

Primary Prevention and Health Outcomes: Treatment of Residential Lead-Based Paint Hazards and the Incidence of Childhood Lead Poisoning

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Abstract

In order to quantify the effects of primary prevention on health outcomes, I investigate the impact of treatment of lead-based paint hazards in housing units on childhood lead poisoning at the census tract level in Chicago, IL. I use the findings from the analysis to weigh the costs of lead interventions against the potential benefits of reducing blood lead levels in children. Childhood lead poisoning presents a particularly useful example of the efficacy of preventive care in reducing the incidence of a disease. There is a clear, well-defined pathway of exposure (deteriorating lead paint in older homes) and no method of secondary care that effectively mitigates the negative health effects. I find that a one-tenth percentage point increase in the proportion of older housing units that have been remediated is associated with a five to six-tenths percentage point reduction in the incidence of childhood lead poisoning. Citywide, this is roughly 1.75 cases of lead poisoning averted for every housing unit remediated. Furthermore, I find evidence that the effect of remediations in preventing the disease has improved over time. The lower bound estimates of the benefits associated with the reduction in lead poisoning - increased expected lifetime earnings and reduced medical expenditures - is roughly twice the estimated costs of the remediations.

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1 Introduction

Childhood lead poisoning is the second most prevalent preventable disease in children¹ in the United States (CDC, 2005). In general, the average blood lead level (BLL) in children has been declining over the last three decades, down 90% since 1978 (EPA, 2005). However, levels among low-income, urban children, particularly those living in older housing, remain high (CDPH, 2004). Furthermore, recent medical studies of the effects of lead poisoning on cognitive ability in children have demonstrated negative impacts at levels previously thought to be below the threshold for concern (Koller et al., 2004).

There are many factors that have contributed to the decline in the prevalence of lead poisoning over the past few decades, foremost of which are the phaseout of leaded gasoline beginning in the early 1970's and a greater awareness of the disease. Currently, the greatest source of lead exposure in children is deteriorating lead-based paint in old, poorly-maintained housing. Thus, future reductions in the incidence of the disease in children will come from the treatment of lead paint hazards in the home.

The primary goal of this paper is to investigate the role that the treatment of lead-based paint in homes has played in the declining rate of childhood lead poisoning in U.S. cities. More specifically, I intend to estimate the impact of lead remediations, a non-medical approach to primary prevention, on the incidence of elevated blood lead levels (EBLs) in Chicago, Illinois. Using these findings, I also provide evidence that the benefits to society from remediations and the resulting reduced lead exposure in children far outweigh the costs of the necessary lead hazard treatments.

Chicago is an ideal location for the study because it has one of the highest rates of childhood lead poisoning as well as one of the most active lead prevention programs in the nation. Chicago has more total cases of lead poisoning in children per year than any other U.S. city (CDPH, 2004).

Rather than measure whether individual remediations prevent cases of lead poisoning, I investigate whether the aggregate remediation efforts in a given area (U.S. census tracts) will reduce the incidence of childhood lead poisoning there. I use the number of housing

¹Asthma is the most prevalent.

units remediated in several different ways to reflect the “health of the housing stock” in an area over time (i.e. the degree to which the housing stock is free of lead hazards). As the health of the housing stock improves, children face a lower risk of exposure to lead and the incidence of lead poisoning should decline.

I observe each variable by year from 1997 to 2003 and by census tract. I begin by estimating a simple linear model of the incidence of childhood lead poisoning. The panel dataset also allows me to include year dummy variables to account for aggregate unobserved factors that drive down EBLs homogeneously across tracts over time. There are potential sources of unobserved heterogeneity that differentially impact the incidence of EBLs in census tracts (i.e. there are “problem tracts” that have high levels of EBLs and lead remediation in homes). If this is the case, OLS estimates of the effect of remediations on EBLs will be biased, leading to a spurious positive relationship between remediation and lead poisoning. Alternatively, I estimate fixed effects models to account for any unobserved census tract impacts. Reverse causality between the remediation variable and the dependent variable (i.e. a fraction of remediations are ordered because a child living there has tested positive for lead poisoning) is another potential problem. Again, OLS estimates will be biased upwards towards zero. To account for the endogeneity of the remediation variables, I investigate different ways to calculate the variable that will purge any reverse causality.

Once I account for census tract fixed effects, a one-tenth percentage point increase in the percentage of housing units remediated (a reasonable increase given the annual changes observed in the data) is associated with a five to six-tenths decrease in the incidence of childhood lead poisoning. Furthermore, the negative impact of remediations on EBLs strengthens over time. There was a sharp decline in the incidence of EBLs from 1997 to 2003 not captured by remediations or any of the control variables. This is evidenced by the strong, negative and consistently increasing coefficients on the time dummy variables, most likely accounting for the reduction in lead from gasoline and increased awareness. When controlling for various housing and sociodemographic characteristics in the panel analysis, the proportion of the population that is black race, a significant risk factor in other studies, is no longer an important predictor of lead poisoning in children. I also provide evidence that certain risk factors have declined in importance over time, however, not because of increased remediations. It

appears that unobserved factors such as increased awareness have had a differential impact over time on various subpopulations.

The increase in lifetime earnings and the reduced medical care expenditures from a one-tenth percentage point increase in remediation are far greater than the corresponding costs. The lower bound estimate of the benefit-cost ratio is 2:1 while the upper bound is roughly 10:1. The largest dollar benefit by far is the increase in the discounted net present value of expected lifetime earnings from an increase in cognitive ability in children (measured as an increase in IQ). The upper bound estimates are reasonable for Chicago given the findings in other studies of nationwide benefits of reductions in mean BLLs.

2 Background

2.1 Exposure Pathways and Health Effects in Children

The two greatest sources of lead (by volume) released into the environment are lead-based paint and leaded gasoline. They are responsible for nearly equal shares of the lead burden in the U.S., measured as millions of metric tons each (Mielke and Reagan, 1998). Lead was used as an additive in almost all industrial and residential paints, primarily to increase durability and improve appearance. It was used as an additive in gasoline to increase octane levels.

Lead was blended with gasoline for the majority of the 20th century. It was first added to gasoline in the U.S. in the 1920's. The usage peaked in the early 1970's at over 2 million metric tons per year (Mielke and Reagan, 1998). However, due to the growing realization in the 1970's of the health effects of lead along with the inclusion of the catalytic converter to almost all vehicles sold in the U.S., the use of lead in gasoline began to decline.² The official phaseout began in 1973 and culminated on January 1, 1996, when the sale of leaded gasoline was banned in the U.S. as part of the Clean Air Act.

Lead was commonly used as an additive in residential paint in the U.S. from the late 19th century until 1978. Lead paint manufacturers voluntarily reduced the lead content of paint throughout the 1950's, although the levels were still high enough to produce a

²Leaded gasoline is not compatible with catalytic converters, which were added to automobiles sold in the U.S. in the 1970's to reduce air pollution.

significant risk of exposure (ATSDR, 1988). Paint manufactured prior to 1950 can contain up to 50% lead by weight (Reissman et al., 2001). In June 1977, the U.S. Consumer Product Safety Commission reduced the legal level of lead in paint to 0.06% by dry solid, effectively banning the practice altogether. Although the ban covered the manufacturing of lead paint, it did not have an impact on existing paint in homes (Mushak and Crocetti, 1990).

Besides paint and gasoline, cultural sources of lead exposure that tend to originate outside the U.S. include pottery, cosmetics, and folk remedies brought to the U.S. from Mexico, Southeast Asia, Africa, and the Middle East (Trotter, 1990; Parry and Eaton, 1991; Shannon 1998). Although these sources can lead to extreme cases of lead poisoning in children (Parry and Eaton, 1991; Shannon, 1998), they present a much smaller source of exposure (both in volume of lead and in the number of children exposed) than paint and gasoline.

While leaded gasoline has been an important source of lead exposure in the U.S., lead found in deteriorating residential paint is currently the greatest source of lead exposure to children (CDC, 1997; Shannon and Graef, 1992; Lanphear et al., 1998; Jacobs et al., 2002; Koller et al., 2004). Prior to the ban in 1978, almost all household interior and exterior paint contained lead. Thus, the overwhelming majority of housing units built before 1978 contain some amount of lead paint. If the paint surfaces are not maintained in homes, it is common for those living there to be exposed to lead contaminated paint chips, dust, and soil. In 2002, an estimated 38 million housing units in the U.S. contained lead-based paint, of which 24 million had lead hazards, defined as deteriorated paint, dust lead, or bare soil (Jacobs et al., 2002).

