The Elephant in the Playground

confronting lead-contaminated soils as an important source of lead burdens to urban populations

Gabriel M. Filippelli and Mark A. S. Laidlaw

ABSTRACT Although significant headway has been made over the past 50 years in understanding and reducing the sources and health risks of lead, the incidence of lead poisoning remains shockingly high in urban regions of the United States. At particular risk are poor people who inhabit the polluted centers of our older cities without the benefits of adequate nutrition, education, and access to health care. To provide a future with fewer environmental and health burdens related to lead, we need to consider the multiple pathways of lead exposure in children, including their continued contact with dust derived from inner-city soils. Recent research into the causes of seasonal variations in blood-lead levels among children has confirmed the importance of soil in lead exposure. "Capping" lead-contaminated soil with lead-free soil or soil amendment appears to be a simple and cost-effective way to reduce the lead load for urban youth.

THERE IS A COMMON—but misguided—perception that lead poisoning is no longer a public-health problem in the United States. Indeed, effective regulations against leaded gasoline and lead-based paint have dramatically reduced lead exposure. Unfortunately, however, the threat to urban neighborhoods across the nation is still very real.

Department of Earth Sciences, Indiana University–Purdue University Indianapolis (IUPUI), Indianapolis, IN 46205
E-mail: gfilippe@iupui.edu.

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In the 20th century, two new applications turned lead toxicity into a widespread problem. First, lead-based paints became the gold standard for new homes in the early part of the century, prized for their durability and bright white color. Second, lead additives for gasoline were developed as an "anti-knock" engine formula in the 1920s, and the explosion of motor vehicles in the middle part of the century was fueled by gasoline doped with tetra-ethyl lead. By the 1970s, Americans encountered lead at every turn.

A number of scientific champions brought lead hazards to the public's attention. In the 1950s, Cal Tech geochemist Clair Patterson was conducting experiments to pinpoint the age of various rocks, but his results were skewed by consistent lead contamination. Further studies showed that lead levels were elevated in certain waters, soils, organisms (Settle and Patterson 1980), even Arctic ice—and most troubling, in the human body. Over the next three decades, Patterson helped to lead a crusade against lead that attracted the vociferous opposition of industry groups. But this effort eventually convinced lawmakers and regulators to outlaw lead in pipes, solder, and finally in gasoline (Bryson 2003). In a parallel fashion, Herbert Needleman fought against industrial, and even university, opponents to his findings of irreversible neurological defects as a result of lead poisoning of children (Needleman, Tuncay, and Shapiro 1972; for an account of this struggle, see Rosner and Markowitz 2005). As a result of efforts by Patterson and Needleman, among others, the number of children affected by lead poisoning in the United States has been reduced by over 80%. In the movies, this triumph would signal the closing credits—but in the real world, the story continues.

While less than 2% of children aged 0–5 years in the United States suffer from lead poisoning today (a value much improved but still a serious public health epidemic), children living in the urban centers of the East and Midwest have lead poisoning rates of 15–20% (NHANES 2003). In 1980, Clair Patterson presaged the current state of environmental insults to urban populations: "Sometime in the near future it probably will be shown that the older urban areas of the United States have been rendered more or less uninhabitable by the millions of tons of poisonous industrial lead residues that have accumulated in cities during the past century" (NRC 1980, p. 271). While many might consider lead poisoning a closed chapter in the annals of public health, recent research shows that the dangers still exist, and that they are elevated among the most at-risk children in our society.

In this essay, we discuss how children are exposed to lead and how they are affected, where the continued sources of lead are in urban areas, and how earth scientists can inform health scientists to enhance the health of the population—particular the poor people who typically inhabit the polluted centers of our older cities. These children face a number of challenges related to lead poisoning, including inadequate nutrition, which leads to soil pica behavior, as well as higher lead absorption rates due to iron deficiency anemia, and inadequate edu-

cation and access to health care. The primary cause of chronic lead loading in urban youth is their continued contact with dust derived from lead-enriched inner-city soils. This new paradigm of the exposure pathway of children to lead, which has been verified by our recent research into the causes of seasonal swings in children's blood lead levels, points to a relatively simple and cost-effective way toward reducing the lead load for urban youth.

