Essays on Preventive Care and Children's Health

by
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ii

ABSTRACT

DAVID J. JONES: Essays on Preventive Care and Children's Health
(Under the direction of Donna B. Gilleskie)

In light of concerns over rising health care expenditures in the U.S., the need to develop low-cost methods of reducing health risks is more pressing than ever before. In many cases, effective preventive care can reduce the need for more expensive curative care later in life. If preventive care can be a sufficiently useful substitute for curative care and a sufficiently cost-effective one, a shift towards this type of behavior could greatly benefit society. While preventive care may be beneficial at all ages, the greatest potential individual and societal benefits may be found in young children during crucial stages of development. It is also reasonable to assume that subsections of the population, for instance those of a lower socioeconomic status and the uninsured, will benefit more from preventive care. Unfortunately, we lack a thorough understanding of the connection between preventive care and reduced health risks. It has proven difficult to establish causal links between preventive behaviors and illnesses later in life. There is also a dearth of evidence concerning factors that impact consumption of preventive care, such as health insurance status. My thesis is composed of two essays that contribute to the understanding of the potential health implications and benefits of preventive care and factors that affect the demand for preventive care in the U.S.

In the first essay, 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, in order to gain a better understanding of the effects of an investment in primary prevention on health. 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 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 two to ten times the estimated costs of the remediations.

In the second essay, I estimate the impact of expansions of two public health insurance programs, Medicaid and the State Children's Health Insurance Program (SCHIP), on the utilization of preventive care among children. Health insurance provides various incentives for individuals to alter their medical care utilization. A host of studies have established a positive association between increased eligibility for public health insurance and medical care utilization of children, but few focus on medical preventive care. This essay is a comprehensive examination of the causal relationship between the increased eligibility for Medicaid and SCHIP over a seven year period from 1997 to 2003 and consumption of well-child care and immunizations. I explore the differential impacts of eligibility across various subpopulations such as parental education, country of origin, and race to investigate how different groups react to the incentives provided by public health insurance. I use variation in the income cutoffs for Medicaid and SCHIP across states, time, and age groups to identify the effect of eligibility for public health insurance. I find that eligibility for public health insurance is associated with a 14 percentage point increase in the likelihood of consuming preventive physician visits and a two percentage point increase in the probability of having a usual source of care. Regarding immunizations, the evidence suggests that, in general, the incentives provided by eligibility are not enough to induce families to increase utilization. Hispanic children and those born outside of the U.S. do not increase utilization when

made eligible to the extent of their counterparts. Black children and those neither black or white take greater advantage of eligibility in establishing a usual source of preventive care. Conversely, the likelihood of having a physician's office as a usual source of preventive care increased more among white children.

To Mom, Dad, Jenn and Kim.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Chapter 1

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

1.1   Introduction

Childhood lead poisoning is the second most prevalent preventable disease in children[1] 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

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[1] Asthma is the most prevalent.

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 the U.S. More specifically, I intend to estimate the impact of an investment in 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.

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 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 incidence of lead poisoning should decline.

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 devastating 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 U.S. city (CDPH, 2004).

I observe each variable by year from 1997 to 2003 and by census tract. I begin by esti-

mating 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 in homes because children living there have tested positive for lead poisoning) is another potential problem. Again, OLS estimates will be biased upwards toward 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. Citywide, from 1997 to 2003, this effect translates to 1.75 cases averted for each additional housing unit treated. Furthermore, the negative impact of remediations on EBLs strengthens over time. I also find that 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.

1.2 Background

1.2.1 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 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 significant

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[2]Leaded 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.

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.

5

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 eliminat-

ing the major source of exposure (i.e., lead-based paint in older poorly-maintained housing).

1.2.2 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 1.1 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 1.1).

1.2.3 Related Literature

This analysis is the first to measure the large scale effects of investment in prevention on the incidence of childhood 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 remediation 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 corre-

lated 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 at 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.

1.3   Data

1.3.1   Data Source and Study Population

The Chicago Department of Public Health (CDPH) provided the total number of lead screens and positive cases[3] 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

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[3]The current CDC definition of childhood lead poisoning is a screen above 10 μg/dL.

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, 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 records.

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.

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[4]Census 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.

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

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

1.3.2   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. 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] Table 1.1 summarizes the calculation of key variables. For the purposes of this paper, a child testing at or above 10 μg/dL is considered lead poisoned. While there are different definitions of lead poisoning in children, 10 μg/dL is the most widely accepted standard (CDC, 2000). The mean across all tracts and all years is 12.72% (Table 1.2). 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 1.2).

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[7]Lead 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 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.

[8]I 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 2006 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.

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 receive public assistance. The yearly values of all census variables, with the exception of the percentage of housing units built prior to 1950[9], 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 1.3 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 1.2). 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 1.3). 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

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[9]This 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.

decline seen in the fourth quartile.

1.4 Methods

1.4.1 Conceptual Framework

I am most interested in the sign and magnitude of the coefficient on the remediation variable. Given that remediations have proven to lower BLLs in children through reduced exposure to lead hazards, a healthier housing stock should, all things considered, lead to a lower incidence of lead poisoning for children living there. Furthermore, I will observe any spillover effect (i.e., if a single remediation prevents lead poisoning in more than one child). For instance, 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. Children playing in and around the housing unit will also benefit from reduced exposure. Additionally, I am interested in how the impact of prevention 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

13

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.

1.4.2 Panel Analysis

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

$$\text{EBL}_{it} = \beta_0 + \beta_1 R_{it} + \beta_2' r_{it} + \beta_3' X_{it} + \alpha_t + \epsilon_{it} \tag{1.1}$$

where EBL_{it} is the percentage of children screened in census tract i that tested positive for lead poisoning measured in year t. 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

14

population receiving public assistance, median household income, and various interactions with year dummy variables. α_t 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.

There is a potential source of unobserved heterogeneity that 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 both in terms of identified cases of lead poisoning and 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 rather than including a dummy variable for each tract.[10] Any time invariant census tract factors are differenced out in the process. As long as the unobserved variation across tracts caused by the problem areas does not vary over time in tracts, the fixed effects will account for the heterogeneity problem. 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:

$$\text{EBL}_{it} = \gamma_1 R_{it} + \gamma_2' r_{it} + \gamma_4' X_{it} + \alpha_t + \gamma_i + \epsilon_{it} \qquad (1.2)$$

A potential problem with the first two specifications is that the percentage of units remediated is potentially endogenous and determined simultaneously with the dependent

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[10]Adding deviations from the mean avoids introducing N-1 additional parameters.

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 a remediation variable that captures only those remediations that occurred in years prior to the given year (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 (i.e., measured at the beginning of 2000). The alternate 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 rather than at the end.

1.5 Results

Using Model 1 (no census tract fixed effects), 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 1.4). 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 there is unobserved variation in the general level of lead poisoning activity across tracts 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 homogeneous 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 risk factor 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 1.5). A one-tenth percentage point increase in the percent remediated (a reasonable expected change given the yearly changes in remediations observed in Table 1.2) is associated with over a five-tenths percentage point reduction in the incidence of EBLs in a census tract. Citywide from 1997 to 2003, this is equivalent to 1.75 fewer cases of lead poisoning per additional remediation. The results are robust 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 percentage point.

In Model 3, the impact of the alternate remediation variable is several standard deviations greater than the original metric (Table 1.6). 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 the alternate remediation construction is also slightly greater when comparing the models 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 alternate 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.

1.6 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 toward 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 prevention outweighs the benefits of the associated reduction in lead poisoned children. In this section I estimate the benefits of an investment in the housing stock and compare them with the cost of the associated remediations.

1.6.1 Benefits

The benefits are the improved health and reductions in treatment costs for those children who would have been lead poisoned if not for the prevention. 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 treatment costs as the average cost of 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.

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 1.7). 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 μg/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 low level 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 μg/dL increase in BLL to a 9.6 point reduction from a one log point increase in BLL (Table 1.7). In a metaanalysis by Schwartz (1994a), the author estimated that a 1 μg/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 μg/dL increase in BLL was associated with a 0.46 decrease in

IQ. More importantly, they found that at BLLs below 10 μg/dL, a 1 μg/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 μg/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 μg/dL to below 10 μg/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 μg/dL rather than 10 μg/dL or even higher.

I incorporate all this information to estimate bounds on the increase in IQ from reductions in childhood lead poisoning (Table 1.8). For the minimum, I use Schwartz's estimate of a 0.245 point increase in IQ for each 1 μg/dL decline in BLL across all BLLs. This is a reasonable minimum since preventing lead poisoning means reducing BLLs below 10 μg/dL and recent studies have shown that the impact on IQ is greater below 10 μg/dL. I also assume a minimum decline of 2.8 μg/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 1.8).

I use the 0.74 IQ point increase associated with a 1 μg/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 μg/dL. I assume a conservative maximum 5 μg/dL drop in BLL for each case of lead poisoning averted. The

5 μg/dL decline is an average change across prevented cases of lead poisoning. It does not assume a 5 μg/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 μg/dL after only twelve months and that some children will be spared BLLs 10 μg/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 1.8).

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 separately. 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.[11] 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 1.8. The average for males and females is reported in columns 1 and 2 in row 7.

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 10 and 19 μg/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 μg/dL is a reasonable expectation for the treatment costs saved for each prevented case of lead poisoning (row 9). This figure is a minimum estimate for treatment cost savings. Remediations reduce lead exposure for all BLLs, not only those near 10 μg/dL. I do not include the potentially large cost savings at higher BLLs (several thousand dollars per case averted) such as reduced hospitalization and chelations.

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[11]The 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.

1.6.2 Costs

It is relatively simple to identify the costs of prevention. The cost is simply the cost of the remediations necessary to treat lead hazards in homes. The prices of remediations are available 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 1.8).[12] 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 1.8). 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.

1.6.3 Benefits Versus Costs

Table 1.8 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 specification in Model 3a (arrow 1, Figure 1).[13] 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 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%

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[12]The median cost is $6,795 per housing unit.

[13]I 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 SE of the number of cases converges. The standard errors for the number of cases and for the cost savings are reported in Table 1.8.

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-tenth percentage point increase in remediations (row 3 * row 8).[14]

Row 9 lists the average cost of curative care for a case of lead poisoning with a BLL between 10 and 19 μg/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.

1.7   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 toward

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[14]In 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(row 1)(row 6, males) + 0.5(row 1)(row 6, females).

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 1.2). 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. There is also a positive spillover to future child tenants in treated housing units. 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. Thus, over time, the preventive action continues to avert cases in siblings, playmates, and future tenants.

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 observed declining importance of race as a predictor of childhood lead poisoning over time (Tables 1.5 and 1.6).

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.

