multiplicative factor of 1.32 (95% CI, 1.26 to 1.37). Overall, the resuspension of Pb contaminated soil explains observed seasonal variation in child BLLs.



INTRODUCTION

Lead (Pb) remains a serious threat to children's health and development—elevated levels of Pb in the blood are associated with impaired cognitive, motor, behavioral, and physical abilities.[1] Even lead-exposed children with blood lead levels (BLLs) below the World Health Organization (WHO) guideline of 10 $\mu g/dL$ for their entire lifetime experience measurable loss in cognition.[2] In response to health risks associated with BLLs below 10 $\mu g/dL$,[3−5] the U.S. Centers for Disease Control and Prevention (CDC) lowered the blood Pb reference value to 5 $\mu g/dL$ in May 2012.

Average BLLs in the U.S. (and globally) declined following the elimination of Pb from most product streams (e.g., gasoline, paint, water pipes, and solder used to seal canned goods). While airborne Pb used to be extremely high in cities, largely from the direct combustion of leaded gasoline and deposition of Pb oxides, much of the current airborne Pb is from these legacy sources. Contemporary air Pb is in the form of resuspended fine particulates.[6−10]

In this paper, we aim to explain the lingering sources of Pb in Detroit, Michigan by analytically reconciling a compelling empirical fact: *average BLLs for children in the northern hemisphere peak in summer and autumn and retreat during winter and spring periods.*[11] In Detroit, the BLLs of children follow this seasonal phenomenon (see Supporting Information (SI)).

As compared to the reference month of January, child BLLs are found to be between 11% and 14% higher in the months of July, August, and September (described in detail in Figure SI1 and Table SI1 of the SI). The seasonal behavior of child BLLs in Detroit is clear. Explaining this seasonal phenomenon is the aim of our paper, and it is our contention that any theory of contemporary Pb risk must logically account for this striking empirical observation.

Our intuition of what could plausibly account for the seasonality of child BLLs in Detroit is derived from a series of known facts. First, similar to many other postindustrial cities, elevated concentrations of environmental Pb are found

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throughout the Detroit metropolitan area,[12] with especially high concentrations of Pb in soils (400−800 mg/kg) located in the interior of the city[13] that correlate with spatial variation in children's BLLs.[14] Outdoor soil is a reservoir of legacy Pb from multiple anthropogenic sources and may explain why many household intervention efforts are unsuccessful.[15] Second, research shows that contemporary atmospheric concentrations of Pb spike during summer and autumn in many U.S. cities, including Washington, DC,[16,17] Boston,[18] Milwaukee,[19] New York,[20] New Jersey,[21] and Chicago.[22] In fact, seasonal variations in child BLLs and atmospheric Pb are strikingly similar. Third, previous research has demonstrated a remarkable ability to predict child BLLs based on climate variables.[23] Taken together, these facts are suggestive of a soil → air dust → child pathway of contemporary Pb exposure, where Pb-contaminated urban soils are resuspended as dust subject to seasonal precipitation regimes, wind, humidity, and other meteorological factors, with air Pb dust inhaled and ingested by unsuspecting children.

To evaluate this hypothesized pathway, this study uses temporally resolved atmospheric soil and Pb data, and matched BLL data from the Detroit metropolitan area. Statistical and numerical modeling are used to determine correlation strengths across a range of environmental and human variables, and specifically to target the contributions of air Pb to child BLLs likely derived from soil resuspension as opposed to point source air Pb emissions.

MATERIALS AND METHODS

To address the soil → air dust → child pathway for Pb exposure, a number of data sources are examined: blood Pb data for 367 839 children from the Michigan Department of Community Health (MDCH); atmospheric soil and Pb data from the U.S. Environmental Protection Agency's (EPAs) Interagency Monitoring of Protected Visual Environments (IMPROVE) database; [24] local weather data from the National Weather Service, National Climatic Data Center; and point location information on Pb-emitting facilities from the EPA's Toxic Release Inventory.[25]

Blood Pb Data. Children's blood Pb data for the tricounty area encompassing the City of Detroit was obtained from the Michigan Department of Community Health (MDCH). The data set contains blood samples collected from January 2001 through December 2009. Blood Pb measurements are reported as integers in units of micrograms per deciliter of blood (μg/dL). MDCH data also contain information on the census tract residential location of each child, the month and year of sample collection, child age in years (0−10), child sex (male = 1, female = 0), and the blood draw type (1 = capillary, 0 = venous). As with previous research,[26] we analyze child BLLs as a continuous variable (μg/dL) and as dichotomous variable ($\geq$5 μg/dL = 1, < 5 μg/dL = 0).

Atmospheric Pb and Soil Data. Atmospheric soil and Pb aerosol data were obtained from IMPROVE for the period of January 2001 to December 2009 (Station 261630001; additional stations presented in the SI). To derive atmospheric soil estimates, we use a mineral equation based on the elemental composition of soil.[27] Soil composition is derived by the quadratic sum of aluminum (Al), silica (Si), calcium (Ca), iron (Fe), and titanium (Ti) concentrations, assuming independence of measurement uncertainties as described by:

$$[\mathrm{d(soil)}]^2 = [2.20 \times \mathrm{d(Al)}]^2 + [2.49 \times \mathrm{d(Si)}]^2 + [1.63 \times \mathrm{d(Ca)}]^2 + [2.42 \times \mathrm{d(Fe)}]^2 + [1.94 \times \mathrm{d(Ti)}]^2 \quad (1)$$

Both atmospheric soil and Pb aerosol quantities are measured in units of μg/m^3. The quantity of soil derived using this equation is an estimate subject to spatial variability of soil composition and anthropogenic interferences.