EFFECTS OF LEAD ON HUMANS

Compared to other chemicals of environmental concern, the uptake mechanisms and toxicological effects of lead are relatively well understood. The major pathway of lead uptake in humans is via ingestion, where lead is absorbed in the intestine and incorporated in the body, initiating a series of toxicological effects. Physiological absorption potential for lead is dependent mainly on age: the portion of ingested lead that is taken up in the body is typically less than 5% for adults, whereas it is as high as 50% for children (Maddaloni et al. 1998; Ziegler et al. 1978). Because of their high absorption efficiency and the rapid neural differentiation during early brain and nervous system development, children are especially vulnerable to permanent effects of lead poisoning, which include irreversible neurologic disorders such as lowered intelligence and increased rates of attention-deficit hyperactivity disorder. When lead is incorporated into bone material, the bone becomes a long-term source of lead in the body, releasing that lead on time scales of months to years. For this reason, children treated by medical interventions like blood chelation may continue to have toxic levels of lead in their blood (Roberts et al. 2001). Furthermore, as neither the placenta nor mammary glands are a perfect barrier to lead, pregnant and lactating mothers with elevated blood-lead levels may themselves pose a health risk to babies and fetuses.

The health standards for lead levels in blood have been revised downward over the years as medical research has determined toxicological effects of lead in even low quantities. The U.S. Centers for Disease Control and Prevention (CDC) in 1991 chose 10 micrograms per deciliter as an initial screening level for lead in children's blood, although subsequent studies are still unable to find a "safe" lower level of lead, with levels below 10 micrograms per deciliter still causing some toxicological effects (Canfield et al. 2003; Chiodo et al. 2007; Nigg et al. 2008). The full spectrum of toxicological effects of lead is still not known and deserves further study. But the persistent presence of lead in children is a public-health issue of the first order.

From Occupational to Residential: The Paint Problem

Lead has been used by humans for thousands of years and its toxicity has been known for centuries, but it was not until the Industrial Revolution that this issue became a widespread problem. Lead was once a topic of concern mainly in occupational health circles, as severe lead poisoning was observed in industrial facilities including tetraethyl lead plants (Markowitz and Rosner 2002). But in the late 1800s, lead crept into homes in paint. Lead had been added to paint for centuries—distinctive colors are achieved with the addition of metals to paints, with bold white being the benefit of lead addition. However, a boom in residential housing development in the early part of the 20th century resulted in national-scale advertising blitz for "white lead paint" and the application of lead-based paints in millions of new homes. The addition of lead, in practice up to 15% by weight, enhanced the durability and flexibility of paints. Many single- and multi-family dwellings had lead-based paints in their walls, window sashes, and doorways. Even brick and stone houses often employed lead-based paints in windows and doors. Although lead enhanced durability, paint has its functional limits, and its degradation around friction points (doorways, window sashes), combined with the exploratory nature and oral fixation of young children, resulted in the first widespread tragedy of lead. As children were being admitted to hospitals with symptoms of severe and chronic lead poisoning, the link to lead-based paints became apparent. In the 1940s, pressure from the health profession and consumer advocate groups succeeded in legislation prohibiting the addition of lead to house paints. Although lead is still allowed in industrial applications like bridge paints, the banning of lead in house paints, which began in 1950 and became final in the early 1970s, gave hope for a lead-free future for children. This lead-free future never came to fruition for two reasons: the explosion of automobile use after World War II, fueled by leaded gasoline (more on this later), and the inevitable degradation of lead-based paint in and around homes.

The continued poisoning of children from lead-based paints was a sadly predictable outcome. The fact that house paint applied after 1950 was usually lead-free didn't change the lead content of old paint. Anybody who has refinished an older home is aware of the problem—what do you do with the lead paint on the walls, sills, and doorways? The popular way to refinish trim work and windows is the most problematic. Sanding of lead-based paints converts the paint from a glue-type solid with limited bioavailability into millions of fine particulates with relatively high lead content *and* very high bioavailability, due to the high surface-area/mass ratio of these particles. In many of the acute cases of lead poisoning in children in the United States today, contact with lead has resulted from home refinishing or remodeling. This problem can bridge class and race—rehabbing of older homes is often a luxury of the upper-middle class, as they restore a historic home to its original luster.