Several studies found evidence that lead dust is the primary pathway of exposure among lead paint sources (Jacobs et al., 2002; Koller et al., 2004). Evidence also suggests that lead from deteriorating lead-based paint contributes more lead to contaminated soil than any other source (Lanphear and Roghmann, 1997; Jacobs et al., 2002; Brown and Jacobs, 2006). Thus, even though a large amount of fine lead dust was deposited in the environment during the widespread use of leaded gasoline, lead from paint poses the greatest current threat of lead exposure to children.

Children are at a greater risk of exposure to lead in paint than adults for several reasons.

Infants are especially susceptible to lead contaminated floor dust from deteriorating paint, as young children tend to exhibit high levels of hand to mouth activity (CDC, 1997; Koller et al., 2004). Slightly older children also face an elevated risk of exposure to lead, although from lead-based paint chips and dust, mainly found around windowsills. In addition, lead gives paint a sweet taste that increases the likelihood that children will ingest the lead based paint (LSH, 2005).

The negative effects of lead are particularly damaging to young children. First, children absorb and retain more lead than adults (Ziegler et al., 1978; McCabe, 1979; Koller et al., 2004;). The lead is stored in bones and soft tissue and may later remobilize into the blood stream (Shannon and Graef, 1992). Furthermore, at this age, the neurological development of children is particularly susceptible to the negative influences of lead (McCabe, 1979; Koller et al., 2004; Goldstein, 1990). A well-developed literature argues that these neurological impacts reduce cognitive ability and lead to behavioral problems and learning disabilities later in life. Exposure to lead can also lead to kidney failure, damage of the central nervous system, and even death in cases of extremely high exposure.

Once a child is lead poisoned, there is little that medical intervention can do to reverse the detrimental effects lead causes in the human body. Children who have been severely poisoned generally undergo chelation therapy, a procedure that helps remove some of the lead from the body and reduce blood lead levels. Although removing lead from the bloodstream can help minimize potential future health effects and declines in cognitive ability, it cannot reverse the damage already done from lead exposure (Rogan et al., 2001). While lead has only a two week half life in the blood, it has roughly a two year half life in the brain (Lidsky and Schnedier, 2003). Furthermore, chelation is only effective in reducing BLLs in the long-run when provided in conjunction with remediations (Shannon, 1998; Chisolm Jr., 1990). If the sources of exposure are not addressed, then children moving back into the housing units will be reintroduced to the original hazards and BLLs will likely rebound.

The most effective way to combat lead poisoning is to prevent it from occurring. Thus, those involved in childhood lead poisoning prevention have focused their efforts on eliminating the major source of exposure (i.e., lead-based paint in older poorly-maintained housing).

2.2 Lead Inspections and Abatements

There are three different classes of lead inspection. The most comprehensive type of inspection is the “lead inspection” during which all painted surfaces inside and outside of the home are sampled and tested. In a “risk assessment”, an inspector will locate and test any damaged paint surfaces, dust on floors and windows, surfaces licked or chewed on by children, and soil. A “lead hazard screen” is a limited version of the risk assessment. An inspector will determine which type of inspection is appropriate for a housing unit based on a visual inspection. For the purposes of this paper, I will refer to any type of environmental screen as an inspection. Refer to Table 1 for definitions of key terms.

In Chicago, a housing unit can be inspected for three reasons: 1) if the unit was built before 1978 and receives federal funding, e.g. Federal Housing Authority and Section 8 housing, 2) if a child under six years of age living in the unit has tested positive for blood lead poisoning, or 3) if the housing unit has violated a city housing code.

In all three of the cases listed above, if a lead hazard is identified, the hazard must be addressed with mitigation or an abatement. According to state law, a hazard is sufficiently mitigated if “the area no longer produces a hazardous level of leaded chips, flakes, dust, or any other form of lead substance” (IDPH, 1992). The definition of abatement, a stricter standard, requires that all lead-based paint surfaces be removed or covered such that there is no threat of lead exposure for at least twenty years. In Chicago, lead inspectors are by law allowed to force property owners to either mitigate or abate, depending on the severity, any lead hazards identified in an inspection. For the purposes of this paper, all mitigations and abatements will be referred to as lead remediations (Table 1).

2.3 Related Literature

This analysis is the first to measure the large scale effects of lead hazard control on the incidence of lead poisoning. In an aggregate level study by Bailey et al. (1998), the authors found that after controlling for a host of proven risk factors, census tracts in a county with a history of abatement activity had lower rates of childhood lead poisoning than tracts in a similar county with no such history. The study did not have any inspection or remediation

data and did not attempt to measure the specific impact of remediations on the incidence of childhood lead poisoning.

The majority of studies that examine the impact of lead hazard control have focused on the efficacy of individual remediations in preventing future EBLs in children currently living there. The purpose of the studies is to determine whether a remediation could actually increase exposure rather than result in the intended reduction in exposure. They do not examine the population effects of large scale remediation efforts. The literature has shown that when done according to the current EPA guidelines which include the control of lead dust, addressing lead-based paint hazards in a unit can effectively reduce the blood lead levels of children living there (Charney et al., 1983; Farfel et al., 1994; Galke et al., 2001). There are studies that have found contradictory evidence that children's blood lead levels increase when they are reintroduced to remediated housing units (Chisolm, 1990; Rey-Alvarez and Menke-Hargrave; 1987, Shannon and Graef, 1992). However, each of these studies noted that this was not the case when the remediations were done correctly and in particular, when lead dust was addressed. Furthermore, all of these studies were performed prior the passage of the EPA's lead safe work practices, which stress the importance of lead contaminated dust (EPA, 1992).

A large body of work has been devoted to understanding the various risk factors associated with childhood lead poisoning. While these studies do not test the impact of remediations, they help identify primary exposure pathways and sensitive populations, which are crucial in constructing models of childhood lead poisoning. As previously discussed, the hypothesized primary exposure pathway among children is deteriorating lead-based paint in older housing units. The risk factor literature supports this claim. Numerous studies have found that the age of housing, particularly housing built before 1950, is an especially strong predictor of EBLs (Pirkle et al., 1998; Reissman et al., 2001; Sargent et al., 1995; Sargent et al., 1997). Furthermore, variables that might proxy for the condition of housing, such as renter versus owner occupancy and vacant housing have proven to be positively correlated with childhood lead poisoning (Reissman et al., 2001; Sargent et al., 1995; Sargent et al., 1997). Other risk factors for lead poisoning include black race, Hispanic origin, and income, which remain significant across all ages of housing (Crocetti et al., 1990; Melman et al.,

1998; Pirkle et al., 1998). Gender and urban setting are not significant predictors of EBLs once the aforementioned risk factors are considered (Melman et al., 1998; Pirkle et al., 1998).

Recent literature has focused on the reduced cognitive ability in children exposed to what were previously considered low levels of exposure. The literature suggests that rather serious declines occur in cognitive ability, generally measured as a reduction in IQ points, among children exposed to levels under the current definition of lead poisoning (Koller et al., 2004). Several studies raise questions relating to the methods of the former group of analyses and urge caution in interpreting the seriousness of the demonstrated reduction in IQ (Kaufman, 2001A; Kaufman, 2001B; Hebben, 2001). I provide a more in depth discussion of this debate and the estimated reductions of IQ from lead exposure at the end of this paper.

3 Data

3.1 Data Source and Study Population

The Chicago Department of Public Health (CDPH) provided the total number of lead screens and positive cases³ for children under six years of age by census tract and year for 1997 through 2003. Given that individual blood lead data are confidential, the aggregation must be done in such a way that individual data cannot be “backed out” of the statistics. To maintain the confidentiality of the individuals in the dataset, any incidence statistic calculated with less than five children (an arbitrary cutoff set by the CDPH) in the denominator must be suppressed. For instance, if, in a given geographical unit, less than five children are screened for lead poisoning, reporting the percentage of those that tested positive could jeopardize the confidentiality of those individual results. The largest number of census tracts suppressed in any given year is 45 out of the 866 census tracts in Chicago blood lead data.⁴

The CDPH provided their environmental data files, which contain inspection, violation, and remediation data for every address inspected for lead in Chicago.⁵ Unlike the blood lead data, the results of individual lead inspections are available to the public. Furthermore,

³The current CDC definition of childhood lead poisoning is a screen above 10 $\mu\text{g}/\text{dL}$.

⁴Census tracts with less than five screens tend to have zero or very few children under six years of age living there. The average number of children less than six years of age in census tracts in which blood lead data are suppressed is 14 compared to 315 for all other tracts.