Local Weather Data. Given that local weather conditions influence atmospheric concentrations of soil and Pb, we collected data describing the 24 h average relative humidity (%), sea level pressure (mb), temperature (°C), visibility (km), and wind speed (kmph) on the day of atmospheric readings.[6]

Point Source Pb. Under section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA), firms that release, transfer, or dispose of listed toxins are required to submit annual reports to the EPA detailing quantities of toxins emitted. Data are published under the Toxic Release Inventory (TRI) system.[25] Over the period of observation, 2001 to 2009, a total of 22 Pb-emitting facilities operated in Detroit. In analyses that follow, we estimate whether the presence of a point source polluter of Pb in a child's residential zip code predicts BLL outcomes.

Empirical Strategy. First, we analyze the extent to which daily variation in atmospheric Pb is explained by atmospheric soil.[6] The expectation is that atmospheric Pb and soil are statistically correlated, and that weather-adjusted Pb and soil concentrations in the atmosphere have distinct seasonality, rising and falling simultaneously over the calendar year. Insofar as atmospheric concentrations of soil and Pb move together seasonally, the first link in our soil → air dust → child pathway for Pb exposure is statistically corroborated. We use a least-squares regression procedure to examine the association between atmospheric soil and Pb. Formally, letting y_t denote atmospheric Pb in Detroit on day t our regression estimator is modeled as

$$y_t = \beta_0 + \beta_1 S_t + \Gamma_1 W_t + \varepsilon_t \quad (2)$$

where β_0 is the model constant; S_t is the atmospheric soil reading on day t; W_t is a vector of local weather conditions on the day atmospheric Pb and soil are measured, and ε_t is the error term, with $\varepsilon_t \sim IDD\ (0,\sigma_y^2)$. After testing whether atmospheric Pb levels in Detroit are statistically associated with atmospheric soil, we analyze the extent to which variation in child BLLs might be explained by atmospheric Pb concentrations, the second link in our hypothesized pathway.

To determine this relationship, we first analyze child blood Pb as a continuous variable (μg/dL). A census tract fixed effects regression procedure was used to analyze child BLLs. Letting y_{ijt} denote the BLL of child i in census tract j in month t the regression estimator is modeled as

$$y_{ijt} = \alpha_j + \beta_1 L_t + \beta_2 P_j + \Gamma_1 A_i + \Gamma_2 C_i + \Gamma_3 M_i + \Gamma_4 Z_{it} + \varepsilon_{ij} \quad (3)$$

where α_j is the census tract fixed-effect accounting for unobserved heterogeneity at the neighborhood level; L_t is the average monthly weather-adjusted atmospheric Pb level derived from eq 2 (child BLLs are indexed by month, warranting change in the time-step of Pb aerosol data); P_j is a dummy variable equal to 1 if a Pb emitting facility operates in the zip code of a child's residential location; A_i is the age of the child in years; C_i is a dummy variable equal to 1 if the blood draw was