Table 1.1: 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 μg/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
Key variables	
incidence of lead poisoning	[# of children under age 6 with a positive test]/ [# of children under age 6 screened], by tract and year
% remediations	[# of pre-78 units with at least 1 rem in or prior to current year]/ [# of pre-78 units], by tract and year
% rem (alternate)	[# of pre-78 units with at least 1 rem prior to current year]/ [# of pre-78 units], by tract and year

Table 1.2: Summary Means by Year

variable	1997-2003	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 (alt)	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 (alt)	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 1.3: Mean %EBLs by the dependent, abatement, and control variables (quartiles)

Variable	1997	1998	1999	2000	2001	2002	2003
% remediated							
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
% remediated (alternate)							
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 1.4: 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 1.5: 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 1.6: Results from Model 3 - Census Tract Fixed Effects with Alternate 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 1.7: Summary of BLL and IQ loss literature

Citation	BLL increase	IQ point decrease
Canfeld et al. 2003	10 μg/dL increase in mean BLL	4.6
Canfeld et al. 2003	1 μg/dL increase in mean BLL	0.46
Canfeld et al. 2003	1 μg/dL inc in mean BLL for those never > 10 μg/dL	0.74
Lanphear et al. 2005	2.4 to 10 μg/dL	3.9
Lanphear et al. 2005	10 to 20 μg/dL	1.9
Lanphear et al. 2005	20 to 30 μg/dL	1.1
Schwartz 1994	10 to 20 μg/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 μg/dL	4.3

Note: Schwartz, 2002 is a metaanalysis.

Table 1.8: Benefits and costs associated with a one-tenth increase in the remediation variable

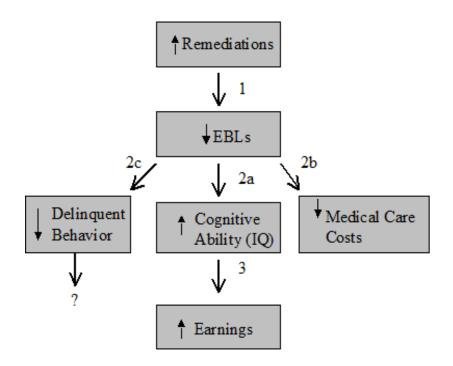| Event	Schwartz (1994) min	Schwartz (1994) max	Salkever (1995) min	Salkever (1995) max
Pre-Calculations				
1. number of remediations required for one-tenth % increase	2,081	2,081	2,081	2,081
2. average cost of remediation in Chicago, IL	7,750	7,750	7,750	7,750
3. reduction in cases of lead poisoning	3,506	3,506	3,506	3,506
(standard deviation)	(727)	(727)	(727)	(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 increase				
male	1.76	1.76	1.93	1.93
female			3.23	3.23
6. % increase in earnings from one less case (row 4 × row 5)				
male	1.21	6.51	1.33	7.14
female			2.23	11.93
7. expected lifetime earnings (males and females)				
male	700,329	700,329	881,027	881,027
female			519,631	519,631
8. change in exp lifetime earnings from one less case (row 4 × row 5)	8,473.98	45,591.42	11,652.72	62,448.88
9. average cost of short term treatment for low level case of lead poisoning	64.34	64.34	64.34	64.34
Benefits in thousands				
10. total change in earnings (row 3 × row 8)	29,710	159,844	40,854	218,946
11. total reduction in curative care costs (row 3 × row 9)	226	226	226	226
Total Benefits (row 10 + row 11)	**29,935**	**160,069**	**41,080**	**219,171**
Costs in thousands				
Total Costs in thousands (row 1 × row 2)	**16,128**	**16,128**	**16,128**	**16,128**
Difference between Benefits and Costs in thousands	**13,808**	**143,941**	**24,952**	**203,044**
(standard deviation)	(6,207)	(33,192)	(8,518)	(45,447)

Figure 1.1: Benefit Pathway from Increased Remediation

Chapter 2

The Impact of Public Health Insurance Eligibility on Preventive Care Utilization Among Children

2.1   Introduction

The medical community has long recognized and reported that preventive medical care is an effective method of reducing health risks and driving down future medical costs (Cohen et al., 2008). Preventive care, such as a routine check-ups, is particularly important in young children as it allows a physician to diagnose and correct conditions that could potentially disrupt crucial stages of development. Given this estimated positive productive effect and the benefit of good health for future happiness and prosperity, it is important to identify the factors that restrict consumption of preventive care, e.g., routine well-child care or immunizations. One commonly cited culprit is lack of health insurance. 18% of children in families with incomes less than 200% of the federal poverty level did not have health insurance in 2004 (U.S. Census Bureau, 2007). Other barriers include lack of knowledge about the benefits of preventive care, travel and time costs, differences in the valuation of future health, and the priority assigned by families to preventive care relative to basic necessities, e.g., food, education, and shelter. The expansions of the public insurance program Medicaid and the rise of the State Children's Health Insurance Program (SCHIP) from 1997 to 2003

to cover uninsured children provide an ideal setting to evaluate the role of health insurance availability on preventive care consumption.

In this paper, I estimate the impact of expansions of two public health insurance programs, Medicaid and SCHIP, on the consumption of preventive care among children. More specifically, I measure how *eligibility* for these programs effects the decision to consume childhood preventive care. I will also study how changes in the consumption of preventive care vary among subgroups, i.e., by race, country of origin, and parental education.

I use the variation in Medicaid and SCHIP eligibility criteria from 1997 to 2003 to identify the impact of public health insurance availability on preventive care consumption. Observed insurance coverage is an individual (or household) choice, even among those eligible for Medicaid and SCHIP. In the study sample, over half of individuals who are eligible for government provided health insurance do not enroll (comparable studies have found similar rates using different nationally representative surveys and samples). I avoid biased estimates of the effect of coverage on preventive care use by estimating the impact of individual public health insurance eligibility, which varies exogenously across state and time during the period of study, rather than the impact of the observed health insurance choice. States choose the income levels and the age groups that their plans will cover. From 1997 to 2003 (as well as during the previous and subsequent years), there was considerable variation in the eligibility rules by state and time. Using the detailed income, state, and age data in the National Health Interview Survey (NHIS), I construct individual level eligibility measures for each child in the sample.

Two potential sources of bias remain when estimating the effect of eligibility on consumption of preventive care: (1) omitted unobservable state and age group variation that differentially impact eligibility rules and health care consumption and (2) an endogeneity bias if, for example, sicker children have higher levels of utilization and are more likely to be eligible because previous health care costs have driven down family income. If not addressed, these sources will lead to biased coefficients on the relationship between consumption and eligibility. I follow the methodology outlined in Currie and Gruber (1996) to address these additional sources of bias, i.e., a combination of a full set of state/time/age fixed effects and

a simulated state level eligibility metric to instrument for individual eligibility.

The two types of preventive care considered in this essay are preventive physician visits and immunizations. The preventive visit variables characterize the regularity and source of routine medical care. The immunization variables measure whether a child (0 to 6 years of age) has ever had a particular vaccine (5 total), whether they are up-to date, and whether the vaccines were administered at the age recommended by the CDC.

Eligibility for public health insurance is associated with a 14 percentage point increase in the likelihood that a child has had a routine check-up in the previous twelve months. Similarly, eligibility is associated with an increased likelihood of having visited any physician in the last twelve months (10 percentage points), a usual source of care (4.3 percentage points), and a usual source of preventive care (2.0 percentage points). Eligibility does not increase the likelihood of having a physician's office as a usual source of general or preventive care. The impacts of eligibility are less than half of those for enrollment in public health insurance - although the effects are clearly biased due to omitted unobserved variables that influence both enrollment and utilization.

I find that eligibility is associated with a one to two percentage point increase in the likelihood of having single doses of and being up-to-date for the diphtheria and polio vaccines. Eligibility has no positive effect on the other three immunizations. Conversely, enrollment has a strong positive effect on the timeliness and completeness of the immunization series (without considering the endogenity of Medicaid and SCHIP enrollment). This finding provides some evidence that there are unobserved characteristics that impact both enrollment and utilization of immunization that are distinct from eligibility. Perhaps having a physician's office as a usual source of care (a variable influenced by enrollment but not by eligibility) is an important factor in whether children have all their immunizations and have them on time.

Evidence suggests that certain subpopulations react differently to the incentives provided by public health insurance with regard to utilization of preventive care. The probability of having a usual source of preventive care increases more for black children and those neither black or white under eligibility. However, the increase in the likelihood of having

a physician's office as the usual source of preventive care is higher among white children. Hispanic children and those born outside the U.S. were less likely to increase consumption of the preventive medical visit variables considered in this study.

2.2 Background

2.2.1 Medicaid expansion and the creation of SCHIP

Since the early 1980's there have been several major federal expansions in Medicaid coverage for young children and pregnant women. The purpose of the expansions was to reduce the tens of millions of uninsured Americans by relaxing the income and age requirements for enrollment into the program. Each successive expansion lowered the maximum family income level (as a percentage of the federal poverty level - FPL) and increased the age at which children could be considered eligible for Medicaid. Beginning in 1990, states were required to cover pregnant women and children up to age 5 whose incomes were below 133% of the FPL and had the option to cover them up to 185% while still receiving federal matching funds. By 1996, there were 31.5 million Americans enrolled in Medicaid (11.8 percent of the total population). Additionally, 29% of children in households with incomes from 100% to 125% of the FPL went all of 1996 without insurance (U.S. Census Bureau, 2007).

In 1997, under the Balanced Budget Act, the federal government created the State Children's Health Insurance Program (SCHIP) to further extend health coverage to uninsured children. The program was designed to provide coverage to children who are not eligible for Medicaid and not covered by private insurance (in particular, children in families with incomes between 100% and 200% of the FPL). The federal government allowed states to set their income eligibility cutoff up to 200% of the FPL or 50 percentage points above where their cutoff was on March of 1997 (whichever is higher) while still receiving matching funds. Although the federal spending on the program was capped at 40 billion dollars over 10 years, the matching rate was set higher than that under Medicaid.

While the federal government has set the eligibility maximum at 200% for the purposes of matching funds, each state has the freedom to decide their own eligibility cutoffs based

on age and a percentage of the FPL. Over time, states have altered their guidelines to cover more and more families. The uptake and magnitude of these expansions vary greatly by state. Thus, there is significant variation in the eligibility rules for Medicaid and SCHIP by state over time.

