Chapter 5: Child Health Interventions
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## I. Introduction

The connection between poor health and low income has long been recognized. Along almost any dimension, the poor suffer from worse health. Even among children, who are generally healthy, poorer individuals report worse health outcomes, and this relationship only sharpens in adulthood. According to data from the National Health Interview Survey, while nearly 90 percent of non-poor children report very good or excellent health, fewer than 70 percent of poor children do, and among adults 45-64 the percentages range from slightly over 60 percent among the non-poor to under 30 percent among the poor (see Figure 1). Similar patterns hold for severe health problems, with significantly more poor adults and children reporting a condition that limits their daily activities (see Figure 2). From the cradle to the grave, poverty and poor health are closely entwined.

Determining whether interventions targeting childhood health are likely to have a beneficial long-run effect on a child's life chances is more difficult, however.[1] Perhaps even more than most individual characteristics, health is a "black box"–a complicated and only partially understood function of genes, other biological factors, environment, and behavior. Moreover, health interacts with various other inputs provided by the child's family or society such as income and education to determine a child's life chances. While there is evidence

[1]See Currie (2008) for a thoughtful survey of this question and of the question whether parental circumstances affect child health.

linking poor health in childhood to worse adult non-health outcomes, the direction of causality is unclear. For example, Case, Lubotsky, and Paxson (2002) find that having one of a set of identified medical conditions is associated with fewer years of completed schooling, but that the association is smaller for children with higher incomes. While this evidence is consistent with poor health having a causal effect on education (perhaps through days of school missed or difficulty studying), it is also consistent with poverty or some other related factor causing both the poor health and the reduction in schooling, with little or no direct link between health and education.

In Figure 3 I outline the possible causal links between child health and adult income, as well as the possible intermediaries by which child health may be affected. There are three main pathways by which child health can affect adult income. Probably the most direct way is through the impact child health has on adult health, which in turn will affect adult earnings capacity. However, child health may also affect adult earnings even if the child does not become an unhealthy adult, if the child's ill health affects the quantity or quality of education the child receives. If, for example, the child misses a great deal of school or is unable to learn while in school because of health problems, the child will have accumulated lower amounts of human capital than he or she would have otherwise. Finally, child health may affect adult earnings more indirectly, through the income the child's family has available. For example, having a severely or chronically ill child to take care of may reduce the amount of work the child's parent is able to do. Income in childhood in turn affects childhood health–by enabling the purchase of health insurance/health care, better environment, and other goods such as nutritious food.

In this chapter, I present and analyze evidence on whether and how child health

interventions affect adult labor market outcomes. This evidence has two parts. First, the interventions must actually affect child health in a measurable way. Second, there must be a link between that improvement in health and adult labor market outcomes. This link may be direct (e.g. the improvement in health permits higher levels of earnings) or indirect (e.g. the improvement in health permits greater levels of education). It is important to emphasize that even if an intervention does not show measurable effects on factors affecting adult labor market outcomes, it may have benefits beyond the scope of this book. I discuss such benefits briefly at the end of the chapter.

To keep the length of this chapter manageable, I focus on major health-related interventions and on a set of conditions that have relatively high prevalence in the US population. I do not focus on interventions that have targeted less prevalent (though possibly more severe) conditions. For example, folic acid fortification of grain products, which targeted neural tube defects (such as spina bifida and anencephaly) was associated with a reduction in neural tube defect prevalence from 0.0378 percent of all births to 0.0305 percent (Honein et al. 2001). Prevalence rates such as these are so small they preclude sizeable impacts on poverty reduction at the population level. In addition, I do not survey the literature on successful interventions from the past that have resulted in the near complete elimination of particular diseases or conditions in the US (such as public health infrastructure to provide clean water and sanitation or widespread immunization for particular diseases). Finally, I do not survey the quite substantial literature on the relationships between child health and adult functioning in developing country contexts, since the health problems faced by children in developing countries are in most cases more severe than the health problems of US children.

I begin this survey with a discussion of interventions intended to promote general health among children, including expanded access to health insurance through Medicaid and the State Children's Health Insurance Program (SCHIP) and nutritional supplements to pregnant women and infants through the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC). I then move on to discuss a few specific conditions with relatively high prevalence in the population that have been the focus both of interventions and research on the links between health and adult outcomes. These conditions are: asthma, mental health (particularly attention deficit hyperactivity disorder), dental health, obesity/overweight, and exposure to environmental toxins (including elevated blood lead levels and air pollution). While some of these conditions are also targeted by the general health care interventions of Medicaid and SCHIP, they are sufficiently important to merit separate discussion.