⁵The file was first cleaned by Abt Associates in Cambridge Massachusetts as part of another project.

lead inspections are not necessarily tied to positive blood lead results in children and thus, releasing inspection data at the housing unit level does not compromise the confidentiality of medical tests results.

For every address inspected in Chicago, the database contains whether a violation occurred and a subsequent remediation if necessary. For inspections performed between 1997 and 2003, the data contain violation status, whether the violation has been addressed and the date of remediation.

The control variables were taken from the 1990 and 2000 U.S. Censuses. The census variables are reported for each census tract in Chicago for these two years. Chicago was divided into 876 census tracts in 1990 and 2000. The number of demolitions of residential buildings in Chicago was provided by the Chicago Metropolitan Agency for Planning (CMAP).

The blood lead and inspections files contain information from 866 and 785 census tracts, respectively.⁶ I received data for seven years, 1997 through 2003, for each census tract, for a total of 6,069 tract/year observations. The inspection data contains information on 3,465 tract/year combinations. Tracts with no housing data are assumed to have had no inspections or remediations in that year. The final sample contains blood lead, inspection, and census data by census tract and year for 866 tracts over 7 years. Since the census is performed only once per decade, I do not have yearly data for census variables. I will discuss ways in which I deal with this obstacle in the next section of this chapter.

3.2 Measurement of Key Variables

The data contain an observation for each census tract for each year from 1997-2003. The dependent variable is the incidence of childhood blood lead poisoning, by census tract from 1997 to 2003 in Chicago, Illinois in 2003. The variable is calculated as the number of children under six years of age living in a census tract testing positive for lead poisoning divided by the total number of children under six screened in that tract, in each year.⁷ For the purposes

⁶Although, as noted previously, some tract/year observations are suppressed due to confidentiality concerns.

⁷Lead screens are mandatory for all children at various ages in Chicago. Therefore, the sample of screens should be representative of all children in Chicago. However, there is undoubtedly some level of targeted screening, which would bias the EBL rate upward. The rate of screening in Chicago is high enough that this

of this paper, a child testing at or above $10 \mu\text{g}/\text{dL}$ is considered lead poisoned. While there are different definitions of lead poisoning in children, $10 \mu\text{g}/\text{dL}$ is the most widely accepted standard (CDC, 2000). Table 1 summarizes the calculation of key variables. The mean across all tracts and all years is 12.72% (Table 2). As expected, there is a marked downward trend in the incidence of EBLs by year.

The remediation variable is calculated as the number of addresses remediated at least once in a census tract up to or in the year of the observation divided by the number of pre-1978 housing units in the tract.⁸ This metric, in effect, measures the healthiness of the housing stock in a census tract at a point in time. I also calculate the variable as the number of addresses remediated at least once up to but not including the current year divided by the number of pre-1978 housing units in the tract. This metric is, in a sense, a lagged version of the former remediation variable. It measures the “healthiness” of the housing stock at the beginning of the given year rather than at the end. The mean value for the standard and lagged remediation variables are 1.53% and 1.41%, respectively (Table 2). The lagged value is necessarily lower since it contains the same information as the standard metric minus the current year’s remediation data. It is likely that the remediation variables increase over time in each tract since the numerators are cumulative totals of remediations and the denominators, the number of pre-1978 housing units, are relatively stable.

The complete set of control variables and instruments, all by census tract, are: the percentage of housing units built prior to 1950, the percentage of occupied housing units that are occupied by the owner, the percentage of housing units occupied, median household income (year 2000 dollars), the proportion of the population that is black race, the proportion that is of Hispanic origin, and the percentage of families that receives public assistance. The

is not likely a problem. I also estimated all models using the total number of children rather than the number of screens as the denominator in the percentage of children with an EBL. The impact of remediations was only slightly lower. This is to be expected given that this metric is the absolute minimum of the percentage of children with an EBL.

⁸I estimate the annual number of pre-1978 housing units using the number of pre-1980 units from the 1990 census and the yearly number of demolitions of residential buildings. I assume that the number of housing units built in the 1970’s was evenly distributed across the decade. Thus, I subtract two years of housing data from the pre-1980 number to arrive at an estimate for pre-1978 housing units measured in 1990. I assume that any residential building demolished in Chicago prior to 2004 was constructed prior to 1978. Thus each demolition will decrease the number of pre-1978 housing units by one. Since there are likely to be multiple housing units in many of the demolished buildings, this metric overestimates the number of pre-1978 housing units per year.

yearly values of all census variables, with the exception of the percentage of housing units built prior to 1950⁹, are estimated with the assumption that the variables increase or decrease at a constant linear rate between the 1990 and 2000 censuses continuing until 2003.

I constructed a variable that approximates the risk of exposure in a census tract and year (i.e. a way of weighting areas by the potential total lead exposure to children). The metric denotes whether a tract is in the top 25% of tracts with respect to both the number of children in poverty and the number of housing units built prior to 1950. The risk variable measures whether there is likely to be a high level of poorly-maintained, older housing in an area.

Table 3 reports the incidence of EBLs by remediation and census variables. Without controlling for any other factors, tracts with higher levels of remediations also have higher rates of EBLs (Table 2). EBLs increase with the age of housing and the proportion of the population that is black race and decline with rates of owner occupancy and median income. It is clear that the rate of lead poisoning has declined over time across all variables (Table 3). In general, the relationships between quartiles persist in all years. Furthermore, the effect of time has a consistent impact for all quartiles within a variable. For example, the pattern of decline in EBLs seen in the first quartile for black race is very similar to the decline seen in the fourth quartile.

4 Methods

4.1 Conceptual Framework

I am most interested in the sign and magnitude of the coefficient on the remediation variable. Given that remediations have been proven to reduce BLLs in children, a healthier housing stock should, all things considered, lead to a lower incidence of childhood lead poisoning. The treatment of lead hazards in the home reduces the potential exposure both to children currently living in the home and to those that will live there in the future. Thus, a strong

⁹This variable is calculated in a similar fashion to the number of pre-1978 housing units. I assume that each residential demolition is in a building built before 1950. I begin with the number of pre-1950 housing units from the 1990 census. For each year, I subtract the number of demolitions from the numerator and denominator to arrive at the yearly percentage of units built prior to 1950.

history of lead remediation should decrease childhood lead poisoning. Remediation reduces the likelihood that these children will have elevated blood lead levels and thus reduces the overall incidence of the disease in that area. If there is a spillover effect (i.e., if a single remediation prevents lead poisoning in more than one child, then I should also observe this in the results). I am also interested in how the impact of remediation varies over time. Because lead safe work practices and lead dust control have improved over time, remediations in later years may be more effective.

The list of covariates was chosen because they provide evidence for the age and condition of the housing stock in the census tracts. Lead exposure tends to be greatest in older, poorly-maintained housing. The percentage of units built prior to 1950 is an estimate of the percentage of units in a census tract that contain lead paint. The percentages of units that are owner occupied, families receiving public assistance, and median household income are related to the condition of the housing stock. Controlling for all other risk factors, older housing and the percentage of families receiving public assistance should have a positive relationship with EBLs, while owner occupancy and median income should have an inverse relationship.

The race and Hispanic origin variables were included because past studies attempting to predict lead poisoning in children have shown that areas with high proportions of black and Hispanic residents tend to have higher incidences of lead poisoning (Melman et al., 1998; Pirkle et al., 1998). The association with black race should subside once the age and condition of housing are properly controlled for since there is no medical or theoretical basis for black children to have higher EBLs. In contrast, Hispanic origin may retain a significant positive relationship with EBLs since Hispanic children are much more likely to be exposed to lead from cultural sources such as pottery and folk remedies.

I also estimate the different impact of remediations among populations sensitive to exposure to lead. Areas with a greater percentage of young children living in older housing will be more likely to react favorably to remediations. Similarly, it is worthwhile to investigate whether remediations have a greater impact in areas with a high proportion of black residents, a group that despite the nationwide decreases in BLLs still maintains a relatively high rate of poisoning among children. I estimate the differential impacts across various subsections of

the population, by interacting the risk and race variables with the measures of the “health” of the housing stock. If remediations are effectively targeted to reach these populations, they may have a greater impact on the reduction of EBLs in high-risk tracts. I also include time interactions with the race and risk variables to determine whether the impact of these factors on lead lead poisoning have changed over time.