The federal government allows states some freedom in how they design their SCHIP. Regarding program structure and implementation, states were given three options: (1) form a separate program, (2) incorporate it into Medicaid, or (3) implement a combination of the two. By 2000 all 50 states and the District of Columbia had adopted an SCHIP program: 16 separate, 16 as part of Medicaid, and 19 combinations (Dubay et al., 2002). States with separate Medicaid and SCHIP programs screen families to see if they are eligible for Medicaid. By rule, families who are eligible for Medicaid are enrolled into this program rather than SCHIP. If a state implemented SCHIP as part of Medicaid, they are required to offer enrollees the same set of benefits they receive under Medicaid. Separate programs are allowed to vary their benefit packages, although there is a set of minimum benefits required of all programs. Separate programs are also allowed to apply limited cost sharing measures to beneficiaries. Therefore, SCHIPs vary in benefits and costs and consequently vary in attractiveness to potential enrollees.

According to a congressionally-mandated evaluation of the SCHIP initiative (Congressional Budget Office, 2003), the program is having success in reaching the target population, i.e., poor, uninsured children. 91% of enrollees in 2002 were in families with incomes less than 200% of the FPL (23% were between 150% and 199%). Furthermore, 43% of enrollees had no insurance in the six months prior to enrolling. While 28% of enrollees had private insurance in the six months prior, half of these lost this coverage at some point during this time and one quarter responded that their coverage was "unaffordable". Children 0 to 5 years of age made up 19% of the sample while children six to twelve and those older than 13 made up 48% and 33%, respectively. The higher enrollment rates among older children could be because younger children are eligible at lower incomes and thus are enrolled in Medicaid. Hispanic, white, and black children made up 49%, 32%, and 12% of the sample, respectively.

2.2.2 Related Literature

In the mid-1990s, there were a number of studies that used the variability in Medicaid expansions across states to examine the impact of public insurance on a wide variety of health utilization and outcome variables. Since the creation of SCHIP in 1997, there have been studies that have drawn from and expanded upon the methodologies developed in the earlier papers to examine this new form of public insurance. In this section I discuss studies that examine the impact of both Medicaid and SCHIP expansions as they relate to my work.

Prior studies of Medicaid expansion seek to answer two basic questions: (1) What was the uptake of public health insurance during Medicaid expansion and to what degree did this crowd out private insurance and (2) Did health care utilization and outcomes among children increase under Medicaid expansion?

The studies focusing on the latter question have estimated the impact of expansions on a variety of child health utilization measures. Currie and Gruber (1996) found that eligibility between 1984 and 1992 was associated with a 9.6 percentage point reduction in the probability of going 12 months without a visit to a physician (a halving of the baseline percentage). Two other studies found similar effects for poor black and Hispanic Medicaid enrollees and for children between 1993 and 1994 with insurance (Racine, 2001; Newacheck, 1998). Dafny and Gruber (2004) found that a 10 percent increase in eligibility is associated with an 8.4% increase in hospitalizations. These studies are primarily concerned with whether expansion led to increased curative utilization. They ignore the role increased eligibility may have played in changing preventive care in children.

There have been several studies that have looked at Medicaid and its role in preventive care. Currie and Grogger (2001) examined the impact of various Medicaid policy changes on adequate prenatal care across different segments of the population from 1990-1996. They found that increases in the income cutoff for Medicaid eligibility led to increased use of "adequate" prenatal care among white women, but there was no effect for black women.

Kaestner et al. (2000) examined the impact of ambulatory care sensitive (ACS) hospitalizations in children. Their hypothesis is that increased eligibility should lead to better

primary care among enrollees and that since better primary care has shown to reduce ACS hospitalizations that increased eligibility should reduce such hospitalizations. The authors found strong evidence that eligibility decreased the incidence of ACS hospitalizations among children between 2-6 years of age in "very low income" families. There was little evidence that Medicaid expansions had an impact on ACS hospitalizations among children of other ages and incomes. The authors were forced to use median income at the zip code level rather than individual level data. Thus the claim that they were measuring the impact of eligibility is dubious.

Currie and Thomas (1996) study the utilization of checkups and physician visits due to illness by race and insurance type. The authors exploit the longitudinal nature of National Longitudinal Survey of Children and Mothers (NLSCM) from 1986 and 1988 to control for unobserved child fixed effects. They found that white children on Medicaid have roughly 15% more check-ups than those with private or no insurance while there is no significant difference between the numbers of check-ups for black children across all insurance types. Currie and Thomas (1996) comes closest to this study in terms of its focus on differential levels of preventive care although they measure the impact of actual enrollment. As the study uses 1986 and 1988 data only, one cannot comment on the role of policy shifts over time and evolving eligibility rules on the dependent variables. In addition, this study adds more preventive care variables, e.g., immunizations, and extends the analysis to SCHIP coverage.

There is a body of research that has examined the national impact of SCHIP on utilization of care. These papers can be grouped into two broad categories according to the outcomes they study: (1) access to or utilization of general physician visits and (2) specific preventive care variables such as immunizations or dental visits.

Papers in the first group tend to focus on having a regular source of care and the occurrence of a visit to a general practitioner. They do not distinguish the visits as preventive, i.e. well child visits or check-ups. Their goal is to measure the impact of SCHIP on the access to and utilization of general physician visits. In a forthcoming paper by Wang et al., the authors find that SCHIP availability increases the probability of a visit to a "general doctor"

in the past 12 months by 7.4%. Furthermore, the impact of SCHIP was amplified as the length of availability in an area increased. Davidoff (2005) found that among children with chronic health conditions, eligibility increased the probability of a general physician visit by 2.4%. In addition, several studies have found that SCHIP eligibility decreases unmet dental care need and increases the probability of dental visits over the past year (Wang, 2007; Davidoff, 2005). One study found evidence that uninsured children are more likely to have no preventive dental visits than those with SCHIP or private insurance (Kenney et al., 2005).

One recent paper directly addresses public health insurance and the likelihood of a preventive physician visit. Perry and Kenney (2007) find that publicly insured children are roughly 4 percentage points more likely to have had one preventive visit than full-year privately insured children and 16.5 percentage points more likely than full-year uninsured children. The study does not address the endogeneity of insurance choice. The authors simply report point estimates of the impact of public insurance while controlling for a host of sociodemographic factors.

Joyce and Racine (2003) measured the impact for children 19 to 36 months of age between 1995 and 2003 living in an area where SCHIP is available and having an income below 200% of the FPL on being "up-to-date" for certain immunizations. The authors focused on the difference in impacts between "poor" and "non-poor" children. The only significant impact was found for varicella vaccinations.

The evidence regarding the impact of Medicaid/SCHIP and race on preventive care is varied. For instance, while several studies have found evidence that Hispanic and/or black Americans increased utilization under expansion relative to white Americans (Kenney et al., 2005; Racine et al., 2001; Congressional Budget Office, 2003; Currie and Thomas, 1995), other studies have found the opposite effect (Currie and Grogger, 2002; Currie and Gruber, 1996). Public insurance and parental education seem to have a positive impact on various measures of preventive care (Currie and Grogger, 2002; Currie and Gruber, 1996; Congressional Budget Office, 2003).

The lack of studies focusing on the effect of public insurance expansion through the

44

creation of SCHIP on purely preventive care variables is the most glaring hole in the literature. Even the studies that use preventive care variables as outcomes group them with all other types of medical care rather than treating prevention as a separate type of care. Currie and Gruber (1996) use routine physician check-ups as their measure of utilization out of convenience simply because it should be unaffected by morbidity unlike most curative care variables, i.e., curative medical care may decrease under expansion since individuals are likely to use more preventive care and will therefore be healthier in general.[1] In addition to filling this gap, I will add to the body of SCHIP studies by constructing individual eligibility using age, income, and state of residence. This methodology has been used in the past, although the majority of studies simply measure the impact of being poor and living in an area with an SCHIP program rather than simulating individual eligibility. I will also add to the work in Joyce and Racine (2003) by including whether immunizations were administered on time (according to Centers for Disease Control and Prevention - CDC - recommendations) as a dependent variable as well as if they were up-to-date at the time of the survey for a wider range of ages (0 to 6 years of age rather than 19 to 36 months).

2.3   Motivation

The goal of this essay is to determine how the aforementioned "eligibility" impacts the utilization patterns of a crucial component of medical care: well-child physician visits and immunizations. Routine check-ups are known to minimize the likelihood of developing future conditions as well as minimizing the damaging effects of current conditions. Foremost among the long, varied list of conditions are diabetes, asthma, and the host of illnesses prevented by immunizations. These and other illnesses can disrupt the delicate development of young children. Thus, the costs of illnesses are generally greater in children than in adults. The benefits of preventing childhood illnesses are also greater. Among children, those eligible for public insurance are particularly interesting as they come from relatively low-income homes that tend to be underinsured compared to the rest of the population.

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[1]This argument ignores the possibility of ex ante moral hazard.

Childhood immunizations are necessary to protect children from a myriad of infectious diseases such as diphtheria, polio, measles, and HIB.[2] While new borns receive immunity to certain diseases from their mothers, it lasts only several months. If a child is not vaccinated at this point and is exposed to one of the preventable diseases, they may become severely ill. It is also imperative for the efficacy of the immunizations that they be delivered on time and that children receive all the doses in the series as recommended by the CDC (Appendix A.1). Strong immunization programs also create a positive externality in which each individual vaccination reduces the likelihood of contraction for everyone in the community. Although vaccine preventable diseases are at a record low in the U.S., the viruses and bacteria that cause them still exist. It is rare that we observe them due to very aggressive immunization programs and laws. To keep the prevalence of these diseases at the current level, we must continue the current level of vaccination.

I choose to measure the impact of eligibility rather than actual enrollment. The effects of eligibility and enrollment on the consumption of preventive care are closely related and equally interesting, however, they provide us with very different information. Eligibility is a government policy tool used to control the availability of public health insurance. A methodology that estimates the impact of eligibility is measuring the effect of state and federal regulations. Conveniently, eligibility rules vary greatly by by state and age over time. Although income and state of residence (which determine eligibility) are choices, many have argued that individual eligibility is a source of exogenous variation (discussed further in the Methods section). Individual eligibility is certainly very closely related to enrollment as the decision to enroll is conditional on being eligible. However, enrollment is a choice by households and individuals that depends on factors other than eligibility. Less than half of the eligible children in the sample are enrolled in Medicaid or SCHIP.

An important specific goal of this paper is to examine how various subpopulations respond to public health insurance eligibility. Several studies have shown that factors such as race, ethnicity, and parental education influence the consumption of health care (including preventive care) in children (Yu, 2002). One proposed explanation for the difference among

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[2]Haemophilus influenzae type b.

subgroups is variation in health insurance status. Thus, if the gap in health insurance closes, so too might the gap in utilization. I estimate the differential impact of eligibility on consumption of preventive care among subgroups to investigate whether or not health insurance status can explain differences in utilization.