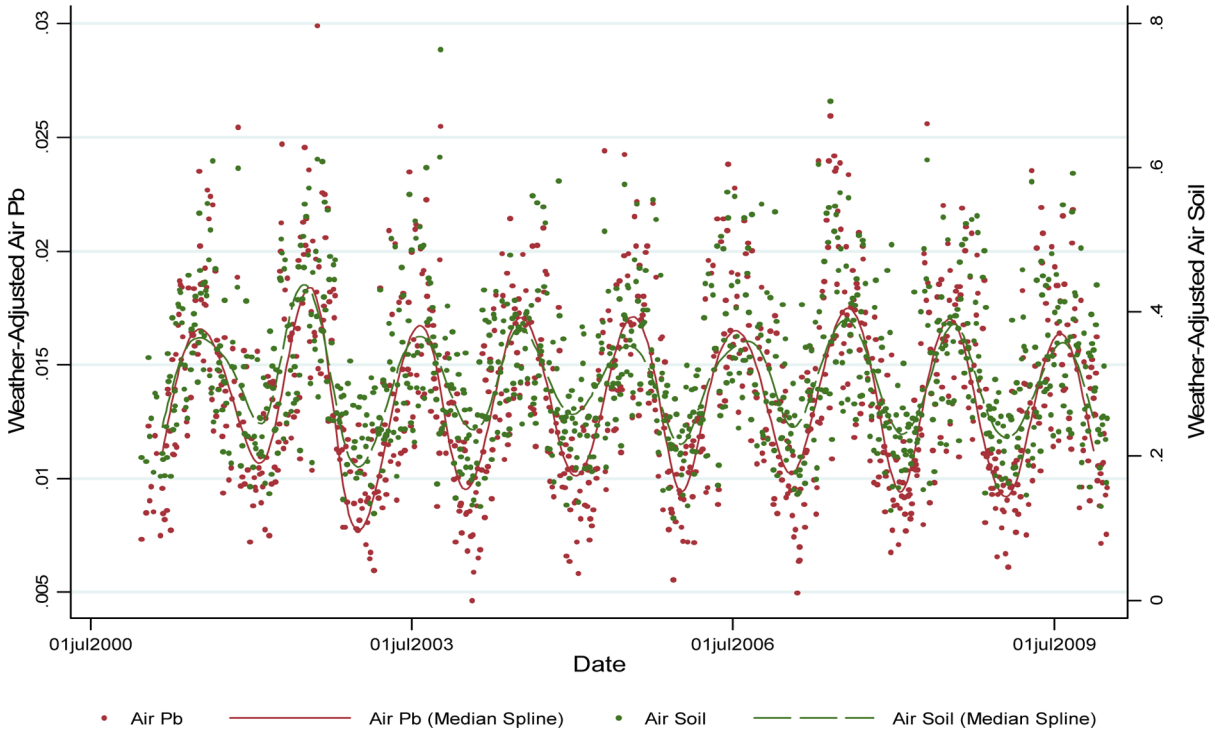

Figure 1. Scatterplot with median splines of weather adjusted air Pb and air soil in time (daily). Weather-adjusted air Pb and soil estimates ($\mu g/m^3$) (Models 3 and 4 described in SI) are graphed on the daily time-step, fitting distributions of air Pb and soil values with median splines. Time is on the x-axis, weather-adjusted atmospheric Pb is on the y_1-axis and atmospheric soil is on the y_2-axis. Air Pb values are in maroon and air soil values are in forest green, with corresponding median splines colored similarly.

capillary; M_i is a dummy variable equal to 1 if the child is male; Z_{it} corresponds to the year the blood draw occurred, and ε_{ij} is the error term, with $\varepsilon_t \sim IDD\ (0,\sigma_y^2)$.

Second, using a conditional fixed effect logistic regression procedure, we analyze whether or not a child's BLL is ≥5 μg/dL (corresponding to the CDCs new reference value) as a function of atmospheric Pb. Letting Y represent the threshold BLL of a child, where Y equals 1 if a child's BLL is ≥5 μg/dL, and Y equals 0 if a child's BLL is <5 μg/dL. We specify the following reduced form logistic equation for the probability of threshold exceedance (Y) for child i in census tract j in month t:

$$\begin{aligned}\text{Prob}(Y_{ijt} = 1 | L_t, P_j, A_i, C_i, M_i, Z_{it}) \\ = \Lambda[\alpha_j + \beta_1 L_t + \beta_2 P_j + \Gamma_1 A_i + \Gamma_2 C_i + \Gamma_3 M_i \\ + \Gamma_4 Z_{it}]\end{aligned} \quad (4)$$

where $\Lambda[\cdot]$ is the CDF of the logistic distribution. The definition of other terms carries from eq 3. The theoretical expectation under eqs 3 and 4 is that child BLL outcomes rise with atmospheric Pb.

Statistical models of child BLLs divide children by reported ages in years. Two reasons motivate this decision. First, low gastric exclusion for Pb in children and high dissolution potential of particulates (due to high surface area to mass ratios) are known to elevate BLLs in children in age-dependent ways.[28] Second, based on strong age-related risk factors observed for children,[29] we logically assume that the effects of airborne exposure are best observed in especially young children (ages 0–2) since they are *relatively* immobile and more insulated from other known sources of Pb (e.g., paint chips, direct interaction with Pb contaminated soils). Therefore, inasmuch as the proposed soil → air dust → child pathway for Pb exposure is a plausible description of contemporary Pb risk, coefficients on atmospheric Pb in eqs 3 and 4 ought to be noticeably higher in children less than 2 years of age.

RESULTS AND DISCUSSION

Atmospheric Soil and Pb Relationships. Ordinary least-squares regression models were rendered to predict atmospheric Pb as a function of air soil in Detroit, Michigan from January 2001 to December 2009. Variables are log transformed and elasticities are identified (model details provided in SI). The first model (Model 1) results indicate a 1% increase in atmospheric soil results in a 0.48% (95% CI, 0.38 to 0.58%) increase in atmospheric Pb. The association of air Pb and air soil, adjusting for local weather conditions, is presented as Model 2. Results are similar, with air Pb increasing 0.39% (95% CI, 0.28 to 0.50%) for every 1% increase in atmospheric soil. Standardized betas indicate the atmospheric soil is the strongest predictor of air Pb among variables examined. Note that both atmospheric Pb and soil are similarly sensitive to local weather conditions, rising significantly with average temperature and sea level pressure, and declining significantly with average visibility and wind speed (Models 3 and 4). While traffic-induced resuspension of nonsoil particles (derived from wheel weights, brake pads, etc.) is known to be a significant source of atmospheric particulate matter and metals,[30] including a proxy for this variable did not alter the effect observed from atmospheric soil (detailed discussion provided in SI Table SI2).