4.2 Panel Analysis

The basic specification for the incidence of EBLs in a census tract is:

$$EBL_{it} = \beta_0 + \beta_1 R_{it} + \beta_2' r_{it} + \beta_3' X_{it} + \beta_4' Y_{it} + \epsilon_{it} \quad (1)$$

where EBL_{it} is the percentage of children screened in census tract i that tested positive for lead poisoning. The variable R_{it} is the measure of the health of the housing stock, calculated as the percentage of pre-1978 housing units remediated. The vector r_{it} is the remediation variable interacted with the race and risk variables as well as the year dummy variables. The vector X_{it} contains exogenous covariates, which may include the percentage of housing units built before 1950, the proportions of the population that are black race and Hispanic origin, the percentage of housing units that are occupied, the percentage of occupied housing units that are owner occupied, the percentage of the population receiving public assistance, median household income, and various interactions with year dummy variables. Y_{it} is a vector of year dummy variables.

In the first specification (Model 1) I regress the EBL outcome on the percentage of housing units remediated, a set of covariates, and year dummy variables. Inclusion of the year dummies controls for any unobserved time effect that affects all census tracts. For instance, the effect of the ban in gasoline and the subsequent reduction in the amount of ambient lead over time would be picked up by the year dummies.

A potential source of unobserved heterogeneity could lead to biased parameter estimates of the remediation variable. Per conversations with CDPH staff, some areas in Chicago are “problem” regions (i.e. they have a high level of lead activity whether it be identified cases of lead poisoning or remediations). Thus, census tracts with high levels of

EBLs will also tend to have high levels of remediation. The differential employment of lead prevention resources in these areas equates to endogenous program placement. A failure to account for this endogeneity, will cause the estimates for the impact of remediations on EBLs to be biased upwards.

In order to account for “problem” tracts, I include census tract fixed effects (Model 2). I replace the dependent and explanatory variables with deviations of individual observations in a census tract from the mean for that tract to avoid introducing N-1 additional parameters. The census tract fixed effects are differenced out in the process. In order for these OLS estimates to be unbiased there must be no correlation between the differenced right hand side variables and the error term. The model is:

$$EBL_{it} - \overline{EBL}_i = \gamma_1(R_{it} - \overline{R}_i) + \gamma_2'(r_{it} - \overline{r}_i) + \gamma_3'(Y_{it} - \overline{Y}_i) + \gamma_4'(X_{it} - \overline{X}_i) + (\epsilon_{it} - \overline{\epsilon}_i) \quad (2)$$

A potential problem with the first two specifications is that the percentage of units remediated is potentially endogenous and determined simultaneously with the dependent variable. This is the case if remediations are the result of a positive blood lead test. If so, remediations are not a source of exogenous variation and in fact, blood lead tests and remediations are highly correlated. As mentioned earlier, this problem of reverse causality will cause OLS estimates of the impact of remediations to be biased upwards. Fortunately, not all remediations are the result of a positive blood lead test. It was noted in the background section of the paper that remediations are also ordered in all federally-assisted housing units built before 1978 and can be ordered in any unit built before 1978 with identified lead hazards. The CDPH does not record the impetus for any lead inspection or remediation and thus one is left to speculate what percentage of the units were remediated because of a positive blood lead screen. However, given any reverse causality between EBLs and remediations, the endogeneity problem remains and must be addressed.

The way in which the remediation variables are constructed should alleviate the endogeneity problem. Reverse causality is only a problem if the remediation occurs after a positive blood lead test (i.e. the inspection is ordered because a poisoned child is living there). The remediation metrics in this paper measure a history of lead poisoning in a given

area. The history includes remediations from past years and the current year. I measure the impact of this history on the current incidence of lead poisoning. Since current cases of lead poisoning cannot drive past remediations, the problem of reverse causality should be minor.

To further purge the analysis of reverse causality, I use the lagged remediation variable that captures only those remediations that occurred in years prior to the year in question (Model 3). For instance, in 2000, the dependent variable is the number of EBLs in 2000 divided by the number of children screened in 2000. The lagged remediation variable is the percentage of addresses remediated up to the year 2000. As mentioned above, since EBLs in 2000 most likely do not drive remediations in years prior to 2000, there is no reverse causality. Thus, I am estimating the impact of remediations in years past on the incidence of EBLs in the current year. The specification is identical to equation 2 with the exception that the remediation variable (including interaction terms) is measured at the beginning of year t .

5 Results

Using Model 1, I first include no interactions of the remediation variable with other variables (Model 1a) and then interact it with race, risk, and time (Model 1b). In Model 1a, the percentage of units remediated has a large positive impact on the incidence of EBLs (Table 4). A one percentage point increase in the percent of units remediated is associated with a 2.39 percentage point increase in the proportion of lead screens that uncovered an EBL. Interactions between the remediation variable and time in Model 1b indicate that the positive effect is much more pronounced in the earlier years. The total impact of remediations in 2003 is nearly zero. The coefficients for the remediation variables in both Models 1a and 1b are precisely measured at the 1% level. The signs of the remediation effects are troubling since increased remediation should reduce the incidence of EBLs. This result lends evidence to the theory that this remediation variable is endogenous and thus, the OLS estimate is biased upwards.

The year dummies all have significant large negative impacts on the incidence of childhood lead poisoning. Furthermore, the estimates increase in magnitude, thus lead poisoning

is decreasing in all tracts over time. The year dummies pick up the unobserved homogenous effects of time. For instance, the amount of lead dust deposited from lead gasoline use is consistent over an area with uniform vehicle traffic. This level of exposure declines over time at a relatively constant rate in all census tracts and thus, is picked up in the coefficients on the year dummy variables.

The effects of other explanatory variables are as expected. The percentage of owner occupied housing and the median household income in a census tract have a negative impact on the incidence of EBLs. High owner occupancy and median income are associated with a housing stock in good condition and thus, a low likelihood of exposure to lead hazards. The percentage of pre-1950 housing had a positive impact on the percent of EBLs. The result is not surprising, as the primary source of lead exposure is lead-based paint in older housing units. As in other lead studies, the proportion of the population of black race also has a positive impact of the incidence of EBLs.

Once I account for the census tract fixed effects (Model 2), the impact of remediations on the incidence on EBLs is large and negative (Table 5). A one-tenth percentage point increase in the difference between the percent remediated in a given year (a reasonable expected change given the yearly changes in remediations observed in Table 2) and the average percent remediated for all years in a census tract is associated with over a five-tenths percentage point reduction in the difference between the incidence of EBLs in a given year and the average incidence for all years. This finding is analogous to saying that a one-tenth percent increase in the percentage of units remediated in a year would lead to a five-tenths percent decrease in the incidence of EBLs in that census tract. Citywide, this is equivalent to 1.75 fewer cases of lead poisoning per additional remediation. The results are robust to the different remediation variables and to alternative specifications that include various sets of control variables. The reduction in EBLs from remediations is consistently between five to six-tenths of a percent.

In Model 3, the impact of the lagged remediation variable is several standard deviations greater than the original metric (Table 6). A one-tenth percentage point increase in remediation is associated with almost a six-tenths percentage point increase in EBLs (Model 3a). The total effect of lagged remediation is also slightly greater when comparing the mod-

els with interactions (Models 2b and 3b). This lends some evidence that there is a degree of reverse causality in the remediations included in the first metric. While the results with and without the lagged remediation variable are different, when translated into the number of averted cases of lead poisoning, the difference is less than one hundred out of several thousand total cases.

Remediations have a greater negative impact on EBLs over time. When I add remediation/time interactions the effect of remediation in 1997 is not significantly different than zero in Models 2b and 3b. However, remediations in subsequent years have a negative and increasing impact on EBLs over time.

With census tract fixed effects, the year impacts are still significant and positive, however, they are smaller in magnitude. It appears that the year effects in Model 1 were capturing some of the census tract effects that are accounted for in the fixed effects model. Lastly, black race is no longer a powerful predictor of EBLs. It is likely that black race was proxying for “problem tract” effects in the earlier models. The impact of race and to some extent the risk variable (measured as tracts with high levels of older housing and children in poverty) play a declining role in the positive prediction of EBLs over time. In particular, the positive relationship between race and EBLs observed in 1997 (although imprecisely measured) disappears over time. The reduction cannot be explained by remediations as they have no greater impact in areas with a high proportion of black residents nor in “high-risk” areas.

6 Cost-Benefit Analysis

Because primary prevention is really the only way to prevent the devastating health effects that lead poisoning causes in children, it seems clear that funds and efforts should be devoted towards eliminating exposure rather than treating children once they are sick. However, the rate of childhood lead poisoning is declining by several percentage points each year without the contributions of lead remediations. It is possible that the cost of remediations outweigh the benefits of the associated reduction in lead poisoned children. In this section I estimate the benefits of improving the housing stock and compare them with the cost of the associated

remediations.