2.3.1 Conceptual Framework

The availability of public health insurance creates incentives to alter preventive medical care decisions by changing the out-of-pocket cost of both preventive and curative medical care. First, by providing the option of free (or close to free) care and driving down out-of-pocket preventive medical costs, public health insurance reduces the negative influence of preventive care on household budgets. On the other hand, health insurance reduces the cost of future illnesses by lowering the out-of-pocket cost of curative care. Thus, if individuals are sufficiently risk neutral, they might be willing to face the risk of future illnesses rather than invest in prevention in the current period. Public insurance, by changing the cost of medical care, provides two opposing forces on the consumption of prevention.

Whether individuals increase or decrease utilization when eligible depends largely upon the probability and severity of illnesses as well as the efficacy of both preventive and curative medical care in improving health. For instance, if the probability of an illness is very low or the efficacy of preventive care in lowering the probability of illness is low, the price incentives created by public health insurance may have little effect on utilization of prevention. Conversely, if the probability of illness is very high, then even a very small price incentive may have a large impact on utilization. Similarly, the relative efficacy of preventive and curative medical care in improving health will determine how the price incentives of health insurance influence utilization. If preventive medical care has a large positive impact on health, then insurance should provide a strong incentive to consume more prevention. However, if curative care is particularly effective in increasing health but the out-of-pocket costs relatively high, then public health insurance might provide a strong incentive to incur the risk of future illnesses rather than consuming preventive care in the current period. In this case, preventive care, is in a sense an inferior good.

47

The availability of public health insurance decreases the out-of-pocket costs for routine checkups and other prevention related visits. Medicaid and SCHIP plans in all 50 states cover at least one well-child physician visit per year. Thus, one would expect that preventive visits would increase among those with no insurance and no chance of public coverage, in which all costs are out of pocket, when public insurance is made available. Preventive physician visits might also increase among those with private insurance under increased eligibility as they would pay lower or no premiums and co-payments under Medicaid or SCHIP. This impact could be dampened in states where the SCHIP has a cost sharing component as the potential savings of public insurance are partially eroded by the premiums attached to these programs.

The role of public health insurance eligibility in the consumption of childhood immunizations is less clear. Basic immunizations are funded for all Medicaid, uninsured, and underinsured (those with private insurance that does not cover immunizations) children under the Vaccinations for Children Program (VCP). Age eligible children can receive CDC recommended immunizations at any registered VCP provider for an administration fee which ranges from 10 to 15 dollars. According to the CDC, "most" pediatricians as well as many family practice providers, general practitioners, and sub-specialty health care providers are enrolled in the VCP (over 50,000 enrollees nationwide).[3] Thus, there is no out-of-pocket cost savings for those receiving Medicaid or SCHIP benefits (save the administration fee each visit) and little incentive to alter utilization of immunizations. Another potential limitation of the role of public health insurance in the consumption of immunizations is that there is little variation in whether a child has received at least one dose of a vaccination. With the exception of Varicella[4], over 80% of the sample received at least one dose of each of the other five shots (Appendix A.2)[5] - consistent across eligibility and enrollment status (Tables

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[3]There is no mention of whether there are fewer registered providers in traditionally under served areas. This would allow public health insurance a larger role in increasing utilization of immunizations in these areas.

[4]The rate of utilization of the varicella vaccine is lower than the other immunizations and is increasing rapidly over the course of this study because it was not approved by the Food and Drug Administration (FDA) until 1995.

[5]The rate is over 90% if the parent provided the information.

2.1 and 2.2).

However, there is significant variation by eligibility and enrollment in the percentage of children whose immunizations are up-to-date and whether or not they were administered on time (Tables 2.1 and 2.2) according to CDC recommendations. Eligibility could have a significant positive effect on the timing and completeness of immunization series by increasing regular child contact with physicians and awareness rather than through price.

Preventive care could potentially decline due to the increased availability of public health insurance, i.e., the ex ante moral hazard problem. According to the ex ante moral hazard dilemma, people engage in riskier behavior when they are insulated from the consequences of a negative outcome. In health terms, people reduce healthy behaviors (preventive care) when they have access to health insurance and can therefore afford treatment (curative care) when they become sick.

Given the ex ante moral hazard problem, preventive and curative care are types of substitutes. Individuals substitute healthy behaviors today for a procedure or treatment tomorrow once they are ill. If we can assume that most parents do not "gamble" with their child's health and that the price incentives are strong enough, the ex ante moral hazard problem should not be a concern. Furthermore, there are no effective sources of curative care for the diseases prevented by immunizations. Ex ante moral hazard is more of a consideration for non-medical preventive care such as diet, exercise, and general health behaviors in adults.

2.4   Data

2.4.1   Data Source and Study Population

Individual level data are obtained from the 1997-2003 waves of the National Health Interview Survey (NHIS). The NHIS is an annual survey of the health of the non-institutionalized population of the U.S. conducted by the National Center for Health Statistics under the CDC. Each year a new set of households from across the U.S. take part in the survey. The survey contains of a core set of questions asked of each member of the household. In addition

to the core component, one sample child and one adult are chosen at random to answer an additional set of health related questions. The sampled children also take part in a separate immunization module. However, most of the questions in the immunization module are asked only of sampled children 0-7 years of age. Thus, the sample for the vaccination analysis is restricted to children 0-7 years of age. The core and sample questionnaires are redesigned every fifteen years. The most recent changes were made in 1997.

The estimation sample includes all children 0 to 18 years of age selected to participate in the sample child survey (with the exception of the immunization models which, as mentioned previously, include only children 0-7 years of age). I begin the sample in 1997 because the questionnaire was redesigned at this time. The 1997 version includes additional questions about preventive physician visits as well as a more comprehensive set of immunizations. Even though NHIS data is available for 2004 and 2005, I chose to end the sample in 2003 because the waves after this point do not include the immunization component other than several questions about the influenza vaccine. Thus, the variables used in this study are reported consistently in the NHIS surveys from 1997 to 2003.

The entire NHIS sample from 1997 to 2003 contains 686,233 individuals, 193,980 of which are children. Of those children, 100,402 were randomly selected to take part in the sample child and immunization modules (roughly 14,000 in each year). The NHIS reports immunization data for each participating child from one of two different sources. The information is either taken from a vaccination card or provided by the child's parent/guardian. Of the children participating in the immunization module, 26% had immunization records. The immunization questions were answered by a parent for the remaining 74% of the sample.

A study of vaccination cards versus medical records showed that in general, cards approximated the actual records quite well (Fierman et al., 1996). Suarez (1997) found that parental recall was not a very reliable approximation of immunizations on medical records. Parents tended to under report vaccinations. However, the authors found that the combination of parental recall and immunizations cards were accurate predictors of vaccinations. The studies report that the ability to accurately recall immunizations did not vary by race, education, or health insurance.

In the NHIS sample, the percentage of children up-to-date and having ever received a vaccine are much higher (for all immunizations) when a parent reported the information (Appendix A.2). Since the survey only reports immunizations from one source for each child, I cannot determine whether the parents over report or the vaccination cards under represent the percentage of children receiving shots. If we trust the vaccine cards, then it appears that parents are over reporting immunizations. Conversely, the immunization cards could be missing information. This is unlikely, however, as the survey allows parents to fill in any shots missing from the cards. The samples are similar in observable sociodemographic traits with several slight differences (Table 2.3). For example, the percentage of children with immunization cards is slightly higher among those of Hispanic origin and those living in the Midwest and West. I separate the sample into two groups, children with immunization records and those with information provided by parents, and run the analysis on each sample separately.

2.4.2 Measurement of Key Variables

The dependent variables fall into two categories, preventive physician visits and childhood immunizations. The first group includes the following variables indicating whether or not the child: (1) has had a preventive check-up in the past 12 months, (2) has had any physician visit in the past 12 months, (3) has a usual place for preventive care, (4) has a physician's office as a usual source of preventive care, (5) has a usual place for general care, and (6) has a physician's office as a usual source of general care.

The immunizations included in the sample are: (1) Diphtheria, Tetanus, Pertussis (DPaT), (2) Polio, (3) Measles, Mumps, Rubella (MMR), (4) Haemophilus influenzae type b (HIB), and (5) Varicella (chicken pox). There are three potential ways to measure the consumption of these vaccinations in children. The first is whether a child has ever received a shot as the measure of a vaccination. The second measure is if a child is up-to-date for each immunization separately at the time of the survey, according to the official recommendations of the CDC (Appendix A.1). I am able to determine each child's status using their age at the time of the survey and the count of each type of immunization. The last metric

is whether a child received the immunization by the recommended date, again per CDC recommendations. I am able to determine timeliness of vaccinations using the age of the child and the date of each vaccination for those children that a shot record is present.

I use information in the NHIS along with state level Medicaid and SCHIP eligibility rules to construct a measure of eligibility for every child in the sample. Individual eligibility is determined by the state of residence, income as a percentage of the federal poverty level (FPL), and the age of the child. States set eligibility cutoffs for each age group based on family income as a percentage of the FPL. Although state identifiers are censored in the NHIS public use files and income information is coded into broadly defined groups to protect the identities of the respondents, I have gained access to the unrestricted data so that I can construct the necessary eligibility variable. The maximum income level as a percentage of FPL for infants varies greatly by state from 1997 to 2003. By 2000, children aged 0-18 are covered at some level in every state. However, the level of coverage still varies from state to state. Furthermore, there is variation in the maximum income as a percentage of FPL by age groups within states in a given year.

The right-hand-side variables included in the various models are summarized in Appendices C and D. The explanatory variables were all taken from the core component of the NHIS. The variables control for individual, parental, and family level sociodemographic traits. The composition of the sample has remained uniform from 1997-2003 with a few exceptions. Private insurance has declined somewhat while Medicaid has risen from 18% in 1997 to over 20% in 2003. SCHIP coverage rose from roughly 1% in 1999 to almost 6% in 2003. Not surprisingly, the percentage of children eligible for either Medicaid or SCHIP rose dramatically from 1997 to 2003 due to increasingly generous eligibility rules. The rise is particularly noticable in 1998 and 1999 when many states adopted maximum %FPLs of 200 and above.

Appendices E and B summarize utilization of preventive physician visits and immunizations by year, respectively. Utilization is consistently high for all the preventive visit variables (Appendix A.5). Similarly, immunization rates when reported by parents and "any recorded shots" taken from the vaccine cards are very high with little variation over time

(Appendix A.2)[6]. However, the proportions of children receiving vaccinations on time have increased over time for each type of immunization.