## II. Interventions Targeting General Health

### *A. Medicaid and the State Children's Health Insurance Program*

In terms of number of children affected, the most significant intervention targeting children's health since the mid-1960s has been the Medicaid program. It is a joint federal-state program; generally, the federal government sets mandatory and optional provisions within which states must act. Initially Medicaid covered two groups of low-income individuals: recipients of Aid to Families with Dependent Children (AFDC) and low-income elderly (for services not covered by Medicare, such as some out of pocket expenditures and nursing home care). A few years later, when the Supplemental Security Income (SSI) program was created to aid poor

disabled individuals, Medicaid coverage was required for SSI recipients as well.[2]

The services Medicaid provides to covered children include a rich set of preventative and curative care services under the Early and Periodic Screening, Diagnostic, and Treatment (EPSDT) requirement. However, there were two factors that limited the potential effectiveness of the program. First, by tying Medicaid eligibility for children almost entirely to eligibility for AFDC, eligibility for the program was limited to the very poorest children, and moreover only to children in single parent families. Second, the program has typically reimbursed doctors at rates that are well below the rates paid by private insurers, which has led to very low participation rates among physicians, particularly in some areas and among some specialties.

Beginning in the mid- to late-1980s, Congress passed a series of laws intended to address the first issue–that many poor children were ineligible for the program. These laws substantially reduced the link between Medicaid eligibility and AFDC eligibility by extending Medicaid coverage to children and pregnant women in families with incomes above the AFDC thresholds (generally to levels between 100 percent and 185 percent of the federal poverty level, depending on the state and age of the child). Following these expansions of Medicaid, in 1997 a new program was passed that further expanded access to health insurance for low income children. The State Children's Health Insurance Program (SCHIP) is also a state-federal partnership, although it was designed to give states somewhat more flexibility in designing their programs. States could either expand Medicaid eligibility or create a new program for children who did not qualify for Medicaid. In either case, income eligibility limits moved further up the income

[2]See http://www.kff.org/medicaid/timeline/pf_entire.htm for further information on the history of the Medicaid program.

distribution, with the eligibility limits ranging between one and four times the poverty line depending on the state. In total, expansions of Medicaid eligibility and implementation of SCHIP increased eligibility rates for public insurance, from about 16 percent of all children prior to the expansions to roughly 40 percent of all children.[3]

*B. The Impact of Medicaid Expansions and SCHIP for Children*

Along with the increase in access to additional low-income children, the Medicaid expansions and SCHIP implementation have offered researchers an opportunity to assess the impact of public health insurance for poor children. Prior to the expansions, Medicaid was linked so tightly to receipt of AFDC and SSI that it was not possible to distinguish between the effects of Medicaid and the effects of the other programs on children's outcomes. Moreover, there was no variation in eligibility along any dimension that was not plausibly related to outcomes directly. By contrast, the form of the Medicaid expansions provided useful variation along the dimensions of age, state, and time, permitting researchers to examine the impact of Medicaid and SCHIP using quasi-experimental approaches.[4]

[3]For the most part, the second issue–that of physician reimbursement and participation–has remained largely unaddressed, although in designing their SCHIP programs some states have chosen to provide a somewhat less complete benefits package while attempting to reimburse physicians at closer to private market rates.

[4]A particularly good example of exogenous variation in the expansions is the expansion enacted in the Omnibus Budget Reconciliation Act of 1990, in which for historical reasons the essentially random birth date cutoff for eligibility (only children born after September 30, 1983 were eligible) was chosen.

An obvious first question to ask when examining the impact of a health insurance expansion is whether in fact any additional children gained insurance as a result of the policy. There has been a fairly substantial literature on this question, with some debate over the relative importance of "crowding out"–the phenomenon whereby children who already have the option of health insurance through a private source enroll in Medicaid instead. Although researchers remain divided on the relative importance of crowding out versus take-up behavior among the uninsured, there is consensus that the expansions did indeed increase health insurance coverage among low-income children. Researchers have generally found around an 8-10 percentage point reduction in uninsurance due to the Medicaid expansions, and a 5-8 percentage point reduction in uninsurance due to SCHIP (see Table 1). This increase in insurance coverage rates largely occurred for children with family incomes between 30 and 150 percent of the federal poverty level, as can be seen in an updated version of Figure 1 from Card and Shore-Sheppard (2004) (see Figure 4).