To confront this problem, many health and environmental agencies at the national, state, and local levels have been waging a war of remediation and education about the hazards of lead. Most of the remediation efforts have been focused on safely removing or covering lead-based paints in homes. With millions of dollars in grants and incentives to owners and landlords, lead-based paints have been removed or covered all over the country. The agencies involved have touted these efforts as a success, holding up the clear improvement in the number of children affected by lead over the past 25 years. In a national health assessment survey in the late 1970s, 88% of the nation's children (0–5 years of age) had blood-lead levels above that deemed safe by today's standards (10 micrograms per deciliter); in a follow-up survey in the 1990s, that number was down to 2.2% (NHANES 2003), with annual improvements seen in interim surveys up to today. When people are asked what they consider the key pathway for lead to children, they invariably respond that kids get lead poisoning from eating paint chips.

When medical, scientific, and regulatory findings reach the collective psyche of society, a paradigm is formed. This paradigm, that lead-based paints still constitute the biggest risk to children with respect to lead, and that the remediation of lead-based paint sources has in the past and will continue in the future to provide the chief benefits to children's health, is firmly entrenched. The seduction of this idea is easy to see—images of toddler's bite marks of painted window sills, X-rays revealing paint chips in a child's stomach, a photo of a white-clothed team of remediation experts removing lead-based paint from a building—can be superimposed to create an image of a neat, clean, and effective solution to this problem. This seduction is now even in the courtroom, where several high-profile cases brought before juries revolve around large paint producers, like Sherwin-Williams, who are being sued for producing lead-based paints over 60 years ago. Clearly, corporate and industrial responsibility should extend to producing products that knowingly endanger the health of people and the environment, and the idea of reparations to support remediation of this public-health menace is appealing.

But what if the paradigm is inadequate? What if poor, urban youth are no longer being poisoned just by chipping paint, but now predominantly by the soil around them? What if the vision of a white-clad team of specialists sweeping through a housing project, removing lead from the walls and leaving in its wake sparkling new lead-free paint, needs to be replaced by an image much more messy and comprehensive to solve this problem of social injustice? How will we know when to shift resources for education and remediation in another direction? We believe that the time is now. This belief is bolstered by a series of findings that suggest that soil—particularly the fine dust that derives from soil during dry periods and blankets horizontal surfaces inside and outside of homes—is a prime culprit in the poisoning of our children by lead. The inability of remediation of paint alone to reduce the blood-lead levels of urban youth is one clue that we have been missing a key additional source of lead to our children.

Lead-Contaminated Dust from Soils as a Vehicle for Chronic Lead Poisoning of Urban Youth

We have hit the wall in terms of improving the lead-poisoning outlook for some children, particular those living at or below the poverty level in older cities. Even after decades of active intervention, these urban youth have lead-poisoning rates that are up to 10 times the national average. In 1994, a summary statement from a national health survey stated that "the exposure to lead at levels that may adversely affect the health of children remains a problem especially for those who are minority, urban, and from low-income families. Strategies to identify the most vulnerable risk groups are necessary to further reduce lead exposure in the United States" (Brody et al. 1994, p. 277). These socioeconomic risk factors include poor nutrition with the potential for pica behavior (a subconscious desire to ingest soil and dust to overcome nutritional deficits), and inadequate pediatric health care. Additionally, and of critical importance for improving the health outcome of urban youth, these risk factors also include poor home maintenance with high rental percentages, significant proportions of urban housing with high dust and dirt exposure, and relatively low awareness of the links between behavior and health.

In particular, the continued poisoning of urban youth from the very dirt and soil upon which they live is the key to a new emerging paradigm—namely, that the continuing source of lead exposure to children is lead-enriched soils, and particularly dust resuspended from these soils, that are prevalent in cities, especially older ones (Filippelli et al. 2005; Laidlaw et al. 2005; Mielke and Reagan 1998). The source of lead to the soils includes degraded lead-based paints, but also lead deposited from tailpipes, the result of 60 years of leaded gasoline combustion. In fact, the improvement in the national average of blood-lead levels may be due in large part to the banning of lead as an additive in gasoline in 1980.

The production and use of lead for gasoline additives was spurred by the need to control the explosion of gasoline in the cylinders of internal combustion engines. The formulation of tetraethyl lead as a fuel additive was "perfected" in the 1920s, resulting in the adoption of a global fuel standard that contained about 2% lead oxide by weight. An early warning sign went up when scores of workers in plants producing tetra ethyl lead additives were severely poisoned by lead toxicity, although a concerted industrial cover-up limited public awareness of this situation (Markowitz and Rosner 2002). But concerns about the environmental impacts of tetraethyl lead were shelved as the automobile age dawned and affordable transportation dramatically altered the American landscape. The peak in lead use for this application followed the trend in automobile use in America: by 1970, 250,000 tons of lead were used in gasoline and emitted from tailpipes every year.