Tables 2.2, 2.3, and 2.4 report the utilization of preventive care variables by public health insurance eligibility and by enrollment status for children *not enrolled in a private insurance plan.* The utilization rates of physician based preventive care are higher for those eligible for public health insurance (Table 2.4 - top panel). The percentage of children eligible for either Medicaid or SCHIP with a routine well-child checkup in the previous 12 months is almost 4 percentage points higher than those not eligible for public insurance. The differences are even greater between those enrolled and not enrolled in Medicaid or SCHIP (Table 2.4 - bottom panel). Surprisingly, children eligible for public health insurance are slightly *less* likely to have had immunizations - with the exception of the varicella vaccine[7] (Table 2.2). However, those actually enrolled in either Medicaid or SCHIP are more likely to be up-to-date and have had their immunizations on time - particularly when reported on a vaccination card (Table 2.3).

Table 2.5 reports the differences in utilization of a subset of the preventive care variables by race, Hispanic origin, U.S. birth, and maternal education (a subset of the covariates). In general, children of Hispanic origin, born outside the U.S., and those with mother's of low educational attainment had lower levels of preventive care consumption. Utilization by race depends on the type of care. Black children were more likely to have had a routine checkup over the past 12 months and a usual source of preventive care. White children were more likely to have a physician as their usual source of preventive care and receive their immunizations on time.

Table 2.6 shows how the selected subgroups vary by public health insurance for routine checkups. The bold figures can be interpreted as the difference in how subgroups differ by public health insurance status in the consumption of preventive care. For example, the difference in utilization of routine check-ups between those eligible and non-eligible is

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[6]The only exception is the Varicella vaccine which was only approved in 1995.

[7]One previous study found a positive impact of eligibility on varicella but not other vaccines. The rationalization for the impact is that there is room for improvement for the up-take of the varicella vaccination while the utilization of the other recommended immunizations is near 100%

greater for those born in the U.S. and for those with lower levels of maternal education. The differences for white children and those of Hispanic origin were only slightly greater than the comparison groups. This provides a small amount of evidence that these groups react more to the incentives provided by public health insurance eligibility. To be convinced, we must control for a host of confounding variables such as income/poverty, age, and family size.

2.5 Methods

I estimate a linear probability model[8] of preventive medical care utilization among children. I exploit the state and age variation in Medicaid and SCHIP eligibility over time to estimate the impact of eligibility on preventive care. There is significant variation in the income cutoffs by state and age over time. Thus, public health insurance eligibility is primarily determined by state legislative environments rather than individual and family choices. I run all models with actual enrollment in Medicaid/SCHIP from the NHIS as a comparison to the results with eligibility. The basic specification is:

$$R_{it} = \beta_0 + \beta_1 I_{it} + \beta_2' X_{it} + \beta_3' Y_{it} + \beta_4' C_{st} + \beta_5 P_{it} + \delta_s + \theta_t + \alpha_i + \alpha_i \times \delta_s + \alpha_i \times \theta_t + \epsilon_{it} \quad (2.1)$$

R: whether or not the child utilized the preventive care

I: Medicaid/SCHIP eligibility

X: a vector of individual and family characteristics (gender, race, ethnicity, U.S. birth, income, maternal education, number of family members)

Y: select covariates interacted with eligibility

C: state plan characteristics by year

P: whether or not the child is covered by private insurance δ: State dummies

θ: year dummies

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[8]I estimate a linear probability model for ease of computation, particularly in estimating and interpreting interaction terms, and for consistency in the 2SLS estimations.

α: age group dummies

I_{it} denotes eligibility for either Medicaid *or* SCHIP[9]. The preventive care variables (listed in Tables 2.1 and 2.5), R_{it}, measure preventive physician visits and immunization utilization. I control for child and family traits that determine Medicaid/SCHIP eligibility (income and the age of the child) as well as exogenous factors that might affect utilization (gender, race, ethnicity, U.S. birth, family size, and maternal education). I also control for a state Medicaid/SCHIP plan characteristics that might influence individual choice to consume preventive care - foremost of which are whether the plan has a cost sharing component and whether Medicaid and SCHIP are joint or separate programs.

Even when using eligibility rather than insurance choice, there are still several estimation issues: (1) there are possible omitted group level unobservables that influence both Medicaid eligibility and utilization of preventive care, and (2) individual eligibility is potentially endogenous, e.g., sick children that require more preventive care may reduce parental income leading to a spurious positive relationship between eligibility and utilization. I include self-reported health status as an initial attempt to control for the preexisting health stock of children. However, the reliability of self reported health is highly dubious as a true measure of health and very few parents responded that their child was in either of the lower two health categories, fair or poor (Appendix A.3).

There may be state and age group unobservables that impact both preventive medical care utilization as well as Medicaid eligibility rules (problem 1 discussed above). For instance, relatively wealthy states may have both more generous Medicaid policies and higher medical care utilization (Currie and Gruber 1996). To account for omitted variable bias, I include state (δ), time (θ), and age (α) dummies. To control for any differences in utilization across age groups over time and age groups within states I include interactions between age and year as well as age and state. Thus, the identification comes from changes in eligibility within states over time, and changes within age groups in states over time.

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[9]In states with separate Medicaid and SCHIP programs, SCHIP will provide coverage to many low income families that do not meet the eligibility criteria for Medicaid. States attempt to ensure that any children eligible for Medicaid are enrolled in that program rather than SCHIP.

In order to account for the individual level endogeneity bias outlined above (issue 2), I instrument child eligibility with a state level Medicaid/SCHIP eligibility metric. By instrumenting state level eligibility by age and year for each child, I purge the analysis of any individual level omitted variable bias, i.e., variables that influence both utilization and eligibility. However, as with the Currie and Gruber 1996 study, the NHIS does not have enough observations to construct an eligibility rate for each state/year/age combination. To create a large enough sample, I first extract a random sample of 300 children for each year and age group combination (300*7*18). I then apply each state's eligibility criteria to this nationally representative sample yielding 51 different versions of individual eligibility. Using this sample, I calculate a state level eligibility rate for each state/age/year combination. I assign the single appropriate state/age/year instrument to each child. This metric has the added benefit of varying only by the "legislative environment" in each state rather than by the state and time economic conditions that might affect both eligibility and consumption of preventive care.

2.6   Results

Table 2.7 presents estimates for the impact of both eligibility for and enrollment in public health insurance programs (Medicaid and SCHIP) on the utilization of six measures of routine medical care. The second and third columns report the impact of eligibility using a linear probability model estimated by OLS and a 2SLS approach with individual eligibility instrumented by a simulated group eligibility rate. Without the instruments, eligibility is associated with a 4.0 percentage point increase in the likelihood of having a routine check-up in the last 12 months and a 1.9 percentage point increase in any visit to a health professional in the last 12 months. These changes are 13.3% and 14.8% increases over the baseline percentages for those eligble for public insurance but without any visits over the past 12 months. The results in column two also suggest that eligibility increases the likelihood of having a usual source of general care and preventive care by 1.3 and 0.8 percentage points, respectively. These may seem like modest changes, however they are

11.6% and 10.7% increases over the baseline for those eligible but without a usual source of care. There is no evidence that eligibility increases the likelihood that a child's usual source of care is a physician's office.

Estimates using individual level eligibility could potentially be biased upwards or downwards, i.e., sick children with high levels of utilization could create enough of a financial burden so that the family becomes eligible or conversely, there could be unobserved traits that increase the probability that a child is eligible that also make them less likely to consume preventive medical care. The 2SLS results in column 3 suggest that the estimates in column 2 are, in fact, biased downward. Eligibility is now associated with 13.9 and 9.9 percentage point increases in the likelihood of having a routine checkup or any physician visit over the past 12 months, respectively (46.3% and 76.9% increases over the baseline percentages). Eligibility is associated with 4.3 and 2.0 percentage point increases in the likelihood that a child has a usual source of general and preventive care, respectively (36.1% and 26.8% increases over the baseline percentages). Thus, when instrumenting for indivual level eligibility using a simulated group rate, I find that eligibility increases the likelihood that children will consume preventive medical care by 25% to 75% depending on the measure of prevention. As in column two, eligibility does not have an impact on the likelihood of having a physician as a usual source of care[10].

Column 1 contains the point estimates for the effect of actual enrollment on utilization of preventive medical care as a comparison to columns 2 and 3. The effects of enrollment on the probabilities of a routine check-up and any physician visit are very similar to the impacts of eligibility. This provides some evidence that there is little difference between

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[10]I include the simulated age/state/year group eligibililty rates as well as the interactions between the rates and time as instruments for individual level eligibility in the first stage. The instruments perform extremely well in predicting individual level eligibility. The coefficients for group eligibility and eligibility-time interactions are large and precisely measured. The partial R squared value is 0.051 and thus, the instruments explain a relatively large percentage of the variance in individual eligibility. The F statistic is large enough to reject the null that the regressors are not jointly significant at the 1% level. I ran tests of the overidentifying restrictions using a Sargan statistic, which has a Chi-squared distribution with the number of degress of freedom equal to the number of instruments minus the number of regressors. The test checks whether the instruments can be excluded from the 2nd stage equation. The null hypothesis is that the instruments can in fact be excluded from the 2nd stage equation and thus are valid. The test did not reject at the 15% significance level. There is therefore, some weak evidence that simulated eligibility can be excluded from the main equation. For each dependent variable, I can reject the null hypothesis at the 1% level that the OLS and 2SLS estimates are equal using a Hausman statistic.

being eligible for and enrolling in public health insurance when it comes to a single routine visit. However, the effect of enrollment on the probabilities of a usual source of preventive care and having a physician as a usual source of preventive care are much larger. This suggests that there are some other unobserved factors mediating the decision to enroll other than simply being eligible.

Table 2.8 provides estimates of the impact of eligibility and enrollment on the utilization of immunizations for children 0-6 years of age, i.e. whether a child has ever had one of six shots, whether they are up-to-date, and whether the shots were administered on time. Table 2.9 provides the same information, however, the source of the immunization information is the parent rather than an official vaccination health card. Eligibility has no effect on the utilization of immunizations with the exception of DTP and polio vaccinations when reported by a parent (Table 2.9). When reported by a parent, eligibility increases the probability that a child has had and is up-to-date for the DTP and polio series by roughly 1-2 percentage points. These changes represent 50% and 20% increases over the baseline percentages for having had any shots and whether they are up-to-date respectively.