As the intervention represented by Medicaid and SCHIP is to provide health insurance, rather than health per se, it is worth considering what having health insurance may do to improve a child's chances of being out of poverty in adulthood. Conceptually, health insurance may work to improve the health of the child, through preventative care, early detection of problems, and access to treatment. Indeed, several of the interventions discussed later in the sections on specific health conditions require access to health care, which is made easier with health insurance. Health insurance is also likely to play a financial role, protecting income and assets against the risk of bad health shocks. To the extent that family income in childhood reduces the chance a child will be poor in adulthood, the financial protection afforded by health insurance

may be important.

In a useful survey of the literature on the relationship between health insurance and health, Levy and Meltzer (2004) show that while there are a wealth of studies on this relationship, the bulk of them are purely observational, suggesting a positive association but not demonstrating a causal relationship. The evidence from the smaller number of quasi-experimental and truly experimental (i.e. the RAND Health Insurance Experiment) studies is somewhat mixed, but in general Levy and Meltzer conclude that vulnerable populations (such as infants, children, and low-income individuals) have the most to gain from having health insurance and that they do indeed benefit. Research on the Medicaid expansions (some of which is surveyed by Levy and Meltzer) bears this out.

One mechanism by which health insurance can improve health is by improving access to health care. Since all children are supposed to have at least one visit to the doctor per year, one measure of access is whether expanded health insurance coverage increased the fraction of children who visited the doctor at least once. By this measure, the Medicaid expansions and SCHIP did indeed improve access. Estimates of the impact of the expansions range from a 1 percentage point increase to a 9 percent point increase in the fraction of children with at least one visit to the doctor in the past year, depending on which expansion is being considered (see Table 1). The values in the upper range of these estimates are fairly large, suggesting that making a child eligible for Medicaid lowers the probability the child goes without a visit by almost half. However, it is worth noting that the standard errors on these estimates are also fairly large, enabling the researchers to rule out no effect, but leaving a wide range of possible effects. Other measures of utilization of care increased as well. In particular, hospital use increased among

children made eligible for the expansions, with the existing evidence suggesting that the Medicaid expansions led to an increase in overall hospitalization rates but a reduction in ambulatory care sensitive hospitalizations (see Table 1).

Ideally, increased access to care would lead to improved health both in the short run and in the longer run. To date there has been little research on whether this is the case, largely because of a lack of data available to answer the question. Since incidence rates of even relatively widespread conditions and diseases are fairly low among young children, it is difficult to detect effects in the sample sizes available in national survey data. Currie and Gruber (1996a) examine child mortality in the US vital statistics data, which has the advantage of being calculated from the universe of US death certificates. They find that the child mortality rate fell in the wake of the Medicaid expansions, with a reduction of 0.13 percentage points in mortality for every 10 percentage point increase in Medicaid eligibility. While this estimate is fairly imprecisely measured, it does indicate that there was an effect of Medicaid on child health. This conclusion is reinforced by the fact that Currie and Gruber find no evidence of an effect on deaths from "external causes" (accidents, homicides, suicides, etc.) but do find an effect on deaths from "internal causes." Nevertheless, further examination of the impact of the expansions on short run and long run health outcomes is needed to more fully assess the impact of the expansions for children.

### *C. The Impact of Medicaid Expansions for Pregnant Women and Infants*

One question that has received somewhat more attention is whether the expansions for pregnant women and infants have improved infant health outcomes. While existing research

generally indicates that the expansions increased prenatal care use or its adequacy[5] (see Table 2), the evidence for an effect on infant health outcomes is much weaker. Probably the strongest evidence for an effect on infant health comes from Currie and Gruber (1996b). Using vital statistics data on the fraction of births that are low birth weight (LBW) and the infant mortality rate by state and year, they find evidence both for a reduction in low birth weight incidence and a reduction in infant mortality. However, these reductions appear only to come from the earliest expansions that were aimed at women well below the poverty line; later expansions aimed at women with incomes as high as the poverty line or slightly higher show no statistically significant effect. A series of state-level case studies (see Table 2) find similarly equivocal results, as does a later study by Currie and Grogger (2002). Overall, the effects of expanded access to Medicaid for pregnant women on infant health appear to be weakly positive, though the results vary depending on the group targeted and the outcome studied.

In considering whether the Medicaid expansions for pregnant women and infants are likely to affect poverty, additional information is needed beyond the expansions' impacts on prenatal care and infant health. In particular, a link between infant health and long run outcomes must be established. The key issue in establishing this link is determining whether a causal relationship can be shown. In the case of birth weight, such a causal relationship appears to exist. The bulk of the literature examining this causal relationship relies on within-twin variation in birth weight. Most studies in this literature find that increases in birth weight lead to small but statistically significant increases in outcomes such as education, IQ scores, and earnings (see

[5] "Adequate" prenatal care is care that begins early in the pregnancy and continues for a minimum number of visits.