6.1 Benefits

The benefits are the improved health and reductions in curative care costs for those children who would have been lead poisoned if not for the remediations. Figure 1 shows the basic pathway from increased remediations to the resulting benefits. The impact of increased remediations on EBLs is represented by arrow 1. Arrow 2a is the effect of reduced BLLs on cognitive ability. I estimate the decline in curative care costs as the average cost of medical treatment for childhood lead poisoning multiplied by the number of cases prevented (arrow 2b). Lead exposure in children has also been linked to behavioral disorders and criminal activity, primarily due to the damaging neurological effects of lead (arrow 2c). Unfortunately, it is difficult to estimate the proportion of crime and delinquent behavior attributable to lead. Therefore, I note that there are potential benefits to society in this area from reduced EBLs, but I do not include estimates of such benefits in the analysis.

6.1.1 Effect on Earnings

One way to measure the health gains of the decline in lead poisoning is the prevention of declines in cognitive ability (arrow 2a). There is a well-developed literature that estimates levels of cognitive decline from lead as reductions in childhood IQ (Table 7). Schwartz (1994) and Salkever (1995) quantified the benefit of improved IQ as the increase in expected lifetime earnings (arrow 3). The increase in earnings potential is the primary benefit addressed in this paper. I incorporate the results of the two studies in my benefits estimation with several caveats discussed below.

The earnings benefits of increased remediations can be interpreted as a combination of arrows 1, 2a, and 3 in Figure 1. Quantifying these benefits is problematic for two reasons: 1) It is difficult to measure the benefit of an additional IQ point in dollars (arrow 3) and 2) I measure the impact of reductions in the percentage and therefore the number of lead poisoned children (arrow 1) while the cognitive effects literature generally measures the impact of the average lifetime BLL on IQ. I cannot determine the level of the reduction but simply that it has dropped below the 10 $\mu\text{g}/\text{dL}$ threshold (arrow 2a).

There is a debate in the recent literature as to the magnitude and importance of the impact of BLLs on cognitive ability. The majority of studies have found that lead exposure is associated with a reduction in childhood IQ. There are a few studies that point out that the cognitive declines associated with lead are very small compared to those of other proven factors (Heben, 2001; Kaufman, 2001a; Kaufman, 2001b). They also raise questions related to the methods of analyses that have found significant declines in IQ from lead exposure. However, even the authors of these papers note that there are likely impacts on cognitive ability at low levels of lead exposure.

The range of the magnitude of the effects varies from a 0.46 point reduction associated with a 1 $\mu\text{g}/\text{dL}$ increase in BLL to a 9.6 point reduction from a one log point increase in BLL (Table 7). In a metaanalysis by Schwartz (1994a), the author estimated that a 1 $\mu\text{g}/\text{dL}$ increase in BLL was associated with a 0.245 point decline in IQ across all BLLs. Canfeld et al. (2003) found that a 1 $\mu\text{g}/\text{dL}$ increase in BLL was associated with a 0.46 decrease in IQ. More importantly, they found that at BLLs below 10 $\mu\text{g}/\text{dL}$, a 1 $\mu\text{g}/\text{dL}$ increase in BLL was associated with a 0.74 decrease in IQ. A host of studies confirm that there are indeed significant detrimental effects on IQ associated with BLLs below 10 $\mu\text{g}/\text{dL}$ and that in fact, the effects are greater at lower BLLs (Bellinger and Needleman, 2003; Canfeld et al., 2003; Lidsky and Schneider, 2003).

While I have a reliable estimate of the number of lead poisoning cases prevented, I do not observe the magnitude of the decline in BLL. At best, I can assert that in each case, the BLL declines from above 10 $\mu\text{g}/\text{dL}$ to below 10 $\mu\text{g}/\text{dL}$. In an individual child level study, the authors found that the mean BLL of children moving back into remediated homes declined from 11.0 to 9.3 (-1.7) after six months and from 11.0 to 8.2 (-2.8) after twelve months (Galke et al., 2001). They did not investigate whether BLLs continued to decline after twelve months. These are useful estimates if I assume that children are already lead poisoned. However, a major assertion in this paper is that a fraction of children moving or born into remediated homes will be spared from lead exposure altogether. Thus, it is reasonable to expect that some children will have BLLs of 0 $\mu\text{g}/\text{dL}$ rather than 10 $\mu\text{g}/\text{dL}$ or even higher.

I incorporate all this information to estimate bounds on the increase in IQ from re-

ductions in childhood lead poisoning (Table 8). For the minimum, I use Schwartz's estimate of a 0.245 point increase in IQ for each 1 $\mu\text{g}/\text{dL}$ decline in BLL across all BLLs. This is a reasonable minimum since preventing lead poisoning means reducing BLLs below 10 $\mu\text{g}/\text{dL}$ and recent studies have shown that the impact on IQ is greater below 10 $\mu\text{g}/\text{dL}$. I also assume a minimum decline of 2.8 $\mu\text{g}/\text{dL}$ (from 11.0 to 8.2) found in Galke et al. (2001) after twelve months. This is a reasonable minimum reduction in BLL for reasons discussed above. Thus, the lower bound on the increase in IQ associated with the prevention of a single case of childhood lead poisoning is 0.69 ($0.245 * 2.8$ - row 4, column1 Table 8).

I use the 0.74 IQ point increase associated with a 1 $\mu\text{g}/\text{dL}$ decline at lower BLLs found in Canfeld et al. (2003) as a conservative maximum rise in IQ. This is a reasonable estimate given that any prevented case of lead poisoning will result in a BLL below 10 $\mu\text{g}/\text{dL}$. I assume a conservative maximum 5 $\mu\text{g}/\text{dL}$ drop in BLL for each case of lead poisoning averted. The 5 $\mu\text{g}/\text{dL}$ decline is an average change across prevented cases of lead poisoning. It does not assume a 5 $\mu\text{g}/\text{dL}$ decline for each case averted. Some individuals will observe a small decrease while others will see a large drop in BLL. Given that Galke et al. (2001) found that mean BLLs of poisoned children drop 2.8 $\mu\text{g}/\text{dL}$ after only twelve months and that some children will be spared BLLs 10 $\mu\text{g}/\text{dL}$ or greater, this is a reasonable estimate. Using these two estimates, the upper bound on the increase in IQ associated with the prevention of a single case of lead poisoning is 3.7 ($0.74 * 5.0$ - row 4, column 2 Table 8).

Several recent studies have sought to estimate the impact of IQ on earnings potential (Schwartz, 1994; Salkever,1995). In general, they estimate the total effect of IQ as the direct effect on earnings through lower cognitive ability, and the indirect effects through schooling and lower rates of participation in the labor force. They argue that IQ loss affects schooling and therefore expected wages and the level of participation (i.e., hours worked). Schwartz found that a one point increase in IQ was associated with a combined 1.76% increase in expected lifetime earnings. Salkever found a 1.931% increase for males and a 3.225% increase for females. Both studies estimate a linear relationship between IQ and earnings

These estimates are problematic for several reasons. First it is unlikely that IQ impacts earnings equally across all potential workers. This casts doubt on their assumption of a linear relationship between IQ and earnings, particularly when looking at such a small incremental

change (one IQ point). Second, the importance of a one or two point change in IQ for an individual is questionable in relation to the number of other traits that impact earnings (e.g., socioeconomic status and family characteristics). However, when considered across the entire population, a several point change seems rather significant, particularly in the tails of the distribution where a small change can move individuals out of the disabled category and conversely, individuals into the genius classification. It is also important to consider that a one point change across the population is the average result. Some individuals will have greater increases in IQ while others will observe very little change. I calculate the increased expected lifetime earnings using the estimates in both Schwartz (1994) and Salkever (1995) with these caveats in mind.

I employ the basic methodology developed in Schwartz (1994) to calculate the discounted net present value of the expected lifetime earnings of a five-year-old child for males and females. I chose to estimate the value for a five-year-old because I am estimating the impact of IQ on earnings and IQ is generally measured in school age children.¹⁰ I use the distribution of wages for males and females at each age measured in 2000 and assume that each child will work from 15 to 65 years of age. Based on several recent studies that have estimated expected lifetime earnings, I assume a 1% annual increase in wages for both males and females (Landrigan, 2002; Muir, 2001; Salkever, 1995; Schwartz, 1994;). I estimate net present value of earnings using a 3% discount rate. The discounted expected lifetime earnings for males and females are reported in rows 7 in Table 8. The average for males and females is reported in columns 1 and 2 in row 7.