The 2SLS results in column 3 report a strong negative relationship between eligibility and the consumption of immunizations. The negative relationship suggests that there is some unobserved factor associated with eligibility that also decreases the likelihood of immunizations. Enrollment does not demonstrate the same negative trend. Column 1 provides evidence that enrollment increases the probability that a child is up-to-date for and received the DTP, polio, and HIB immunizations on time, but no effect on consumption of the other vaccines. As mentioned previously, enrollment has a strong positive effect on the probability of having a usual source of routine care at a physician's office while eligibility does not. A regular source of care at a physician's office could increase the likelihood that a child is up-to-date and on time regarding vaccinations. A usual source of care could be acting as a mediator between eligibility and enrollment to explain the positive effect of enrollment. Without this extra push from enrollment and a physician's office as a usual source of care, the effect of eligibility alone could have no effect or even a negative effect on utilization since public health insurance availability provides a weak price incentive for immunizations (they

are provided free to everyone regardless of insurance status)[11].

The signs of the coefficients on the covariates are listed in Table 2.10. In general the covariates were precisely measured and consistent, with regard to sign, across all specifications and dependent variables. Older children and those in larger families were much less likely to consume preventive medical care. Younger children are more likely to have routine care simply because they are targeted by health professionals for regular well-child checkups. The lower utilization rate in larger families is most likely due to the negative influence of health care on household budgets. For the majority of the dependent variables, gender did not affect utilization. Occasionally male children were slightly more likely to consume preventive care.

Race plays a varying role in the utilization of preventive medical care. Black children and those neither black or white were several percentage points more likely to have had a routine check-up over the past 12 months and have a usual source of preventive care. Conversely, white children were more likely to have seen a health professional in the last 12 months, have a usual source of care, and have a physician's office as a source of general and preventive care. It appears that black families are more likely to consume routine check-ups, controlling for other factors, but less likely to have a physician as source of preventive care. Children born outside the U.S. and those of Hispanic origin were less likely to consume care. The effect is particularly strong for those born outside the U.S., i.e., 10-20% points lower for most measures of preventive care. While those born outside the U.S. are a small percentage of the total population, they have very low utilization rates for the preventive care variables addressed in this study.

Higher levels of maternal education and income were associated with increased utilization of preventive care. Parental education and income are other possible important reasons for

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[11]The first stage in the 2SLS analysis is necessarily different with immunizations as the dependent variables since the sample is children 0-6 years of age rather than 0-18. The instruments do not perform quite as well in predicting individual level eligibility. The coefficients for group eligibility and eligibility-time interactions are smaller, although they are still strong, significant predictors of individual eligibility. The partial R squared value is 0.019 and the F-statistic is large enough to reject the null that the regressors are not jointly significant at the 1% level. The test of the overidentifying restriction did not reject at the 30% significance level. Thus, there is evidence that simulated eligibility can be excluded from the main equation. For each dependent variable (with the exception of MMR in Table 2.9), I can reject the null hypothesis at the 1% level that the OLS and 2SLS estimates are equal using a Hausman statistic.

the lack in take-up of health insurance in the U.S. Even among those eligible for public health insurance, education and income still have strong positive effects on utilization. The positive effect of income is proof that there are still time and travel costs associated with free health care that make families with slightly higher incomes more likely to take advantage of preventive care.

Tables 2.11 and 2.12 contain the coefficients for the interaction effects of eligibility and covariates on two select measures of preventive care - routine check-ups over the past 12 months and usual source of preventive care. The point estimates can be interpreted as the effect that eligibility has among the given group compared to the omitted group, i.e., the relative degree to which the given group takes advantage of eligibility with regard to an increase in prevention. Eligibility increases the probability of having a usual source of preventive care for black children by 7.3 percentage points more than the increase for white children (Table 2.12). Eligibility increases the likelihood of both a routine check-up and a usual source of care by 6.7 and 12.2 more percentage points, respectively, for children neither black or white over white children. Evidence in Table 2.11 suggests that eligibility among Hispanic children and those born outside the U.S. does not impact the probability of a routine check-up as much as it does for their counterparts (6.9 and 12.0 percentage points less for Hispanic children and those born outside the U.S., respectively).

A surprising result is that the increase in the likelihood that a child has a usual source of preventive care from eligibility is lower among children whose mothers have higher levels of education (Table 2.12). In other words, mothers with lower levels of education tend to take advantage of the incentives provided by public health insurance availability to a greater extent than more educated mothers. The proportion of children of highly educated mothers with a usual source of preventive care is over 90% for both those eligible and not eligible for public insurance. It is likely that there is some unobserved trait in the small percentage of highly educated mothers without a source of preventive care for their child that increases the probability of being eligible but lowers the likelihood of utilization. There is no differential impact of education on eligibility by maternal education for the likelihood of a routine check-up over the past 12 months (Table 2.11).

2.7 Discussion

In this paper, I focus on the effect of recent expansions in public health insurance eligibility on preventive medical care. I investigate how eligibility, a policy tool controlled by state governments, influences utilization rather than actual enrollment which is conditional on eligibility but relies on individual choice. Thus, the impact examined in this paper is that of the option of health insurance rather than actually having health insurance. This is a subtle but key difference. I use individual level data and detailed state eligibility criteria to assign eligibility as accurately as possible. Thus I am measuring the actual effect of eligibility rather than, as in some studies, the impact of having a low income in a state and time with generous eligibility criteria.

The findings are within the range of effects found in studies that focus on measures of curative care utilization. The literature finds that eligibility (both recent and earlier Medicaid expansion in the 1980's and 1990's) increases certain measures of curative care consumption from several to over ten percentage points with large jumps in the baseline percentages for those without care. Furthermore, this study supports one of the general findings in Joyce and Racine (2003), i.e., that eligibility has little or no effect on immunizations.

Because certain subpopulations have lower rates of both utilization (Table 2.5) and insurance coverage , it is important to investigate whether eligibility closes or widens this gap. I find that in most cases, eligibility perpetuates the differences in utilization, i.e., those with higher levels of care in the general population tend to also take advantage of eligibility to a greater extent. The best illustration of this behavior is in the difference between those born in and outside of the U.S. Those born inside the U.S. are much more likely to increase their preventive behaviors when made eligible for public insurance. If the incentives provided by public health insurance are not enough to induce changes in preventive medical care consumption for certain groups (and the behavior change is deemed a worthwhile goal), then is important to investigate what barriers remain.

Utilization of the preventive behaviors discussed in this paper will never approach 100%, even under universal eligibility for health insurance. There are other barriers to consumption

such as education and time/travel costs that are not solved by free or near free health insurance. Once the usefullness of eligibility in increasing consumption has been exhausted policy makers must explore these additional barriers to gain any increases in utilization. However, before they do so, they must address whether or not the benefit of the increase in utilization is worth investing the necessary resources. For instance, how great is the benefit of increasing the percentage of children with a yearly routine physician's visit from 90% to 95%? In addition, individuals value future health differently and have varying priorties for how they allocate thier resources to items such as food, education, and health care. The effect of eligibility for public insurance on medical prevention is compelling only if the lack of insurance is a real barrier to to behaviors that promote good health. I argue that routine well-child check-ups and a usual source of care improve the health stock of children by identifying current conditions and reducing the likelihood of future illnesses. Furthermore, I provide strong evidence that eligibility for Medicaid and SCHIP creates an incentive for families to increase routine care. Childhood immunizations are similarly important to individual and public health, however, it appears that eligibility is not enough of an incentive to induce the small percentage of those not receiving the recommended vaccinations to change their behaviors.

Table 2.1: Dependent variables (immunization means), by eligibility and source of information for children 0-7 years of age

Variable	full sample	eligible	not eligible	**difference**
Data source: health card				
observations	12,932	5,719*	5,719*	5,719*
any recorded shots				
DTP	92.55	91.10	94.17	-3.07
polio	89.59	87.79	90.23	-2.44
varicella	27.79	30.38	24.23	6.15
MMR	80.58	75.66	81.03	-4.36
HIB	85.31	83.70	85.48	-1.78
up-to-date				
DTP	78.28	76.68	77.97	-1.29
polio	77.66	76.57	76.70	-0.13
varicella	31.23	35.56	27.54	8.02
MMR	90.59	88.39	91.68	-3.29
HIB	57.17	54.74	56.46	-1.72
received on time				
DTP	44.95	39.16	42.57	-3.41
polio	53.02	48.19	50.21	-2.02
varicella	17.96	20.54	15.89	4.65
MMR	63.52	58.94	59.67	-0.73
HIB	30.15	26.53	28.89	-2.36
Data source: parent/guardian				
observations	23,558	9,330*	9,330*	9,330*
any recorded shots				
DTP	98.40	98.19	97.98	0.21
polio	96.44	95.96	95.52	0.44
varicella	47.66	55.19	46.28	8.91
MMR	96.96	95.88	97.06	-1.18
HIB	90.77	90.26	89.29	0.97
up-to-date				
DTP	91.59	90.80	91.68	-0.88
polio	90.85	89.47	90.36	-0.89
varicella	44.79	52.00	42.96	9.04
MMR	96.57	95.26	96.62	-1.36
HIB	83.16	81.54	82.86	1.32

Note: For columns two and three, the sample is those without a source of private health insurance.

Table 2.2: Dependent variables (immunization means), by *enrollment* (Medicaid or SCHIP) and source of information for children 0-7 years of age

Variable	full sample	enrolled	not enrolled	**eligible-non eligible**
Data source: health card				
observations	12,932	5,719	5,719	5,719
any recorded shots				
DTP	92.55	91.17	94.08	-2.91
polio	89.59	88.59	90.07	-1.48
varicella	27.79	30.91	26.47	4.44
MMR	80.58	75.61	82.84	-7.23
HIB	85.31	85.02	83.28	1.74
up-to-date				
DTP	78.28	77.79	76.04	1.75
polio	77.66	78.92	77.08	1.84
varicella	31.23	36.99	28.95	8.04
MMR	90.59	89.86	90.54	-0.68
HIB	57.17	57.75	49.65	8.10
received on time				
DTP	44.95	42.29	38.83	3.46
polio	53.02	50.77	51.32	-0.55
varicella	17.96	21.72	15.49	6.23
MMR	63.52	63.24	59.31	3.93
HIB	30.15	29.16	24.36	4.80
Data source: parent/guardian				
observations	23,558	9,330	9,330	9,330
any recorded shots				
DTP	98.40	98.10	98.28	-0.18
polio	96.44	95.71	96.88	-1.17
varicella	47.66	57.75	52.40	5.35
MMR	96.96	95.43	97.26	-1.83
HIB	90.77	29.16	24.36	4.80
up-to-date				
DTP	91.59	91.74	91.66	0.08
polio	90.85	90.41	91.11	-0.70
varicella	44.79	54.77	49.21	5.56
MMR	96.57	94.81	96.85	-2.04
HIB	83.16	83.43	83.59	-0.16

Note: For columns two and three, the sample is those without a source of private health insurance.