Roadway Sources of Lead

Overall, about 5 million metric tons of lead was deposited in the environment as a result of the combustion of leaded gasoline (Mielke et al. 1997). Almost all of that lead was originally deposited very close to roadways, with aerosolized combustion products containing lead initially deposited within about 50 meters of a roadway if no obstructions were present. The fate of deposited lead then depended on the conditions of the depositional area. Although intersections of busy streets may have received over one metric ton of lead per year, their impervious surfaces led to continual runoff of lead-enriched particulates down storm drains (and from there into treatment plants or directly into rivers). If the particulate lead was deposited instead on a grassy fringe, like a front yard or park, the lead was effectively retained (Filippelli et al. 2005). In such a setting, the insolubility of lead leads to surface peaks in lead concentration of soils; in relatively undisturbed soils, this surface-lead enrichment may be the product of decades of lead deposition and may reach levels above 1,000 ppm. Thus, surface soils became the repositories of lead deposited over decades—in the case of older roadways, the proximal soils might retain almost all of the lead deposited on them over a period of about 60 years.

The roadway lead is generally highly bioavailable. Immediately upon combustion, tetraethyl-derived lead is precipitated as tiny and poorly mineralized oxides and oxyhydroxides, both of which are much more susceptible to dissolution in gastric systems than is the well-mineralized lead found naturally in soils. Therefore, dust originating from urban soils contaminated by anthropogenic lead is more toxic per unit mass than naturally occurring lead dust. Because of its deposition source, much of the tetraethyl-derived lead is associated with clay size fraction (less than 0.2 microns in diameter) in urban soils. The small size leads to this clay soil fraction being the predominant component of soil "resuspended" during dry and windy periods. Thus, lead in dust blown from urban soils is more potent and concentrated than would be expected from simple measurements of the lead content of the bulk soil.

Diffuse Soil Lead and Children's Health

The original sources of lead in the environment were point sources, including lead-based paints, gasoline-emitted lead, and lead emitted from smelters. But an analysis of many urban areas reveals that these point sources have, to some extent at least, been redistributed to produce regions of lead enrichment (Filippelli et al. 2005). Several factors can lead to redistribution of lead-enriched particles and soil, but the recurrence of a general urban enrichment of soil lead, termed "diffuse soil lead," has been documented in many regions.

One of the characteristics of lead distribution in surface soils of several older cities is a distinct decreasing trend from city center to suburban surroundings, a legacy both of lead deposition, redistribution, and smearing of original point

sources, and less lead deposition in newer suburban neighborhoods due to recent lead controls. The urban roadway example shows both the impact of the point source of lead deposition from leaded gasoline as well as the diffuse soil lead that blankets urban regions. In other words, even at distances away from the roadway, beyond where direct lead deposition occurs (and far away from structures using leaded paint), the background level for lead is significantly higher in the urban roadway transects than in suburban transects. This urban-suburban gradient is one overriding factor affecting the amount of lead loading to individuals, a factor that we will discuss on a larger scale and with respect to human health.

In many urban areas of older cities, large segments of children below the age of six are above the action level for lead in blood; this has been well documented by Howard Mielke of Tulane University for New Orleans and Baltimore, David Johnson for Syracuse, and more recently by us for Indianapolis. The actual distribution of blood-lead levels exceeding action limits is getting more difficult to obtain due to privacy concerns, but in the past blood-lead values could be collected from health department records down to the level of a street address, providing an outstanding way to examine the environmental factors in human health.

To explore the concept of diffuse soil lead and its potential role in affecting children's health in Indianapolis, Filippelli et al. (2005) performed a coupled soil survey and epidemiological analysis. The soil sampling criteria included being greater than 50 meters from roadways and from structures that might have contributed lead-based paint, and was augmented by aerial photographic records over Indianapolis from several time slices (1940, 1970), to rule out the potential for inadvertently sampling soils from disturbed, excavated, or filled areas that might have surface-lead contents characteristic of artificial materials rather than natural soil. As one can imagine in a rapidly developing urban area, this criteria narrowed down acceptable sites to only about 100 distinct sites, many of which were in parks, cemeteries, and school grounds. In contrast to roadway and house-side soil sampling, which might exhibit lead concentrations above 1,000 ppm, the highest soil-lead concentrations were below 480 ppm. The lowest lead concentrations averaged about 50 ppm, which is a typical value for soils in this region. The highest soil-lead concentrations were focused in a bulls-eye pattern directly over the old urban areas of Indianapolis, where the diffuse soil-lead content averaged over 200 ppm (Figure 1). Beyond this central hot spot, lead concentrations decreased toward the outskirts of the city, ultimately falling to background values in the suburban to rural fringes of the city. The central peak is consistent with the long history of lead use in the downtown, but the generally high values even away from point sources supports the argument of a redistribution of lead over time.