As shown in Figure 1, weather-adjusted air Pb and soil estimates (derived from Models 3 and 4) have remarkably similar cyclical properties of periodicity, angle function, and amplitude, peaking in the summer/autumn months of June, July, August, and September, and contracting noticeably in the

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Table 1. Conditional Fixed Effect Logistic Regression Odds Ratios Predicting Blood Pb ($\geq$ 5 μg/dL) in Detroit Children, 2001−2009[a]

	age 0 odds ratio	age 1 odds ratio	age 2 odds ratio	age 3 odds ratio	age 4 odds ratio	age 5 odds ratio	age 6 odds ratio	age 7 odds ratio	age 8−10 odds ratio
air Pb	1.316***	1.251***	1.177***	1.116***	1.093***	1.128***	1.111***	1.076***	1.089***
	(0.027)	(0.011)	(0.011)	(0.0098)	(0.010)	(0.014)	(0.020)	(0.030)	(0.026)
Pb facility	1.052	1.096**	1.123***	1.141***	1.076	0.893	0.911	0.804	1.057
	(0.091)	(0.043)	(0.050)	(0.048)	(0.046)	(0.054)	(0.077)	(0.100)	(0.109)
capillary draw	1.969***	1.590***	1.678***	1.909***	2.011***	1.823***	1.895***	1.394***	2.415***
	(0.083)	(0.029)	(0.037)	(0.041)	(0.045)	(0.056)	(0.084)	(0.136)	(0.257)
male	1.066	1.052***	1.197***	1.169***	1.225***	1.262***	1.304***	1.427***	1.311***
	(0.043)	(0.017)	(0.022)	(0.021)	(0.022)	(0.033)	(0.047)	(0.082)	(0.064)
year	0.877***	0.860***	0.833***	0.833***	0.827***	0.810***	0.813***	0.800***	0.798***
	(0.007)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.006)	(0.010)	(0.008)
N	19,046	75,852	58,322	66,288	66,862	33,878	18,571	8,280	13,122
log likelihood	−7328.2	−42312.1	−33543.5	−37095.8	−35683.7	−17420.0	−8752.6	−3455.4	−5050.8
χ^2	820.8	3684.2	3689.1	4076.6	4132.8	2297.2	1070.8	442.3	658.0
$N_{\text{census tracts}}$	316	370	354	351	350	332	308	287	285

[a]Standard errors in parentheses; ***p < 0.01, **p <0.05.

winter months of December and January. The behavior of both Pb and soil aerosol splines parallel the known cyclical behavior of blood Pb outcomes in children observed across many cities and time periods (details of cyclical behavior presented in SI Table SI1 and Figure SI1).[23]

Next, weather-adjusted air Pb and soil estimates are regressed on monthly dummy variables to observe more precisely how much coefficients rise in the months of July, August, and September. Consistent with findings of Laidlaw et al.,[6] compared to the reference of January, atmospheric Pb levels are 35.7% (95% CI, 28.8 to 42.6%) higher in July; 44.8% (95% CI, 38.0 to 51.7%) higher in August; and 40.0% (95% CI, 33.3 to 47.4%) higher in September. Air soil levels also significantly rise in the months of July (57.8%), August (62.2%), and September (54.0%) as compared to the winter month of January (Model 3, SI Table SI4). Overall, atmospheric Pb and soil have remarkably similar seasonal structure, corroborating the claim that a major source of atmospheric Pb is dust that is resuspended from Pb contaminated urban soils.

Atmospheric Pb and Blood Pb Relationships. Next, we turn attention to the second link in our hypothesized soil → air dust → child pathway. The natural log of child blood Pb is regressed on air Pb, controlling for child sex, blood draw type, and year of observation (statistical details provided in SI Table SI5). Neighborhood (census tract) fixed effects are incorporated into regression results. Recall that these results are grouped by child age. Log transformation of child blood Pb was necessary given high positive skew (5.87) and kurtosis (164.35), effectively eliminating the skew (0.32) and minimizing kurtosis (2.92). An increase of one standard deviation in weather-adjusted air Pb, ~0.0006 μg/m^3, induces an 8.04% (95% CI, 7.1 to 9.0%) increase in blood Pb for children less than 1 year of age. Air Pb significantly increases blood Pb in all children regardless of age, but the increase in child blood Pb for an equivalent unit of air Pb declines noticeably in age. For instance, while it takes approximately 0.0069 μg/m^3 of atmospheric Pb to increase child blood Pb by 10% in children 1 year of age, it requires about 3 times the amount (0.023 μg/m^3) of air Pb to induce a 10% increase in child blood for children 7 years of age.

Interestingly, we also observe an age-specific pattern on differences between males and females in average blood Pb levels (SI Table SI5). No statistically significant difference exists between males and females for children less than 1 year of age. At 1 year of age, a statistically significant difference between boys and girls is apparent and this difference rises incrementally with age. For instance, other things held equal, BLLs in males are only 1.5% higher than females at age 1, but 11.2% higher at age 7. Also worth noting, the effect of a Pb emitting facility in a child's zip code of residence behaves inconsistently by age grouping. For children ages 1−4, we find that residential proximity to a Pb emitting facility increases a child BLL by 2.5−5.5%, depending on the age of the child. For all other ages, proximity to a Pb facility has an effect indistinguishable from chance.