Table 2.3: Explanatory variables by source of immunization data for children 0-7 years of age

Variable	card	parent	difference	
Gender				
male	51.06	51.66	0.60	
female	48.94	48.34	-0.60	
Race				
white	74.69	73.02	-1.67	***
black	13.19	17.56	4.37	***
other	12.12	9.42	-2.70	***
Hispanic origin	31.57	25.30	-6.27	***
Born in U.S.	94.20	94.28	0.08	
Family size				
two	7.38	8.87	1.49	***
three	29.82	29.19	-0.63	
four	34.75	34.14	-0.61	
five	17.31	17.15	-0.16	
> five	10.74	10.65	-0.09	
Mother's education				
no high school degree	22.29	19.39	-2.90	***
high school degree	25.88	29.57	3.69	***
some college	30.24	30.46	0.22	
BA/BS	15.66	14.60	-1.06	***
advanced degree	5.93	5.98	0.05	
Family income				
$0-20,000	23.01	21.18	-1.83	***
$20,000-$35,000	20.86	19.60	-1.26	***
$35,000-$50,000	21.86	21.38	-0.48	
$50,000-$75,000	14.42	15.39	0.97	***
over $75,000	19.85	22.25	2.40	***
Parent reported health status				
excellent	56.42	52.73	3.69	***
very good	26.97	29.08	-2.11	***
good	14.62	16.16	-1.54	***
fair	1.73	1.76	-0.03	
poor	0.26	0.26	0.00	

***indicates significance at the 1% level.

Table 2.4: Dependent variables (preventive physician visit means), by eligibility and enrollment (Medicaid or SCHIP) for children 0-18 years of age

Variable	full sample	eligible	not eligible	**difference**
routine checkup last 12 months	72.18	70.02	66.32	3.70
seen health professional last 12m	88.96	87.14	84.72	2.42
usual source of care	93.30	88.82	87.80	1.02
usual source is a physician's office	76.46	58.23	64.48	-4.25
usual source of preventive care	95.74	92.55	91.61	0.94
usual source is a physician's office	75.57	57.13	63.63	-6.50

Variable	full sample	enrolled	not enrolled	**difference**
routine checkup last 12 months	72.18	76.76	57.16	19.60
seen health professional last 12m	88.96	92.10	76.81	15.29
usual source of care	93.30	95.49	76.88	18.61
usual source is a physician's office	76.46	61.07	51.93	9.14
usual source of preventive care	95.74	97.55	82.97	14.58
usual source is a physician's office	75.57	60.63	50.05	10.58

Note: For columns two and three, the sample is those without a source of private health insurance.

Table 2.5: Subset of dependent variables, by selected explanatory variables

| | Routine checkup | Usual source of | Usual source is a | Vaccine received on time | | | | |
| | last 12 months | preventive care | physician's office | DTP | polio | varicella | MMR | HIB |
Explanatory variables								
Race								
white	70.93	95.73	77.98	46.65	54.72	17.87	64.91	31.69
black	77.72	96.79	69.54	36.05	43.58	17.98	54.66	22.57
other	72.36	93.93	66.95	44.32	52.94	18.56	64.68	28.99
difference (white-black)	-6.79	-1.06	8.44	10.60	11.14	-0.11	10.25	9.12
difference (white-other)	-1.43	1.80	11.03	2.33	1.78	-0.69	0.23	2.70
difference (black-other)	5.36	2.86	2.59	-8.27	-9.36	-0.58	-10.02	-6.42
Hispanic origin								
yes	67.07	91.03	64.05	42.20	53.70	19.62	63.69	27.70
no	74.07	97.48	79.55	46.37	52.65	17.20	63.44	31.42
difference (yes-no)	-7.00	-6.45	-15.50	-4.17	1.05	2.42	0.25	-3.72
Born in U.S.								
yes	73.19	96.69	76.55	45.77	53.16	18.55	64.97	31.10
no	55.68	80.16	56.25	23.94	49.26	9.54	42.71	5.93
difference (yes-no)	17.51	16.53	20.30	21.83	3.90	9.01	22.26	25.17
Mother's education								
no high school degree	63.26	90.06	57.83	38.56	48.27	17.31	59.41	24.97
high school degree	70.77	96.54	75.60	43.05	50.97	15.85	61.64	27.70
some college	75.68	97.69	80.17	47.59	56.60	18.67	64.49	32.50
BA/BS	78.88	98.65	86.50	52.92	57.27	21.77	71.72	36.48
advanced degree	82.90	98.96	89.16	54.43	59.37	21.08	73.81	39.60
difference (HS-No HS deg)	-7.51	-6.48	-17.77	-4.49	-2.70	1.46	-2.23	-2.73
difference (BA/BS-HS deg)	-8.11	-2.11	-10.90	-9.87	-6.30	-5.92	-10.08	-8.78
difference (Adv-BA/BS deg)	-4.02	-0.31	-2.66	-1.51	-2.10	0.69	-2.09	-3.12

Note: The sample for columns 4-8 is children 0-7 years of age.

Table 2.6: Percentage with a **checkup in the last 12 months by selected explanatory variables and eligibility for children 0-18 years of age**

Explanatory variable	eligible	not eligible	**difference**
Race			
white	67.55	64.76	2.79
black	76.26	74.34	1.92
difference (white-black)	-8.71	-9.58	**0.87**
Hispanic origin			
yes	65.16	60.57	4.59
no	72.14	67.69	4.45
difference (yes-no)	-6.98	-7.12	**0.14**
Born in U.S.			
yes	71.70	67.51	4.19
no	50.25	51.21	-0.96
difference (yes-no)	21.45	16.30	**5.15**
Mother's education			
no high school degree	65.80	57.43	8.37
high school degree	72.95	65.89	7.06
some college	75.90	70.37	5.53
BA/BS	71.97	70.49	1.48
advanced degree	77.55	79.38	-1.83
difference (HS-No HS deg)	7.15	8.46	**-1.31**
difference (some col-HS deg)	2.95	4.48	**-1.53**

Note: The sample is those without a source of private health insurance.

Table 2.7: Coefficients for impact of public insurance on preventive physician visits

dependent variables	enrolled (OLS)		eligible (OLS)		eligible (2SLS)	
routine checkup last 12 months	0.138 (0.006)	***	0.040 (0.006)	***	0.139 (0.030)	***
seen health professional last 12m	0.109 (0.004)	***	0.019 (0.004)	***	0.099 (0.020)	***
usual source of care	0.119 (0.003)	***	0.013 (0.003)	***	0.043 (0.015)	**
usual source is a physician's office	0.010 (0.006)	***	-0.002 (0.005)		0.030 (0.029)	
usual source of preventive care	0.089 (0.002)	***	0.008 (0.002)	***	0.020 (0.010)	**
usual source is a physician's office	0.111 (0.005)	***	-0.003 (0.005)		0.042 (0.029)	
fixed effects						
state	Yes		Yes		Yes	
time	Yes		Yes		Yes	
age	Yes		Yes		Yes	

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

Table 2.8: Coefficients for impact of public insurance on immunizations (reported on health card)

dependent variables	enrolled (OLS)		eligible (OLS)		eligible (2SLS)	
any recorded shots						
DTP	-0.003 (0.008)		-0.014 (0.010)		-0.216 (0.058)	***
polio	0.013 (0.010)		-0.013 (0.011)		-0.134 (0.068)	*
varicella	0.028 (0.014)	**	0.009 (0.016)		0.085 (0.095)	
MMR	0.009 (0.011)		0.002 (0.012)		-0.401 (0.076)	***
HIB	0.026 (0.012)	**	-0.009 (0.013)		-0.202 (0.078)	**
up-to-date						
DTP	0.028 (0.014)	**	-0.023 (0.016)		-0.125 (0.093)	
polio	0.051 (0.013)	***	-0.015 (0.016)		-0.241 (0.097)	**
varicella	0.023 (0.018)		-0.009 (0.020)		-0.266 (0.127)	**
MMR	0.005 (0.011)		0.004 (0.012)		-0.347 (0.088)	***
HIB	0.048 (0.017)	**	-0.005 (0.019)		-0.039 (0.111)	
received on time						
DTP	0.029 (0.017)	*	-0.034 (0.020)	*	-0.132 (0.114)	
polio	0.029 (0.017)	*	-0.032 (0.022)		-0.263 (0.115)	**
varicella	0.000 (0.017)		-0.025 (0.019)		0.099 (0.120)	
MMR	-0.023 (0.017)		0.008 (0.020)		-0.250 (0.139)	*
HIB	0.018 (0.016)		-0.014 (0.018)		-0.048 (0.106)	
fixed effects						
state	Yes		Yes		Yes	
time	Yes		Yes		Yes	
age	Yes		Yes		Yes	

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

Table 2.9: Coefficients for impact of public insurance on immunizations (reported by parent)

dependent variables	enrolled (OLS)		eligible (OLS)		eligible (2SLS)	
any recorded shots						
DTP	0.010 (0.004)	**	0.009 (0.004)	**	0.051 (0.037)	
polio	0.011 (0.006)	*	0.014 (0.007)	**	0.089 (0.052)	*
varicella	0.009 (0.015)		-0.017 (0.016)		0.047 (0.119)	
MMR	0.000 (0.008)		-0.007 (0.008)		-0.010 (0.062)	*
HIB	0.013 (0.010)		0.013 (0.010)		-0.163 (0.078)	**
up-to-date						
DTP	0.016 (0.010)	*	0.018 (0.011)	*	-0.061 (0.076)	
polio	0.012 (0.010)		0.020 (0.010)	**	-0.006 (0.081)	
varicella	0.010 (0.015)		-0.008 (0.017)		0.073 (0.121)	
MMR	0.001 (0.008)		-0.005 (0.009)		-0.132 (0.0689)	*
HIB	0.021 (0.013)	*	0.004 (0.015)		-0.267 (0.106)	**
fixed effects						
state	Yes		Yes		Yes	
time	Yes		Yes		Yes	
age	Yes		Yes		Yes	

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

Table 2.10: Signs of coefficients for covariates

covariate (omitted group)	enrolled (OLS)	eligible (OLS)	eligible (2SLS)
gender (female)	0	+/0	+/0
age (youngest group)	-	-	-
race - black (white)	+/-	+/-	+/-/0
race - other (white)	+/-	+/-	+/-/0
Hispanic origin	-	-	-/0
born in U.S.	+	+	+
family size (small size)	-	-	-
mother's education (low level)	+	+	+
family income (low income)	+	+	+
fixed effects			
state	Yes	Yes	Yes
time	Yes	Yes	Yes
age	Yes	Yes	Yes