Combining the distribution of soil lead with that of children's blood-lead poisoning reveals several important characteristics of diffuse soil lead as a potential contributor to children's health. First, the similarity in the distribution of ele-

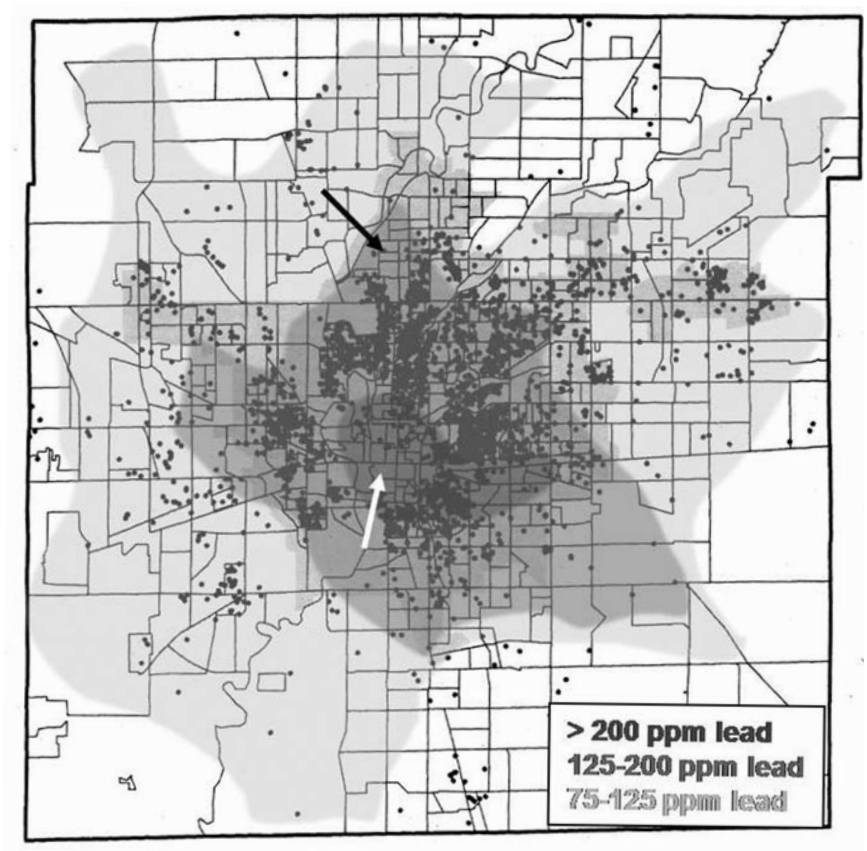

FIGURE 1

Satellite infrared image of Indianapolis (Marion County) in central Indiana; north is up. The concentration of diffuse soil lead (in shaded regions) displays a characteristic pattern of urban enrichment trending toward background values in suburban and rural areas. The overprint of high diffuse soil lead presented here corresponds roughly with the distribution of elevated blood-lead levels in children, displayed as circles for the distribution of children's venous blood samples exhibiting lead concentrations above the level of concern (10 micrograms per deciliter) from 1992 to 1994 in Indianapolis (data from the Indiana State Department of Health). Most elevated blood samples are from the downtown region (significant overlap of multiple positive results occur in this region), with some additional scattered positive results ranging toward the older suburban development to the west and the east. The arrows point to regions with high soil lead but low incidence of lead poisoning, at apparent odds with the direct link between soils and blood. A number of factors, however—socioeconomic status, age, population distribution—act as filters between potential exposure and toxicology. In the case of the lighter lower arrow, the lack of lead poisoning is due to the lack of homes in this industrial corridor, and in the case of the darker upper arrow, this highlights a main street that displays a socioeconomic divide between poverty-line neighborhoods in the near-urban area and upper-middle and upper-class neighborhoods in the northern suburban area.

