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# Primary prevention and health outcomes: Treatment of residential lead-based paint hazards and the prevalence of childhood lead poisoning

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ABSTRACT

In order to gain a better understanding of the effects of an investment in primary prevention on health, I investigate the impact of treatment of lead-based paint hazards in housing units (the preventive action) on childhood lead poisoning (the health outcome) at the census tract level in Chicago, IL. I use the findings from the analysis to simulate and then weigh the costs of lead interventions against the potential benefits of reducing blood lead levels in children. Childhood lead poisoning presents an interesting case study of the potential of preventive care in reducing the prevalence 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 four-tenths percentage point reduction in the prevalence of childhood lead poisoning, an elasticity of roughly 0.5. Citywide, this is roughly 2.5 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 – are two to twenty times the estimated costs of the remediations.



## 1. Introduction

Childhood lead poisoning is the second most prevalent preventable disease (after asthma) in children in the United States (Centers for Disease Control and Prevention – CDC, 2005). In general, the average blood lead level (BLL) in children has been declining over the last three decades, down 90% since 1978 (Envrionmental Protection Agency – EPA, 2005). However, levels among low-income, urban children, particularly those living in older housing, remain high (Chicago Department of Public Health – 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 1970s 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 prevalence 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 the US. More specifically, I intend to estimate the impact of an investment in lead remediations, a non-medical approach to primary prevention, on the prevalence 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.

Rather than measure whether individual remediations prevent cases of lead poisoning, I investigate whether the aggregate remediation efforts in a given area (US census tracts) will reduce the prevalence of childhood lead poisoning there. I use the number of housing 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 investment in prevention increases and the health of the housing stock improves, children face a lower risk of exposure to lead and the prevalence of lead poisoning should decline.

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There are several aspects of childhood lead poisoning and its prevention that provide a useful case for studying preventive care and its impacts on health. First, there is a clear pathway with a short time horizon from exposure to illness in children (lead-based paint hazards in older, poorly-maintained housing units). Thus, it is possible to isolate the effect of prevention on a health outcome from other confounding factors. In addition, the health effects of lead are irreversible. The only effective way to combat the illness is to prevent it from occurring. Lastly the potential benefits of prevention are rather large compared to the costs of increased remediation.

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 US city (CDPH, 2004).

I observe each variable by year from 1997 to 2003 and by census tract. I begin by estimating a simple linear model of the prevalence of childhood lead poisoning controlling for various changes in census tract characteristics over the study period. 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 prevalence 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. 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 in homes because children living there have tested positive for lead poisoning) is another potential problem. Again, OLS estimators will tend to underestimate the true effects of remediation. 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 four-tenths percentage point decrease in the prevalence of childhood lead poisoning, an elasticity of roughly 0.5. Citywide, from 1997 to 2003, this effect translates to 2.5 cases averted for each additional housing unit treated. Furthermore, the negative impact of remediations on EBLs strengthens over time, suggesting that each remediation may prevent EBLs in more than one child over succeeding years. I also find that there was a sharp decline in the prevalence 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. Lastly, when controlling for various housing and sociodemographic characteristics in the panel analysis, the proportion of the population that is black, a significant risk factor in other studies, is no longer an important predictor of lead poisoning in children.

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 20:1. 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 when compared to other studies finding large nationwide benefits from 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 over the past century have been lead-based paint and leaded gasoline. They are responsible for nearly equal shares of the lead burden in the US, 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 US in the 1920s. The usage peaked in the early 1970s at over 2 million metric tons per year (Mielke and Reagan, 1998). However, due to the growing realization in the 1970s of the health effects of lead along with the inclusion of the catalytic converter to almost all vehicles sold in the US, the use of lead in gasoline began to decline.[1] The official phaseout began in 1973 and culminated on January 1, 1996, when the sale of leaded gasoline was banned in the US as part of the Clean Air Act.

Lead was commonly used as an additive in residential paint in the US from the late 19th century until 1978. Lead paint manufacturers voluntarily reduced the lead content of paint throughout the 1950s, although the levels were still high enough to produce a 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 US 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 US include pottery, cosmetics, and folk remedies brought to the US 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 US, 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 US 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

[1] Leaded gasoline is not compatible with catalytic converters, which were added to automobiles sold in the US in the 1970s to reduce air pollution.