6.1.2 Medical Care Expenditures

In a study by Kemper et al. (1998), the authors estimate the costs associated with each component of curative care for lead poisoned children. Korfmacher (2003) uses these costs and estimates the cost of curative care at various BLLs. The cost of care for BLLs between

¹⁰The expected lifetime earnings for children under five years of age will vary slightly according to the assumed annual increase in real wages (1%). The expected number of years worked is the same for all children, but the younger children will have slightly higher real wages at each age because of the increase in productivity. They will necessarily have larger undiscounted expected lifetime earnings. However, the discount rate (3%) is greater than the rise in productivity. Thus, the discounted net present value of expected earnings for younger children will be less than that for those five years of age.

10 and 19 $\mu\text{g}/\text{dL}$ is \$63.34 per child adjusted to 2000 dollars. This is the cost of basic follow-up care for lead poisoned children including screens, and in-home visits by nurses. Since I measure the decline in cases of lead poisoning, the appropriate cost savings should occur near the threshold for poisoning. The \$63.34 observed from 10 to 19 $\mu\text{g}/\text{dL}$ is a reasonable expectation for the curative costs saved for each prevented case of lead poisoning (row 9). This figure is a minimum estimate for medical care cost savings. Remediations reduce lead exposure for all BLLs, not only those near 10 $\mu\text{g}/\text{dL}$. I do not include the potentially large cost savings at higher BLLs such as reduced hospitalization and chelations.

6.2 Costs

It is relatively simple to identify the costs of improving a housing stock. The cost is simply the cost of the remediations necessary to treat lead hazards in homes. The prices of remediations are available over time and thus, the cost of improving a housing stock is fairly accurately represented by simply the cost of treating lead hazards in a given number of units.

Per conversations with CDPH staff, the average cost under their abatement grant programs is \$7,750 per housing unit (row 2 of Table 8).¹¹ The number of remediations necessary to increase the percentage of pre-1978 housing units treated by one-tenth of a percent is 2,081 (row 1 of Table 8). If the average cost of these remediations is \$7,750, then the total cost of improving remediations by one-tenth of a percent is \$16,128,000.

6.3 Benefits Versus Costs

Table 8 lists all the estimated costs and benefits discussed above. I simulate the estimated number of lead poisoning cases averted from a one-tenth percentage increase in remediations using the results from Model 3a in Table 6 (arrow 1, Figure 1).¹² The additional 2,081 remediations lead to 3,506 fewer cases of childhood lead poisoning (row 3). The result is precisely measured at a 1% significance level. Row 4 lists the lower and upper bounds on the increase in IQ from a prevented case of childhood lead poisoning (arrow 2b). Row 5 reports

¹¹The median cost is \$6,795 per housing unit.

¹²I draw from the variance-covariance matrix assuming a normal distribution and then apply the draw to the vector of coefficients found using Model 3b. For each draw, I estimate the number of cases averted. I repeat this until the SD of the number of cases converges. The standard deviations for the number of cases and for the cost savings are reported in Table 8.

the estimated percentage increase in expected lifetime earnings from a one point increase in IQ taken from Schwartz (1994) and Salkever (1995). Assuming a constant linear relationship between IQ and earnings the percentage increase in lifetime earnings from a prevented case (row 6) is the rise in IQ (row 4) multiplied by the percentage increase from one additional IQ point (row 5). Incorporating the figures from Schwartz (1994), the lower and upper bounds are 1.21% and 6.51%, respectively. Using the figures from Salkever (1995), I find that the lower bound on the percentage changes in earnings is 1.33% for males and 2.23% for females. The upper bounds for males and females are 7.14% and 11.93%, respectively (row 4).

The discounted net present values for estimated lifetime earnings for five year-old boys and girls are \$519,631 and \$881,027, respectively (U.S. BLS, 1999). I use the average value, \$700,329, in the first two columns since I do not have earnings estimated separately for males and females. I multiply the percentage change in earnings (row 6) by the expected earnings (row 7) to calculate the average change in expected lifetime earnings from a prevented case of childhood lead poisoning (row 8). The estimates range from roughly \$8,000 to over \$60,000 per case. Row 10 contains the total increase in earnings from all averted cases of lead poisoning from a one-tenths percent increase in remediations (row 3 * row 8).¹³

Row 9 lists the average cost of curative care for a case of lead poisoning with a BLL between 10 and 19 $\mu\text{g}/\text{dL}$, \$63.34. I multiply this estimate by the total number of cases prevented (row 3) to arrive at the total medical care cost savings from the increase in remediations (row 11). The total estimated benefits from the increase in remediations is the increase in expected lifetime earnings (row 10) plus the costs savings from reduced curative medical care (row 11).

The total savings from a one-tenth percentage point increase in the percentage of housing units remediated ranges from \$14 million to over \$200 million. The estimates incorporating the figures from Salkever (1995) are within two standard errors of the estimates using the Schwartz (1994) figures. The differences between the two benefit/cost ratios are driven by the higher estimates of earnings increases from a reduction in IQ in Salkever (1995), particularly among females.

¹³In columns 3 and 4, I assume that half of the prevented cases are in males and half in females. The change in earnings is thus, $0.5(\text{row } 1)(\text{row } 6, \text{ males}) + 0.5(\text{row } 1)(\text{row } 6, \text{ females})$.

7 Discussion

Once I account for the unobserved effects of census tracts and time, the percentage of addresses remediated in a census tract has an inverse relationship with the incidence of EBLs in that area. Because OLS will provide estimates that are biased upwards towards zero for the impact of remediations, any coefficients can be interpreted as lower bounds (in magnitude) on the range of effects. Furthermore, the magnitude of the impact is feasible given the summary statistics (Table 2). A one-tenth percent increase in the percent of addresses remediated is well within the range of potential changes. The resulting six tenths percent decrease in the incidence is also feasible given the dramatic declines in EBLs over time.

Applying these results citywide, roughly 1.75 cases of lead poisoning are prevented for each housing unit remediated. Because this ratio is greater than 1:1, there is some spillover effect of remediations. This spillover effect can be attributed to several sources. Multiple children living in a housing unit should all benefit from an effective remediation. Thus, it is certainly feasible that all young siblings will observe a decline in BLLs. Although less likely to produce this large of a spillover effect, it is also possible that children visiting the home will have lower BLLs. These spillover effects are potentially larger if the lead hazards are on the outside of the home, primarily in the form of lead contaminated soil. The last possible explanation is that children that were once poisoned receive multiple passing screens. This would bias the percentage of EBLs downward and the impact of remediation upwards. This is unlikely as children with multiple screens are more likely to have positive than negative screens since follow-up is ordered only in poisoned children.

Remediation was more effective in reducing childhood lead poisoning in later years. It has been noted that the requirements for proper lead remediation have become more stringent over time. Most importantly, the regulations now require that lead dust be controlled. Additionally, anyone performing a lead remediation must be certified in lead safe work practices. These strict guidelines may be driving the increased efficacy of remediations over time. This also means that the expiration of older remediations are not having a meaningful negative impact on the “healthiness” of the housing stock. It is likely that once a housing unit has been remediated, parents and landlords work to ensure that the units remain free

of lead hazards.

There is, perhaps, a time-varying factor in tracts that differentially impacts areas deemed to be “high risk” and with a high proportion of black residents. Since the CDPH fully knows which areas are high risk, they may have increased intervention efforts over time in these areas. It is reasonable to assume that this increased awareness over time would decrease the risk of lead exposure in these tracts. A differential impact of awareness would explain the declining importance of race as a predictor of childhood lead poisoning over time (Tables 5 and 6).

Across all the models, the magnitudes of the impacts of the covariates were very small. Even the percent of pre-1978 housing units, thought to be a very accurate predictor of lead poisoning, had little impact on the dependent variable. This finding provides evidence that the condition of the housing is far more important than the age. The age variable was most likely acting as a proxy for condition in other lead studies that found large, significant effects.

The costs savings in Chicago from a small increase in remediation is potentially several hundred million dollars. While this estimate may seem high, it is a reasonable figure for Chicago given the nationwide estimates reported in the literature. Several studies have estimated that the benefit of small reductions in lead exposure nationwide could be tens of billions of dollars (Schwartz, 1994; Salkever, 1995; Landrigan et al., 2002). One study estimated that the benefit of reduced lead exposure since 1976 has been between \$100 and \$300 billion (Grosse et al., 2002). In addition, the benefits of remediation will continue to increase over time. A single remediation can potentially prevent lead exposure for all children living in that home in the future. Therefore, improving the “healthiness” of the housing stock benefits all future generations.