SOURCE: FILIPPELLI ET AL. 2005.

vated soil- and blood-lead values in the downtown areas reveals the potential for diffuse soil lead to be an additional and important factor in children's blood-lead levels. Second, population patterns definitely have some influence on the health distribution data. For example, some areas of the downtown have the highest concentration of diffuse soil lead but almost no incidences of childhood lead poisoning; in this case, this is because this region is an industrial area with no housing (Figure 1, lower lighter arrow). Finally, a socioeconomic filter likely comes between the exposure factor (diffuse soil lead) and the epidemiological factor (blood-lead levels). As an example, the central north-south thoroughfare in Indianapolis is called Meridian Street, which in its near-urban stretch is lined with apartment buildings and relatively low-income rental neighborhoods. This area exhibits high diffuse soil lead concentrations and a high incidence of blood-lead poisoning. But as Meridian moves north into the older suburbs, it becomes a National Historic Landmark, lined by mansions, including the Governor's Residence, with four-acre expansive manicured lawns. The streets bordering Meridian along this stretch are also characterized by well-maintained older homes, owned by upper-middle class families. Although diffuse soil lead concentrations are still high in this area, children's blood-lead levels are generally low; in fact, no incidences of blood-lead levels exceeding action limits were reported in 1992–1994 along this portion of Meridian and surrounding blocks (Figure 1, upper darker arrow).

Although many factors influence the relationship between geology and human health in the story of lead, it is clear that we do not yet understand all of the confounding factors. Furthermore, the generalized approach presented above provides a reference point for further work, but it does not effectively integrate health and geologic data, nor does it present clear recommendations that geologists can make to health specialists in further reducing this public-health hazard, beyond the incredibly costly and disruptive solution of removing all of the contaminated surface soil in urban areas and replacing with clean fill. Several bridging efforts are now being pursued to help further the human health–environmental quality linkage in the context of eliminating childhood lead poisoning. Beyond simply documenting lead distribution and its public-health implications, research has also examined lead in a more detailed manner as a toxicological agent with predictable behavior. For example, isotopic techniques have been utilized to examine the entry mechanisms of lead into the body and the cycling of lead within the body, with a goal of pinpointing lead toxicity in individuals and thus more closely coupling prevention and treatment (Graziano et al. 1996; Gwiazda and Smith 2000). Another tool of promise in assessing lead poisoning is predictive modeling of children's blood-lead levels using climatologic data.

CLIMATIC FACTORS AND A BLOOD-LEAD PREDICTIVE MODEL FOR HEALTH CARE

Several studies have identified a seasonal trend in blood-lead levels, with average monthly blood-lead levels of children from urban areas increasing significantly in summer months, perhaps partly due to increased exposure to lead-based paint on window sills and through increased contact with soils containing lead during the summer. Summer increases of children's blood-lead levels were so prominent over many years in Syracuse that a group researchers led by David Johnson at the State University of New York, College of Environmental Science and Forestry, concluded that the phenomenon is probably caused by the interaction between climate and soils, leading to enhanced dust-lead loading to children (Johnson, McDade, and Griffith 1996).

To better understand this climate/soil/human health link, several projects are underway to investigate in detail variations in children's blood-lead levels as a function of climate and soil factors in several urban areas. The ultimate goal of this effort is to develop a predictive model, whereby a medical researcher can better assess the likelihood of lead poisoning based on seasonal and weather-related factors, as well as blood-lead level data. Laidlaw et al. (2005) used a number of independent climatologic variables, including average monthly soil moisture, PM10 (fine particulates less than 10 microns in diameter, an indicator of air quality and dust concentration in the atmosphere), wind speed, and temperature obtained from state and federal government data sources. They also used blood-lead databases obtained from local and state governmental sources.

This model indicates that soil moisture, wind speed, PM10, temperature, and the monthly dummy variables for March, April, June, July, August, and September explain 87% of the variation in monthly average child blood-lead level concentrations (Laidlaw et al. 2005). Based on this multiple regression model and recently published results from several other American cities (Laidlaw and Filippelli 2008), it appears that the seasonality in children's blood-lead levels is controlled by exposure to lead dust originating from contaminated soils and suspended in the air when several weather-related environmental conditions are present: temperature is high, soil moisture is low, and atmospheric PM10 is elevated (Figure 2). When temperature is high and evapotranspiration maximized, soil moisture becomes low, lead-enriched PM10 dust disperses in the urban environment and is manifest by elevated lead-dust loading. In this case, exposure is via increased dust loads in homes and on contact surfaces, with ingestion being the uptake mechanism and toddlers at greatest risk due to their behavior (crawling, tactile exploration, hand-to-mouth behavior). Although further work using detailed tracking of lead, possibly involving lead isotopic studies, may help to elucidate the connection between seasonality and blood-lead values, the ability of geochemical and meteorological factors to predict blood lead supports the supposition that external loading and exposure drives much of the blood-lead