Table 2).[6]

When considering the results from twin studies such as these, two caveats must be kept in mind. First, differences in birth weight within a twin pair cannot be due to preterm delivery, since twins have the same gestational length. Instead, they must arise from differences in fetal growth rates. These differences are believed to arise primarily because of unequal nutritional intake. Therefore, if the reason for low birth weight matters in the effect of low birth weight on long run outcomes, the results from the twin studies cannot necessarily be extrapolated more broadly. The results of Almond, Chay, and Lee's (2005) study of short run effects of low birth weight suggest that the reason for low birth weight may indeed matter. They examine two different sources of variation in birth weight–that arising from within-twin (and hence nutritional) variation, and that arising from maternal smoking behavior among singleton births–and find substantial differences in outcomes both across these two sources of variation and when compared to typical cross-sectional regression estimates. A second, and related, caveat is that the effects of variation in birth weight between twins may not accurately predict the effects of variation in birth weight across singleton births if parents of twins behave differently as a result of the difference in birth weight. If, for example, parents favor the heavier twin, then part of the estimated effect of birth weight is actually an effect of differential investment in childhood. In addition, twins tend to be lighter at birth, often substantially so, than singletons, so a given

[6] An exception is the study by Berhman and Rosenzweig (2004), which finds effects that are much larger. However, Behrman and Rosenzweig use birth weight divided by gestational length as their measure of infant health, which as Royer (2009) points out may lead to biased estimates since gestational length is measured with considerable error.

reduction in birth weight for a twin may have a larger effect than the same reduction from a higher initial level for a singleton. For example, a 100-gram reduction in birth weight may matter less for a singleton weighing 3500 grams than for a twin weighing 2500 grams.

Despite these caveats, based on twin studies there seems to be a clear link between birth weight and adult outcomes that may plausibly affect poverty. The question then becomes how birth weight can be affected. Biologically, there are two basic mechanisms for increasing birth weight: extending the length of gestation and increasing the weight of the infant conditional on gestational length. The intervention discussed thus far, public provision of health insurance through Medicaid (and SCHIP) is intended to provide access to prenatal care, which arguably could affect both gestational length (for example through the provision of antibiotics to treat genitourinary tract infections, which have been shown to increase the probability of preterm birth) and weight conditional on gestation (for example through smoking cessation interventions). An important point made by Currie and Grogger (2002) is that little is known about the content of prenatal care. The available measures of prenatal care "adequacy" tend to be quantitative (involving number of visits and spacing of visits) so wide variability in content and quality of care may exist even among care generally counted as adequate. This is one of the problems researchers have faced as they have tried to determine whether prenatal care does in fact lead to improved birth outcomes. Another problem is that the evidence linking prenatal care and birth outcomes is generally observational, so that studies of the effectiveness of prenatal care are hampered by the possibility of selection bias. Selection bias in this context may work in two directions: women who are more health conscious and likely to take better care of themselves even in the absence of prenatal care are also more likely to obtain prenatal care, leading to

overestimates of the effectiveness of prenatal care; and women who have high risk pregnancies are also more likely to obtain prenatal care, leading to underestimates of the effectiveness of prenatal care.

Random or quasi-random variation in prenatal care is difficult to obtain. Because of ethical concerns about denying possibly beneficial care to pregnant women, there have been only a handful of randomized trials, and these trials tested typically recommended levels of prenatal care against a regimen of somewhat fewer visits for women identified as having low risk pregnancies (see Sikorski et al. (1996), McDuffie et al. (1997), Clement et al. (1999), and Villar et al. (2001)). Consequently, none of the trials showed any effect of the reduced number of visits on infant health. Evans and Lien (2005) exploit variation in prenatal care access for a somewhat higher risk sample. In 1992, there was a month-long public transit strike in Pittsburgh, which Evans and Lien argue caused exogenous variation in access to prenatal care. Evans and Lien find evidence that black, inner-city residents lost on average one-half a prenatal visit due to the strike. This reduction appears to be statistically significantly correlated with an increase in maternal smoking behavior, but Evans and Lien are unable to identify precise estimates of any other outcome.