Next, conditional fixed effects odds ratios predicting the expected change in the likelihood of a child's BLL exceeding 5 μg/dL are reported in Table 1. A one standard deviation change in air Pb increases the odds of children less than 1 year of age recording a BLL $\geq$ 5 μg/dL by a multiplicative factor of 1.32 (95% CI, 1.26 to 1.37). Again, the deleterious effect of air Pb declines with age. By comparison, for 7 year olds, the probability of having a BLL $\geq$ 5 μg/dL increases 7.8% (95% CI, 2.0 to 13.8%) for an analogous 0.0006 μg/m^3 increase in air Pb. As with our linear model, residential proximity to a point source polluter of Pb is a significant predictor of whether a child's BLL is $\geq$5 μg/dL for children ages 1−3 only.

Figure 2 graphically illustrates the age-dependent association between blood Pb outcomes and air Pb. Overall, results corroborate our intuition that the effects of airborne exposure are most pronounced in younger children (ages 0−2) that are relatively insulated from other sources of Pb (e.g., paint chips, soil Pb).

The age-dependent association between child BLL and air Pb is further visualized by examining the seasonal behavior of age-stratified blood Pb levels and air Pb in time (Figure 3). Three items are notable. First, regardless of age group, average monthly child BLLs in Detroit are definitively seasonal, rising during the summer period and contracting in the winter period. Second, and again regardless of age group, the rise and fall of child BLLs correspond statistically to the behavior of atmospheric Pb. Third, the blood Pb responsiveness to air Pb behaves noticeably differently by age. For children less than 2 years of age, the rise and fall in blood Pb is more congruent

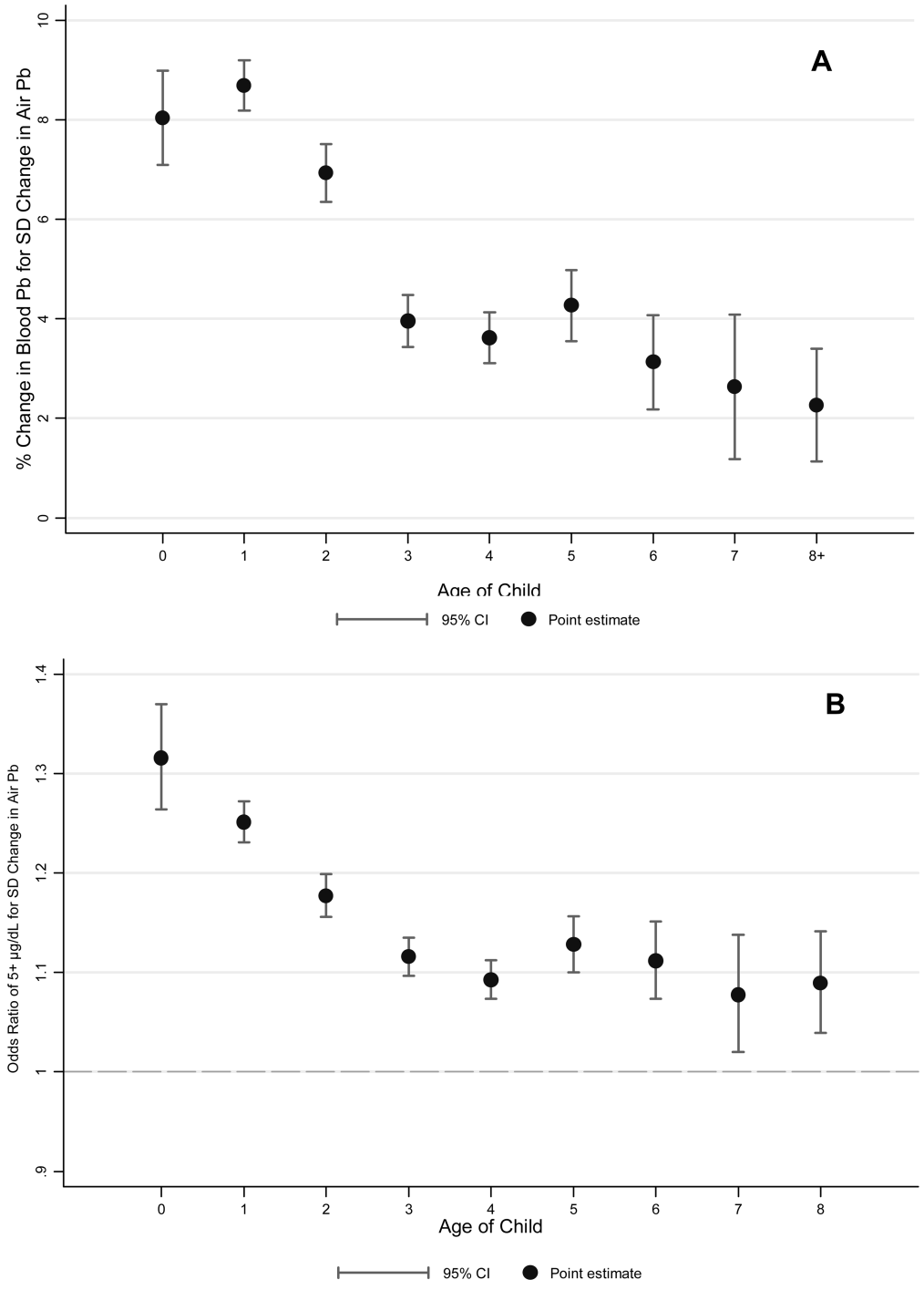

Figure 2. Change in blood Pb outcomes (A) and odds ratio of a 5 μg/dL increase in BLL (B) for standard deviation change in air Pb by age (error bars are 95% confidence intervals).