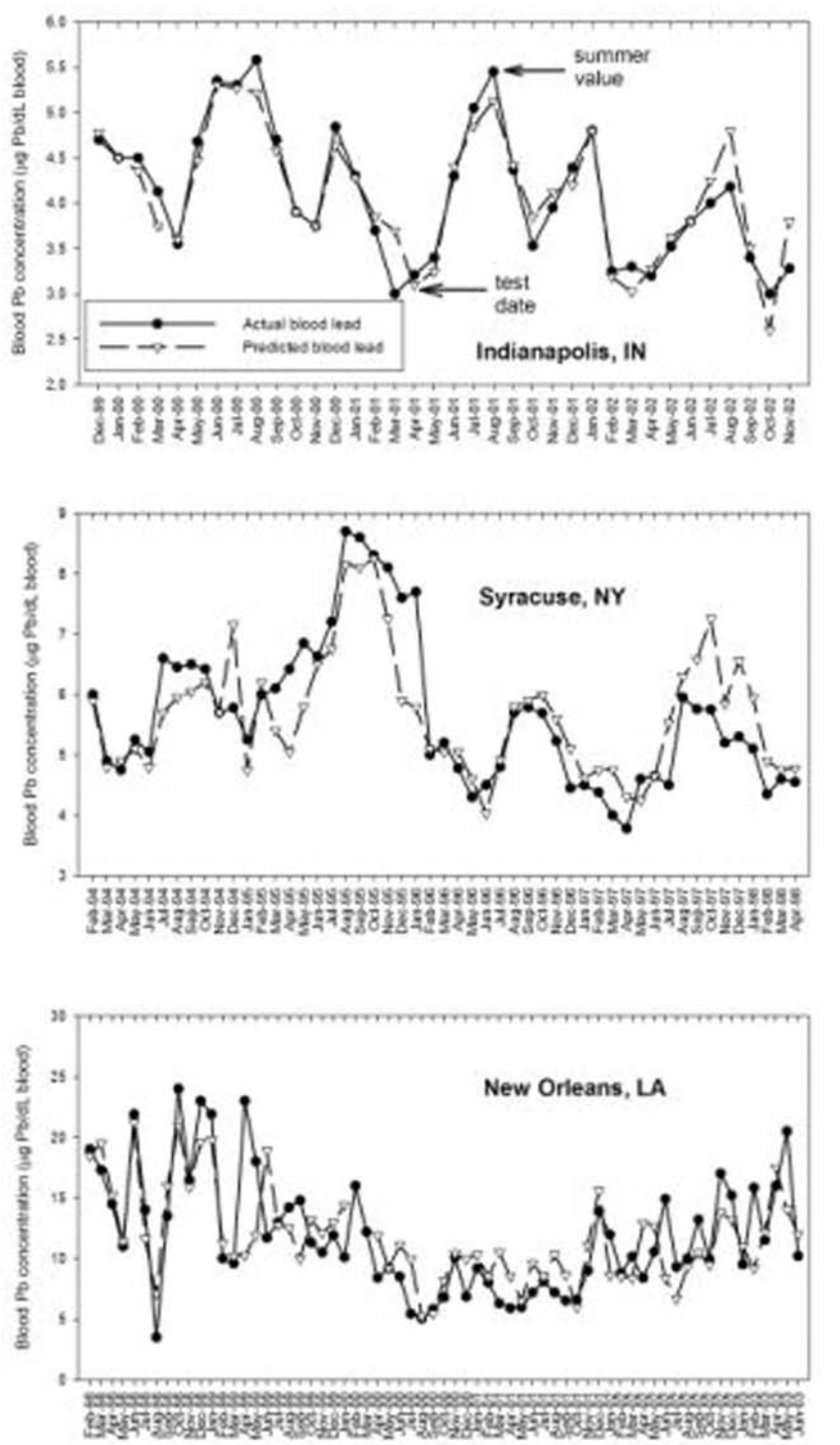
summer value
test date
Actual blood lead
Predicted blood lead
Indianapolis, IN
Blood Pb concentration (µg Pb/dL blood)
Syracuse, NY
New Orleans, LA

FIGURE 2

concentrations. Because resuspension of lead from contaminated soil appears to be driving seasonal child blood-lead fluctuations, lead contaminated soil in and of itself may be the primary driving mechanism of child blood-lead poisoning in the urban environment.