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Table 1: Glossary of key terms

Term	Definition
lead screen	A blood lead test to determine if a child has lead poisoning; a positive result is anything over 10 $\mu\text{g}/\text{dL}$
lead inspection	Any of three types of lead inspection (inspection, risk assessment, lead hazard screen)
lead remediation	Any process by which lead hazards are addressed in a home whether it is a remediation, mitigation, or full abatement
<i>Key variables</i>	
incidence of lead poisoning	$[\# \text{ of children under age 6 with a positive test}] /$ $[\# \text{ of children under age 6 screened}]$, by tract and year
% remediations (3)	$[\# \text{ pre-78 units with at least 1 rem in or prior to current year}] /$ $[\# \text{ pre-78 units}]$, by tract and year
% rem lagged (4)	$[\# \text{ pre-78 units with at least 1 rem prior to current year}] /$ $[\# \text{ pre-78 units}]$, by tract and year

Table 2: Summary Means by Year

variable	total	1997	1998	1999
% EBLs	12.72 (13.03)	20.90 (16.85)	17.17 (14.53)	14.77 (13.04)
% remediated	1.53 (2.34)	1.10 (1.44)	1.20 (1.54)	1.37 (1.99)
% remediated (lagged)	1.41 (2.16)	1.07 (1.41)	1.11 (1.45)	1.21 (1.59)
% of units pre-1950	60.58 (24.11)	60.83 (24.03)	60.74 (24.06)	60.64 (24.10)
% black race	42.80 (43.46)	42.71 (43.56)	42.73 (43.49)	42.74 (43.45)
% hispanic origin	22.98 (28.46)	22.21 (27.43)	22.52 (27.78)	22.81 (28.16)
% on public assistance	9.39 (10.71)	12.41 (12.40)	11.48 (11.83)	10.42 (10.96)
% median HH income	45.52 (28.11)	41.67 (23.04)	42.95 (24.55)	44.23 (26.12)
variable	2000	2001	2002	2003
% EBLs	12.80 (12.09)	10.04 (10.03)	7.90 (8.34)	5.64 (6.20)
% remediated	1.57 (2.49)	1.71 (2.60)	1.83 (2.70)	1.95 (3.01)
% remediated (lagged)	1.37 (1.99)	1.58 (2.50)	1.71 (2.61)	1.84 (2.93)
% of units pre-1950	60.56 (24.14)	60.49 (24.16)	60.43 (24.19)	60.40 (24.20)
% black race	42.75 (43.45)	42.80 (43.41)	42.86 (43.38)	43.02 (43.62)
% hispanic origin	23.00 (28.44)	23.29 (28.90)	23.48 (29.21)	23.67 (29.46)
% on public assistance	9.34 (10.27)	8.35 (9.78)	7.35 (9.39)	6.37 (8.46)
% median HH income	45.52 (27.73)	46.80 (29.39)	48.09 (31.09)	49.37 (32.81)

Note: Standard deviations are in parentheses.

Table 3: Mean %EBLs by the dependent, abatement, and control variables

Variable	1997	1998	1999	2000	2001	2002	2003
% abatement							
1st quartile	9.48	7.24	5.86	5.48	3.60	2.65	2.29
2nd quartile	12.15	9.23	7.98	5.72	4.70	3.52	2.16
3rd quartile	25.80	20.78	16.12	12.55	9.27	6.45	4.43
4th quartile	43.02	35.24	30.06	25.84	20.38	16.07	11.11
% abatement (lagged)							
1st quartile	9.53	7.32	6.04	5.43	3.69	2.61	2.29
2nd quartile	12.08	9.57	7.95	6.26	4.64	3.66	2.33
3rd quartile	25.30	20.67	16.93	13.02	9.13	6.37	4.07
4th quartile	42.29	35.26	30.08	25.87	20.34	15.84	11.03
% pre-1950							
1st quartile	15.76	13.71	11.85	10.14	7.91	5.74	4.20
2nd quartile	26.67	22.41	19.19	16.84	13.92	11.12	7.79
3rd quartile	25.43	20.58	17.15	15.02	11.44	9.68	6.95
4th quartile	15.40	11.84	10.66	8.93	6.70	4.95	3.57
% black race							
1st quartile	8.71	7.44	5.92	4.75	3.75	2.71	2.00
2nd quartile	12.42	8.72	8.41	6.00	4.86	3.60	2.92
3rd quartile	24.60	20.44	17.57	16.13	12.50	10.32	6.42
4th quartile	36.43	31.01	26.66	24.27	19.43	15.57	11.10
% Hispanic origin							
1st quartile	34.42	28.83	24.96	22.34	17.73	13.66	10.14
2nd quartile	19.37	16.56	14.24	13.23	10.44	8.7	5.96
3rd quartile	11.29	9.05	8.09	6.69	5.36	3.89	2.80
4th quartile	16.80	13.01	10.95	8.65	6.75	5.56	3.97
% receiving public assistance							
1st quartile	6.61	5.49	5.15	4.52	4.35	3.57	2.68
2nd quartile	8.81	7.64	7.41	6.64	5.98	4.94	4.48
3rd quartile	18.87	16.75	15.63	14.80	12.56	11.16	8.26
4th quartile	36.35	30.58	26.46	24.32	19.18	15.43	10.74
median HH income							
1st quartile	34.96	28.74	24.27	22.31	17.51	14.26	9.51
2nd quartile	24.06	20.30	17.62	16.30	13.28	10.93	8.43
3rd quartile	12.41	10.83	10.11	8.6	7.29	5.51	3.97
4th quartile	7.57	6.40	5.95	4.65	3.12	2.28	2.02

Note: The 1st quartile is the lowest 25% of values for the given variable, the 4th is the highest 25%.

Table 4: Results from Model 1 (OLS)

Variable	Model					
	1a			1b		
Intercept	8.888	(1.468)	***	10.068	(1.295)	***
% addresses remediated	2.387	(0.070)	***	5.770	(0.201)	***
% pre-1950	0.068	(0.005)	***	0.057	(0.004)	***
% black race	0.131	(0.005)	***	0.183	(0.007)	***
% hispanic origin	0.013	(0.006)	**	-0.003	(0.005)	
% owner occupied	0.010	(0.006)	*	0.005	(0.005)	
% occupied	-0.038	(0.015)	**	-0.078	(0.013)	***
% receiving public assistance	0.138	(0.015)	***	0.010	(0.014)	*
median HH income	0.013	(0.005)	**	-0.010	(0.005)	**
1998 dummy	-3.836	(0.377)	***	-1.606	(0.493)	***
1999 dummy	-6.458	(0.379)	***	-2.222	(0.490)	***
2000 dummy	-8.711	(0.381)	***	-3.425	(0.487)	***
2001 dummy	-11.654	(0.384)	***	-4.093	(0.486)	***
2002 dummy	-13.989	(0.389)	***	-4.675	(0.486)	***
2003 dummy	-16.416	(0.394)	***	-4.771	(0.487)	***
% remediated * black race				0.289	(0.084)	***
% remediated * high risk variable				0.762	(0.187)	***
% remediated * 1998				-1.391	(0.256)	***
% remediated * 1999				-2.238	(0.246)	***
% remediated * 2000				-3.097	(0.236)	***
% remediated * 2001				-3.649	(0.231)	***
% remediated * 2002				-4.210	(0.226)	***
% remediated * 2003				-4.787	(0.222)	***
% black race * 1998				-0.021	(0.009)	**
% black race * 1999				-0.050	(0.009)	***
% black race * 2000				-0.048	(0.009)	***
% black race * 2001				-0.084	(0.009)	***
% black race * 2002				-0.105	(0.009)	***
% black race * 2003				-0.134	(0.009)	***
% high risk * 1998				-0.306	(0.678)	
% high risk * 1999				-0.635	(0.742)	
% high risk * 2000				-1.703	(0.822)	**
% high risk * 2001				-1.606	(0.913)	*
% high risk * 2002				-0.821	(0.968)	
% high risk * 2003				-1.239	(1.049)	