Table 2.11: Coefficients on covariates interacted with eligibility for whether child has had a *routine physician visit in past 12 months* as dependent variable

interaction variable (omitted group)	enrolled (OLS)		eligible (OLS)		eligible (2SLS)	
race (white)						
black	-0.011	(0.009)	-0.001	(0.009)	-0.036	(0.043)
other	0.040	(0.012) ***	0.043	(0.012) ***	0.067	(0.054)
Hispanic origin	-0.015	(0.009) *	-0.013	(0.010)	-0.069	(0.037) *
born in U.S.	0.042	(0.011) ***	0.072	(0.016) ***	0.120	(0.058) **
family size (smallest size)						
three	-0.005	(0.012)	-0.002	(0.013)	0.040	(0.050)
four	-0.006	(0.011)	-0.003	(0.013)	0.006	(0.049)
five	-0.021	(0.013) *	-0.018	(0.015)	-0.038	(0.051)
> five	-0.006	(0.014)	-0.001	(0.016)	-0.025	(0.055)
mother's education (lowest level)						
high school degree	0.007	(0.010)	0.009	(0.011)	-0.050	(0.034)
some college	-0.006	(0.010)	-0.007	(0.011)	-0.091	(0.039) **
BA/BS	-0.008	(0.015)	-0.014	(0.015)	-0.022	(0.098)
advanced degree	-0.020	(0.026)	-0.026	(0.027)	-0.018	(0.202)

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

73

Table 2.12: Coefficients on covariates interacted with eligibility for whether child has a *usual source of preventive care* as dependent variable

interaction variable (omitted group)	enrolled (OLS)		eligible (OLS)		eligible (2SLS)	
race (white)						
black	0.010	(0.003) ***	0.022	(0.004) ***	0.073	(0.021) ***
other	0.026	(0.006) ***	0.034	(0.006) ***	0.122	(0.027) ***
Hispanic origin	-0.024	(0.004) ***	-0.015	(0.005) ***	0.025	(0.018)
born in U.S.	0.055	(0.005) ***	0.115	(0.008) ***	0.029	(0.029)
family size (smallest size)						
three	-0.031	(0.006) ***	-0.009	(0.006) *	-0.012	(0.025)
four	-0.033	(0.005) ***	-0.010	(0.006) *	-0.044	(0.024) *
five	-0.037	(0.006) ***	-0.014	(0.008) *	-0.031	(0.025)
> five	-0.038	(0.007) ***	-0.012	(0.008) *	0.002	(0.027)
mother's education (lowest level)						
high school degree	-0.018	(0.005) ***	-0.005	(0.005)	-0.044	(0.017) **
some college	-0.013	(0.005) **	-0.003	(0.005)	-0.091	(0.019) ***
BA/BS	-0.019	(0.007) **	-0.010	(0.007)	-0.162	(0.049) ***
advanced degree	-0.014	(0.013)	-0.004	(0.013)	-0.262	(0.100) ***

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

Appendix A

Appendix for Chapter Two

Table A.1: CDC recommended immunization schedule for children aged 0-6 years

Vaccine	2 months	4 mon.	6 mon.	12 mon.	15 mon.	18 mon.	19-23 mon.	2-3 years	4-6 years
Diphtheria, Tetanus, Pertussis	DTP	DTP	DTP		– DTP –				DTP
Haemophilus influenzae type b	HIB	HIB	HIB	HIB					
Inactivated Poliovirus	POL	POL	——— POL ———						POL
Measles, Mumps, Rubella				– MMR –					MMR
Varicella				– VAR –					VAR

Table A.2: Summary Means - dependent variables - immunizations, for children 0-7 years of age

Variable	1997-2003	1997	1998	1999	2000	2001	2002	2003
health card								
observations	12,932	2,216	2,107	1,813	1,882	19,83	1,578	1,353
any recorded shots								
DTP	92.55	95.76	94.83	94.10	90.91	90.47	89.73	90.32
polio	89.59	95.13	92.69	91.78	86.45	86.74	85.87	85.66
varicella	27.79	10.88	16.74	24.00	30.03	35.65	40.26	45.76
MMR	80.58	84.53	83.00	81.58	77.04	79.29	78.35	79.03
HIB	85.31	88.40	85.95	85.71	81.99	83.31	85.17	86.40
up-to-date								
DTP	78.28	83.70	78.08	77.77	74.82	77.67	77.59	76.90
polio	77.66	87.91	80.75	76.59	71.13	74.48	75.00	74.27
varicella	31.23	12.22	18.68	26.89	33.77	39.83	45.39	51.33
MMR	90.59	95.27	93.07	91.85	86.60	89.02	88.32	88.59
HIB	57.17	58.37	57.13	57.30	54.23	56.26	57.86	59.68
received on time								
DTP	44.95	43.67	42.58	43.74	43.79	46.78	46.32	49.77
polio	53.02	55.24	53.56	51.27	50.30	53.76	52.89	54.05
varicella	17.96	5.90	9.88	14.88	18.40	23.12	27.53	32.65
MMR	63.52	62.41	62.39	62.66	62.00	63.50	64.18	69.34
HIB	30.15	29.67	29.49	28.71	28.86	29.85	31.83	34.22
parent/guardian								
observations	23,558	3,651	3,332	3,245	3,439	3,407	3,313	3,171
any recorded shots								
DTP	98.40	98.19	98.70	98.28	98.91	98.30	97.99	98.39
polio	96.44	97.77	97.22	96.58	96.49	96.18	95.22	95.38
varicella	47.66	33.90	36.05	41.19	47.17	54.26	60.00	63.05
MMR	96.96	96.49	96.86	96.82	97.01	97.20	97.17	97.23
HIB	90.77	89.65	90.91	91.89	90.55	89.37	90.97	92.25
up-to-date								
DTP	91.59	91.14	92.39	92.20	92.63	90.92	89.98	91.85
polio	90.85	92.29	92.16	91.56	91.53	90.46	88.90	88.58
varicella	44.79	31.91	33.33	38.71	44.58	51.46	56.91	59.82
MMR	96.57	96.12	96.47	96.44	96.61	96.84	96.75	96.79
HIB	83.16	81.74	83.69	85.38	83.97	81.82	81.57	84.11

Table A.3: Summary Means - explanatory variables, for children 0-18 years of age

Variable	97-03	1997	1998	1999	2000	2001	2002	2003
observations	100,402	15,244	14,619	14,217	14,711	14,766	13,570	13,275
gender								
male	51.39	51.17	51.54	51.17	51.75	51.43	51.40	51.30
female	48.61	48.83	48.46	48.83	48.25	48.57	48.60	48.70
age								
0-6	39.21	40.26	39.46	39.31	38.97	39.08	38.87	38.58
7-12	31.00	30.45	31.36	31.25	30.72	31.08	31.01	31.11
13-17	29.79	29.29	29.18	29.44	30.31	29.84	30.12	30.31
race								
white	76.66	77.99	77.55	76.82	75.90	76.55	76.46	75.37
black	14.99	15.19	15.08	14.98	14.98	14.84	14.67	15.21
other	8.35	6.81	7.37	8.20	9.11	8.60	8.86	9.42
Hispanic origin	14.92	13.65	13.74	14.51	14.82	15.35	15.81	16.50
born in U.S.	95.46	95.57	95.75	96.02	94.90	95.00	95.48	95.53
family size								
two	8.34	8.53	8.66	7.84	8.04	8.49	8.17	8.66
three	30.42	30.48	31.16	30.86	30.19	30.17	30.16	29.93
four	35.67	35.56	35.57	36.05	35.72	35.50	36.16	35.14
five	16.44	16.53	15.89	16.27	16.56	16.66	16.37	16.77
> five	9.13	8.91	8.72	8.98	9.49	9.18	9.13	9.50
mother's education								
no high school degree	14.67	15.80	14.29	14.35	15.12	14.35	14.15	14.71
high school degree	29.11	30.62	30.21	29.67	29.39	28.42	28.40	27.17
some college	31.75	31.43	31.65	32.05	31.06	31.94	32.40	31.70
BA/BS	17.34	15.27	16.80	17.34	17.34	18.03	17.52	18.91
advanced degree	7.13	6.87	7.06	6.59	7.09	7.26	7.53	7.50
family income								
$0-20,000	21.70	24.74	22.45	20.95	20.90	20.54	21.29	21.08
$20,000-$35,000	18.75	19.23	19.05	18.36	18.97	17.84	18.41	19.41
$35,000-$50,000	16.79	18.11	17.27	17.23	16.55	17.21	15.98	15.23
$50,000-$75,000	20.47	20.35	20.63	21.01	21.47	20.17	20.11	19.57
over $75,000	22.29	17.58	20.60	22.46	22.11	24.23	24.21	24.71
reported health status								
excellent	53.07	52.63	53.77	53.70	52.25	53.47	53.22	52.44
very good	28.65	28.17	28.35	28.62	29.12	28.60	28.63	29.13
good	16.29	16.96	15.88	15.90	16.75	15.98	15.97	16.54
fair	1.73	1.89	1.73	1.62	1.61	1.71	1.90	1.68
poor	0.26	0.35	0.27	0.16	0.27	0.24	0.27	0.22

Table A.4: Summary Means - Health insurance coverage, for children 0-18 years of age

Variable	1997-2003	1997	1998	1999	2000	2001	2002	2003
observations	100,402	15,244	14,619	14,217	14,711	14,766	13,570	13,275
eligible (Medicaid or SCHIP)	32.78	20.34	25.13	30.13	35.39	38.22	39.76	40.03
enrolled (Medicaid or SCHIP)	20.21	17.63	16.68	17.76	18.93	20.79	24.39	26.14
Medicaid	17.77	17.63	16.68	16.89	16.91	16.76	19.62	20.24
SCHIP	3.52	NA	NA	0.94	2.07	4.06	4.84	5.97
private	63.28	63.76	65.28	65.91	64.12	63.63	60.45	59.28
any coverage (public or private)	86.49	85.52	85.05	86.00	85.49	87.44	87.60	88.62

79

Table A.5: Summary Means - dependent variables - preventive physician visits, for children of 0-18 years of age

Variable	1997-2003	1997	1998	1999	2000	2001	2002	2003
observations	91,633	14,166	13,510	12,733	13,263	13,472	12,367	12,122
routine checkup last 12 months	72.18	74.27	73.69	72.63	71.08	70.52	71.46	71.40
seen health professional last 12m	88.96	89.90	89.49	87.59	88.44	88.97	89.39	89.63
usual source of care	93.30	93.09	93.00	92.33	92.66	93.87	93.72	94.50
usual source is a physician's office	76.46	75.00	76.93	76.22	77.21	76.22	77.76	76.01
usual source of preventive care	95.74	96.16	95.94	94.94	95.42	95.75	95.58	96.33
usual source is a physician's office	75.57	74.17	75.79	75.33	76.26	75.58	77.01	75.00

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