However, the seasonal deposition of lead-enriched dust and its ingestion by children may not be the only factor driving the observed seasonal patterns in blood-lead levels. Recent work indicates a potential role for increased sunlight-induced vitamin D synthesis in the summer, which increases gastrointestinal lead absorption and skeletal lead mobilization at least in children from 4–8 years of age (Kemp et al. 2007). In fact, multiple interacting causes may be at play, with factors including lead-enriched soil deposition, as well as age, race, sunlight exposure, and diet.

In addition to the development of hypotheses related to the incorporation of lead into children's system, a promising result of these modelling analyses is the ability to predict toxicity in a given population. In other words, through easily collected atmospheric and soil data a health researcher can determine the expected variation in blood-lead levels of the general population and use this data as a context for evaluating blood-lead level data from individual patients (Figure 2). This is particularly important when attempting to treat blood-lead poisoning using discrete venous sampling events: a blood-lead level for a given patient in the spring, under conditions of high soil moisture, could be significantly higher in the same patient just several months later, when atmospheric conditions increase ambient lead loading.

A NEW WAY FORWARD?

In summary, a newer paradigm of urban lead loading has emerged, one that helps to explain continued chronic lead poisoning and seasonal patterns in blood-lead levels of children. Unlike discrete point sources like lead paint and industrial contact, which are still responsible for most cases of acute lead poisoning, diffuse soil lead is the main avenue for urban lead loading of children. The diffuse soil lead comes from several sources, including leaded gasoline and degraded lead-based

FIGURE 2 CAPTION: *Best-fit model results to predict blood-lead levels in children from Indianapolis, Syracuse, and New Orleans, compared to actual monthly average blood-lead levels. This type of effort can be used to better treat lead poisoning from a public-health perspective by providing clinicians with predicted trends of blood-lead levels (functionally calculated as a percent deviation from mean) at a given blood sampling event, allowing them to calculate the potential increase or decrease with time given normal exposure. The clinician who is analyzing blood-lead test results from, for example, late winter–early spring, could predict that the patient's summertime blood-lead levels would likely be about 50% higher. If this higher predicted level is above the level of concern for the clinician, a follow-up test in the summer might be recommended.*

SOURCE: AFTER LAIDLAW ET AL. 2005.

paints, but in a sense the source no longer matters: because of the ability of surface soils to retain lead, these soils themselves have become the new risk factor for children's health in lead-loaded cities. If the action level for blood lead in children is dropped to 5 or even 2 micrograms per deciliter, we suggest that the dust resuspension paradigm will be central to predicting patterns in lower-level lead poisoning in children, leading to the need for new mitigation strategies.

Widespread contamination of urban soils creates a different challenge for mitigation of lead risks for children, one based on removing surface soils from human contact. Most mitigation efforts for heavily contaminated soils have involved soil removal and replacement, an extremely disruptive, expensive, and not terribly effective option for controlling lead sources in urban areas (Farrell et al. 1998; Weitzman et al. 1993). Recently, another approach, which is much cheaper and appears to be as effective as soil removal, was tested by Howard Mielke in New Orleans. Mielke's approach is simply to cover the contaminated yard soils with about 15 cm of lead-free soil, which in the case of New Orleans came from the nearby Mississippi levee (Mielke et al. 2006). At a fraction of the soil removal cost, this clean soil is simply graded over the old soil layer, hydroseeded (a slurry of seeds and moisture-retaining fill mixture sprayed onto the ground), and left to grow a lawn. This approach "caps" the lead-contaminated soils, removing them from contact by children. The result of initial work is a substantial reduction in the blood-lead levels of children living in the affected homes. Interestingly, Mielke observed that over the course of several months after treatment, soil lead levels in the treated sites began increasing. This increase was due to the resuspension and deposition of soil dust from adjacent untreated yards and neighborhoods that still had high soil lead concentrations. This finding not only supports the paradigm of diffuse soil lead as a culprit in urban areas, but it also indicates that a comprehensive treatment approach is required to provide a long-term benefit.

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