with the seasonal behavior of atmospheric Pb. To be more precise, the model fit of average monthly blood Pb as a function of air Pb is substantially higher for children age 0−2 as compared to children 6 years of age and older (R^2 = 0.706 vs 0.642). Similarly, a standard deviation rise in atmospheric Pb induces a 0.232 μg/dL (95% CI, 0.203 to 0.260 μg/dL) increase in the monthly average blood Pb of children age 0−2 as compared to a 0.152 μg/dL (95% CI, 0.130 to 0.173 μg/dL) increase in children ≥6 years of age.

Taken together, regression results on child BLL outcomes as a function of atmospheric Pb described in Table 1 and graphically summarized in Figure 3 corroborate the last link in our hypothesized soil → air dust → child pathway for contemporary Pb risk.

As a logical check on this hypothesized pathway, we end by considering a statistical counterfactual. We use the term counterfactual conventionally, meaning to imagine an outcome with modification of an antecedent. The antecedent that we logically modify is the soil source of air Pb. By regressing child BLL on the average monthly residual of eq 2, we have a test of the effect of air Pb on child BLLs absent soil resuspension. That is, the residual in eq 2 constitutes atmospheric Pb with

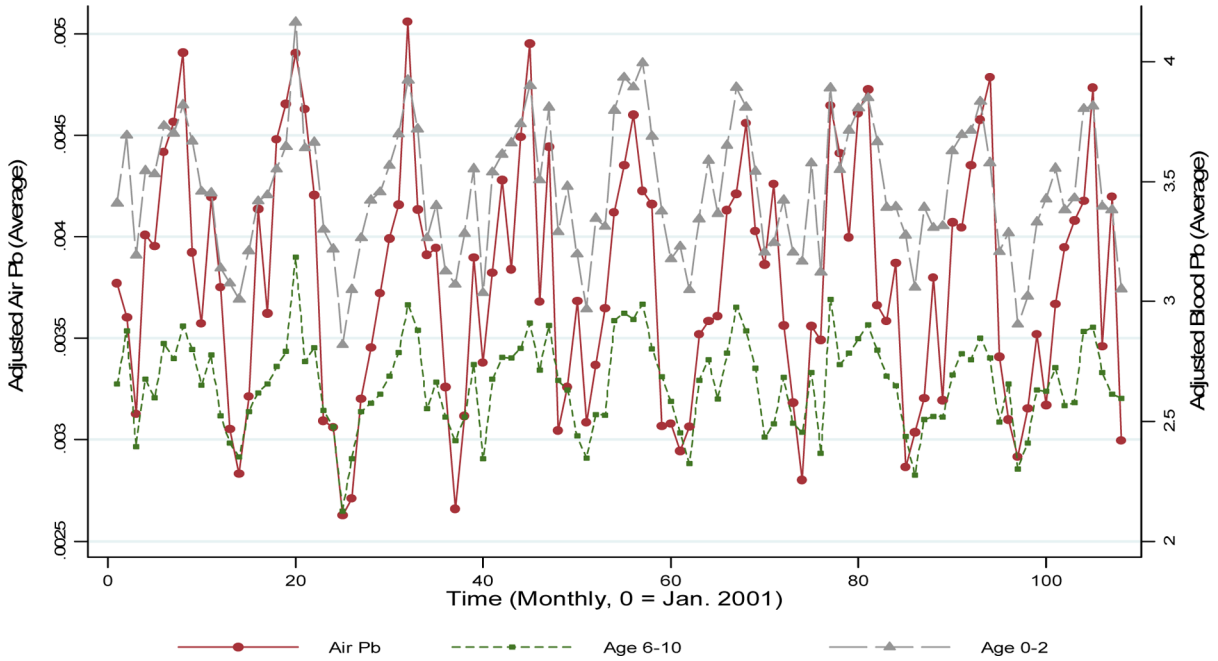

Figure 3. Weather-adjusted air Pb ($\mu g/m^3$) and blood Pb ($\mu g/dL$) by age group. Average monthly child blood Pb levels adjusted by local weather conditions, child gender, method of blood draw, and census tract fixed effects; air Pb estimates are from Model 2 (SI Table SI3).

underlying variation attributable to resuspended soil statistically removed. While the specific content of the residual is unknown, it reflects other unmeasured sources of air Pb; for example, Pb in paint generated by activities such as sanding of home exteriors. Such a test, in effect, interrupts the first link in our causal sequence. Substituting then L_t in eq 4 for ε_t in eq 2, creates then L_t in eq 4 for ε_t in eq 2, creates this counterfactual exercise (results presented in SI Table SI6). With the exception of children age 3, absent soil resuspension, we find that air Pb has an effect on child BLLs indistinguishable from zero. With the soil source of air Pb removed, air Pb has no observable effect on child BLLs—our casual sequence logically disappears.