*** indicates significance at the 1% level; **5% level; *10% level.

Table 5: Results from Model 2 - Census Tract Fixed Effects

Variable	Model					
	2a		2b			
Intercept	1.157	(26.741)		30.740	(26.127)	
% addresses remediated	-5.355	(0.315)	***	0.693	(0.487)	
% pre-1950	0.319	(0.413)		-0.084	(0.403)	
% black race	0.028	(0.060)		0.080	(0.062)	
% Hispanic origin	0.216	(0.032)	***	0.033	(0.026)	
% receiving public assistance	0.321	(0.045)	***	0.061	(0.026)	**
% owner occupied	-0.133	(0.087)	*	-0.134	(0.080)	*
% occupied	-0.021	(0.069)		-0.032	(0.062)	
median HH income	0.078	(0.030)	**	-0.051	(0.029)	*
1998 dummy	-2.965	(0.309)	***	-1.512	(0.402)	***
1999 dummy	-4.272	(0.328)	***	-1.838	(0.389)	***
2000 dummy	-5.066	(0.353)	***	-3.001	(0.393)	***
2001 dummy	-6.744	(0.401)	***	-3.453	(0.408)	***
2002 dummy	-7.987	(0.446)	***	-3.920	(0.426)	***
2003 dummy	-9.392	(0.491)	***	-3.860	(0.463)	***
% remediated * black race				0.010	(0.135)	
% remediated * risk variable				-0.035	(0.226)	
% remediated * 1998				-1.169	(0.261)	***
% remediated * 1999				-1.724	(0.255)	***
% remediated * 2000				-2.153	(0.249)	***
% remediated * 2001				-2.522	(0.265)	***
% remediated * 2002				-2.829	(0.262)	***
% remediated * 2003				-3.200	(0.289)	***
% black race * 1998				-0.017	(0.008)	**
% black race * 1999				-0.041	(0.009)	***
% black race * 2000				-0.037	(0.008)	***
% black race * 2001				-0.066	(0.008)	***
% black race * 2002				-0.086	(0.009)	***
% black race * 2003				-0.112	(0.009)	***
% high risk * 1998				0.149	(0.405)	
% high risk * 1999				-0.101	(0.437)	
% high risk * 2000				-0.735	(0.472)	*
% high risk * 2001				-0.625	(0.521)	
% high risk * 2002				-0.096	(0.579)	
% high risk * 2003				-0.301	(0.649)	

*** indicates significance at the 1% level; **5% level; *10% level.

Table 6: Results from Model 3 - Census Tract Fixed Effects with Lagged Remediation Variable

Variable	Model					
	3a		3b			
Intercept	1.197	(25.436)		33.892	(25.739)	
% addresses remediated	-5.920	(0.294)	***	0.145	(0.484)	
% pre-1950	0.366	(0.391)		-0.109	(0.397)	
% black race	0.018	(0.060)		0.071	(0.062)	
% Hispanic origin	0.200	(0.030)	***	0.030	(0.026)	
% receiving public assistance	0.325	(0.044)	***	0.040	(0.040)	
% owner occupied	-0.120	(0.085)		-0.131	(0.080)	*
% occupied	-0.042	(0.069)		-0.038	(0.062)	
median HH income	0.071	(0.030)	**	-0.054	(0.029)	*
1998 dummy	-3.281	(0.309)	***	-1.510	(0.400)	***
1999 dummy	-4.793	(0.323)	***	-1.833	(0.387)	***
2000 dummy	-5.664	(0.336)	***	-2.979	(0.391)	***
2001 dummy	-6.953	(0.381)	***	-3.403	(0.403)	***
2002 dummy	-8.051	(0.425)	***	-3.886	(0.427)	***
2003 dummy	-9.298	(0.469)	***	-3.820	(0.463)	***
% remediated * black race				0.066	(0.144)	
% remediated * risk variable				-0.037	(0.240)	
% remediated * 1998				-1.215	(0.272)	***
% remediated * 1999				-1.790	(0.262)	***
% remediated * 2000				-2.204	(0.248)	***
% remediated * 2001				-2.518	(0.266)	***
% remediated * 2002				-2.782	(0.263)	***
% remediated * 2003				-3.101	((0.292)	***
% black race * 1998				-0.017	(0.008)	**
% black race * 1999				-0.041	(0.009)	***
% black race * 2000				-0.037	(0.008)	***
% black race * 2001				-0.066	(0.008)	***
% black race * 2002				-0.085	(0.008)	***
% black race * 2003				-0.112	(0.009)	***
% high risk * 1998				0.164	(0.399)	
% high risk * 1999				-0.078	(0.427)	
% high risk * 2000				-0.765	(0.448)	*
% high risk * 2001				-0.560	(0.511)	
% high risk * 2002				0.162	(0.566)	
% high risk * 2003				-0.328	(0.650)	

*** indicates significance at the 1% level; **5% level; *10% level.

Table 7: Summary of BLL and IQ loss literature

Citation	BLL increase	IQ point decrease
Canfeld et al. 2003	10 mcg/dL increase in mean BLL	4.6
Canfeld et al. 2003	1 mcg/dL increase in mean BLL	0.46
Canfeld et al. 2003	1 mcg/dL inc in mean BLL for those never > 10 mcg/dL	0.74
Lanphear et al. 2005	2.4 to 10 mcg/dL	3.9
Lanphear et al. 2005	10 to 20 mcg/dL	1.9
Lanphear et al. 2005	20 to 30 mcg/dL	1.1
Schwartz 1994	10 to 20 mcg/dL	2.6
Tong 2000	2.7 fold increase (one natural log point)	2.6-9.6
Wasserman et al. 2000	50% rise in prenatal BLL	1.07
Wasserman et al. 2000	50% rise in postnatal relative to prenatal levels	2.82
Wasserman et al. 1997	10 to 30 mcg/dL	4.3

Note: Schwartz, 2002 is a metaanalysis.

Table 8: Benefits and costs associated with a one-tenth increase in the remediation variable

Event	Schwartz (1994)		Salkever (1995)	
	min	max	min	max
<i>Pre-Calculations</i>				
1. number of remediations required for one-tenth % increase	2,081	2,081	2,081	2,081
2. average cost of remediation in Chicago, IL	7,750	7,750	7,750	7,750
3. reduction in cases of lead poisoning (standard deviation)	3,506 (727)	3,506 (727)	3,506 (727)	3,506 (727)
4. increase in IQ points from averted case	0.69	3.70	0.69	3.70
5. % increase in expected lifetime earnings from 1 IQ point decline male	1.76	1.76		
female			1.93	1.93
6. % increase in earnings from one less case (row 4 × row 5) male			3.23	3.23
female	1.21	6.51	1.33	7.14
7. expected lifetime earnings (males and females) male			2.23	11.93
female	700,329	700,329		
8. change in exp lifetime earnings from one less case (row 4 × row 5)	8,473.98	45,591.42	881,027	881,027
9. average cost of curative care for case of lead poisoning	64.34	64.34	519,631	519,631
<i>Benefits in thousands</i>				
10. total change in earnings (row 3 × row 8)	29,710	159,844	11,652.72	62,448.88
11. total reduction in curative care costs (row 3 × row 9)	226	226	64.34	64.34
Total Benefits (row 10 + row 11)	29,935	160,069	41,080	219,171
<i>Costs in thousands</i>				
Total Costs in thousands (row 1 × row 2)	16,128	16,128	16,128	16,128
Difference between Benefits and Costs in thousands (standard deviation)	13,808 (6,207)	143,941 (33,192)	24,952 (8,518)	203,044 (45,447)

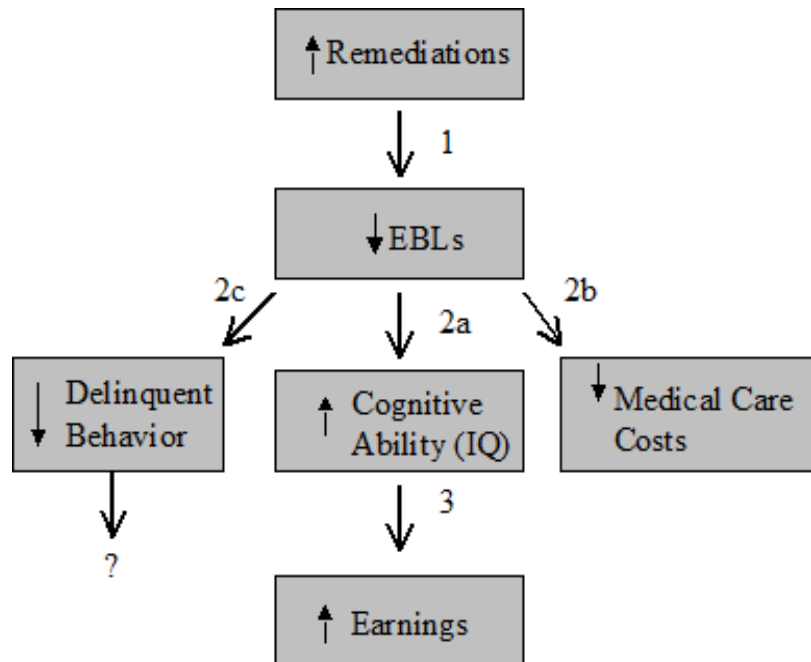


Figure 1: Benefit Pathway from Increased Remediation