Overall, empirical analyses corroborate the hypothesized soil → air dust → child pathway for Pb exposure in Detroit children. The data from this study show that daily variation in atmospheric Pb is associated statistically with daily variation in atmospheric soil, with both air Pb and soil showing remarkably similar seasonal properties that match known/observed seasonal variation in child BLLs. In addition, the data demonstrate that air Pb is a significant correlate of child BLLs regardless of age. As expected, and consistent with prior research, the association between child BLLs and air Pb is especially pronounced for children less than 2 years of age.[31] The main exposure mechanism for these young children is likely inadvertent ingestion of fine particulates through hand-to-mouth behavior, exacerbated by poor gastric exclusion for Pb and behavioral patterns that increase surface contact by hand (e.g., crawling). Direct pulmonary uptake remains an untested alternative for some portion of the BLL response. Results from our statistical counterfactual exercise show that absent soil resuspension, the effect of atmospheric Pb on child BLLs is indistinguishable from chance.

The air → dust → child exposure pathway described here may be observed in other urban areas where the legacy Pb deposition in soils remains a critical environmental burden to human health. Our findings suggest that the federal government's continued emphasis on Pb-based paint may be out-of-step (logically) with the evidence presented and an improvement in child health is likely achievable by focusing on the resuspension of soil Pb as a source of exposure. Given that current education has been found to be ineffective in reducing children's exposure to Pb,[15] we recommend that attention be focused on primary prevention of Pb contaminated soils.

■ ASSOCIATED CONTENT

Supporting Information

Sample statistics including details of regression model coefficients and a discussion of the appropriateness of spatial and temporal resolution. This material is available free of charge via the Internet at http://pubs.acs.org.

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Author Contributions

The manuscript was written through equal contributions of all authors. All authors have given approval to the final version of the manuscript.

Notes

The authors declare no competing financial interest.

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■ REFERENCES

(1) Binns, H. J.; Campbell, C.; Brown, M. J. Interpreting and managing blood lead levels of less than 10 $\mu g/dL$ in children and

reducing childhood exposure to lead: Recommendations of the Centers for Disease Control and Prevention advisory committee on childhood lead poisoning prevention. *Pediatrics* **2007**, *120* (5), E1285−E1298.
(2) Canfield, R. L.; Henderson, C. R.; Cory-Slechta, D. A.; Cox, C.; Jusko, T. A.; Lanphear, B. P. Intellectual impairment in children with blood lead concentrations below 10 mu g per deciliter. *New Eng. J. Med.* **2003**, *348* (16), 1517−1526.
(3) Bellinger, D.; Dietrich, K. N. Low-Level Lead-Exposure and Cognitive Function in Children. *Pediatr. Ann.* **1994**, *23* (11), 600−605.
(4) Lanphear, B. P.; Hornung, R.; Khoury, J.; Yolton, K.; Baghurstl, P.; Bellinger, D. C.; Canfield, R.; Dietricjh, K.; Bornscheim, R.; Rothenberg, S.; Needleman, H.; Schnaas, L.; Wasserman, G.; Roberst, R. Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. *Environ. Health Perspect.* **2005**, *113* (7), 894−899.
(5) Jusko, T. A.; Henderson, C. R.; Lanphear, B. P.; Cory-Slechta, D. A.; Parsons, P. J.; Canfield, R. L. Blood lead concentrations < 10 μg/dL and child intelligence at 6 years of age. *Environ. Health Perspect.* **2008**, *116* (2), 243−248.
(6) Laidlaw, M. A. S.; Zahran, S.; Mielke, H. W.; Taylor, M. P.; Filippelli, G. M. Re-suspension of lead contaminated urban soil as a dominant source of atmospheric lead in Birmingham, Chicago, Detroit and Pittsburgh, USA. *Atmos. Environ.* **2012**, *49*, 302−310.
(7) Mielke, H. W.; Gonzales, C. R.; Powell, E.; Jartun, M.; Mielke, P. W. Nonlinear association between soil lead and blood lead of children in metropolitan New Orleans, Louisiana: 2000−2005. *Sci. Total Environ.* **2007**, *388* (1−3), 43−53.
(8) Mielke, H. W.; Dugas, D.; Mielke, P. W.; Smith, K. S.; Smith, S. L.; Gonzales, C. R. Associations between soil lead and childhood blood lead in urban New Orleans and rural Lafourche Parish of Louisiana. *Environ. Health Perspect.* **1997**, *105* (9), 950−954.
(9) Schroeder, W. H.; Dobson, M.; Kane, D. M.; Johnson, N. D. Toxic Trace Elements Associated with Airborne Particulate Matter: A Review. *JAPCA* **1987**, *37* (11), 1267−1285.
(10) Zahran, S.; Mielke, H. W.; Gonzales, C. R.; Powell, E. T.; Weiler, S. New Orleans before and after Hurricanes Katrina/Rita: A quasi-experiment of the association between soil lead and children's blood lead. *Environ. Sci. Technol.* **2010**, *44* (12), 4433−40.
(11) Hayes, E. B.; McElvaine, M. D.; Orbach, H. G.; Fernandez, A. M.; Lyne, S.; Matte, T. D. Long-term trends in blood lead levels among children in Chicago-Relationship to air lead levels. *Pediatrics* **1994**, *93* (2), 195−200.
(12) Murray, K. S.; Rogers, D. T.; Kaufman, M. M. Heavy Metals in an Urban Watershed in Southeastern Michigan. *J. Environ. Qual.* **2004**, *33* (1), 163−172.
(13) Wendland-Bowyer, W. Free Press Soil Study: Samples Offer Cross-Section of Contamination. *Detroit Free Press*, 2003, January *23*, p 10A.
(14) Bickel, M. J. *Spatial and Temporal Relationships Between Blood Lead and Soil Lead Concentrations in Detroit, Michigan*. Masters Thesis, Wayne State University, Detroit, MI, 2010.
(15) Yeoh, B.; Woolfenden, S.; Lanphear, B.; Ridley, G. F.; Livingstone, N. Household interventions for preventing domestic Pb exposure in children. Cochrane Database of Systematic Reviews, 2012. *4*, Art. CD006047. DOI: 10.1002/14651858.CD006047.pub3.
(16) Green, N. A.; Morris, V. R. Assessment of public health risks associated with atmospheric exposure to PM2.5 in Washington, DC, USA. *Int. J. Environ. Res.Public Health* **2006**, *3*, 86−97.
(17) Melaku, S.; Morris, V.; Raghavan, D.; Hosten, C. Seasonal variation of heavy metals in ambient air and precipitation at a single site in Washington, DC. *Environ. Pollut.* **2008**, *155* (1), 88−98.
(18) USEPA. *Seasonal Rhythms of BLL Levels: Boston, 1979−1983: Final Report*; EPA 747-R-94-003; U.S. Environmental Protection Agency: Washington, DC, 1995.
(19) USEPA. *Seasonal Trends in BLL Levels in Milwaukee: Statistical Methodology*; EPA 747-R-95-010; U.S. Environmental Protection Agency: Washington, DC, 1996.
(20) Billick, I. H.; Curran, A. S.; Shier, D. R. Analysis of pediatric blood lead levels in New York City for 1970−1976. *Environ. Health Perspect.* **1979**, *31*, 83−90.
(21) New Jersey Department of Health (NJDH) and Senior Services Consumer and Environmental Health Services Environmental Public Health Tracking Project. *Analysis of Risk Factors for Elevated Levels of Blood Lead in New Jersey Children, 2000−2004. Demonstration Project on Geographic Patterns of Childhood Blood Lead and Environmental Factors in New Jersey*; Program 03074; Environmental and Health Effects Tracking, National Center for Environmental Health Centers for Disease Control and Prevention (CDC), 2007. http://www.state.nj.us/health/eohs/regional_state/air/analy_risk_pb_children.pdf.
(22) Paode, R. D.; Sofuoglu, S. C.; Sivadechathep, J.; Noll, K. E.; Holsen, T. M.; Keeler, G. J. Dry deposition fluxes and mass size distributions of Pb, Cu, and Zn measured in southern Lake Michigan during AEOLOS. *Environ. Sci. Technol.* **1998**, *32* (11), 1629−1635.
(23) Laidlaw, M. A. S.; Mielke, H. W.; Filippelli, G. M.; Johnson, D. L.; Gonzales, C. R. Seasonality and children's blood lead levels: Developing a predictive model using climatic variables and blood lead data from Indianapolis, Indiana, Syracuse, New York, and New Orleans, Louisiana (USA). *Environ. Health Perspect.* **2005**, *113* (6), 793−800.
(24) Interagency Monitoring of Protected Visual Environments. http://vista.cira.colostate.edu/improve/ (accessed 9 April 2012).
(25) United States Environmental Protection Agency (USEPA). *Toxic Release Inventory (TRI) Program*. http://www.epa.gov/tri/ (accessed 9 July 2012).
(26) Zahran, S.; Mielke, H. W.; Weiler, S.; Gonzales, C. R. Nonlinear Associations between Blood Lead in Children, Age of Child, and Quantity of Soil Lead in Metropolitan New Orleans. *Sci. Total Environ.* **2011**, *409* (7), 1211−1218.
(27) *IMPROVE soil estimation calculation*. http://vista.cira.colostate.edu/improve/Publications/GrayLit/023_SoilEquation/Soil_Eq_Evaluation.pdf (accessed 9 April 2012).
(28) Juhasz, A. L.; Weber, J.; Smith, E. Impact of soil particle size and bioaccessibility on children and adult lead exposure in peri-urban contaminated soils. *J. Hazard. Mater.* **2010**, *186* (2−3), 1870−1879.
(29) Walter, S. D.; Yankel, A. J.; von Lindern, I. H. Age-specific risk factors for lead absorption in children. *Arch. Environ. Health* **1980**, *35* (1), 53−58.
(30) Harrison, R. M.; Jones, A. M.; Gietl, J.; Yin, J.; Green, D. C. Estimation of the Contributions of Brake Dust, Tire Wear, and Resuspension to Nonexhaust Traffic Particles Derived from Atmospheric Measurements. *Environ. Sci. Technol.* **2012**, *46* (12), 6523−6529.
(31) Pino, P.; Walter, T.; Oyarzun, M. J.; Burden, M. J.; Lozoff, B. Rapid drop in infant blood lead levels during the transition to unleaded gasoline use in Santiago, Chile. *Arch. Environ. Health* **2004**, *59* (4), 182−187.