Overall, the evidence presented here suggests that infant health–birth weight in particular–can matter for long run outcomes, but that changing infant health is difficult. While there is consistent evidence that expanding access to public health insurance can increase prenatal care, evidence on the impact of prenatal care on infant health is much weaker. Aside from the findings of Currie and Gruber (1996b) of an improvement in low birth weight rates associated with the earliest Medicaid expansions, few researchers have found compelling

evidence of improvements in infant health from prenatal care use. Researchers have suggested this may be because the content of prenatal care is seldom measured, and indeed there is some evidence that augmented prenatal care services can be beneficial for the health of the infants of the least well-off mothers (see, e.g. Long and Marquis 1998 and Joyce 1999). This evidence also suggests that prenatal care is an intervention for which the effects on the average and marginal infants are not the same, but unfortunately data limitations often preclude precise identification of the effects for the marginal infants.

### *D. Other Effects of Expanded Public Insurance Eligibility*

In addition to direct effects on health through access to health care, expanded eligibility for public insurance may have an impact on the life chances of children through the "insurance component" of health insurance. By providing public insurance to low-income families for their children, the government provides an in-kind transfer of resources. This transfer has an impact on the economic circumstances of the family through the reduction in medical insurance costs and out of pocket expenses that a family would otherwise have to incur for its children. Despite its potential importance, this effect of public health insurance has been surprisingly little studied. Gruber and Yelowitz (1999) examine Medicaid expansions between the mid-1980s and the mid-1990s and find that the average increase in dollars of medical expenditure eligible to be paid by Medicaid over this period led to an approximately $538 increase in annual consumption. They also find evidence for a reduction in wealth holdings, presumably due to a reduction in precautionary saving as a result of the expanded access to Medicaid. Taking a somewhat different approach, Banthin and Selden (2003) compare children who became eligible under the Medicaid expansions to children who were slightly better off financially. They find that there

was a 7 percentage point reduction in the fraction of families with a financial burden from family health care of at least 10% of disposable family income. These are fairly substantial effects, indicating that this is an important area for further research. In particular, it would be useful to know whether these results hold for later expansions of health insurance eligibility as well, and whether the effect of public health insurance availability continues at the same level over time.

*E. The Special Supplemental Nutrition Program for Women, Infants, and Children (WIC)*

Although not a health program per se, the WIC program is intended to improve the health of infants and children through the provision of food and nutrition education to mothers. Pregnant women, infants, and young children in families with incomes below 185 percent of the federal poverty line or who participate in certain programs for low-income families including Medicaid are eligible for WIC. WIC has been in existence since 1972 and has been evaluated often, but virtually all research on WIC has relied on observational research designs, comparing WIC participants with non-participants. Attempts to use quasi-experimental variation (such as different state policies) have proven unsuccessful, as such studies have been unable to eliminate the possibility that the results were driven by other factors varying at the state level.

More recently, a group of studies have relied on large administrative datasets with extensive controls, arguing that any selection bias in WIC participation conditional on these controls is likely to be negative, since observable selection appears negative. These studies find an association between WIC participation and improvements in birth weight, with some studies finding substantial birth weight improvements (see Table 4). However, these results have engendered considerable debate, as it appears that most improvements in birth weight associated with WIC participation have occurred via the mechanism of longer gestations rather than greater

fetal growth. Researchers such as Joyce and his collaborators have argued that WIC effects on gestation length are implausible given results from the medical literature (Joyce, Gibson, and Colman 2005 and Joyce, Racine, and Yunzal-Butler 2008). Moreover, effects on gestation length may be subject to "gestational age bias," the fact that women with longer gestations have more opportunity to enroll in WIC prenatally. Evidence that this may indeed confound estimates of WIC's effect comes from results showing that women who enrolled in the third trimester of their pregnancies had larger "WIC effects" than did women who enrolled in the first or second trimesters (Joyce, Racine, and Yunzal-Butler 2008). Controlling for gestational age results in much smaller estimated WIC effects, though as Ludwig and Miller (2005) point out in their survey of the debate, if WIC *does* have an effect on gestational length this approach would be likely to underestimate WIC's effect. In a recent paper, Figlio, Hamersma, and Roth (2009) are able to solve the problem of endogeneity of WIC participation using an instrumental variable strategy that compares the differential effects of a state policy change on women identified as marginally eligible with women who were marginally ineligible for WIC. While their empirical strategy is preferable in several ways to the strategies used in previous research, their work is hampered by the fact that their resulting analysis sample is quite small, so most of the effects they estimate have very wide confidence intervals. The only statistically significant effect they identify is a reduction in the probability of low birth weight; effects on average birth weight, gestational age, and prematurity are all statistically indistinguishable from zero. Overall, the preponderance of existing evidence suggests that WIC is likely to have an effect on birth weight, though that effect is likely to be somewhat below the large effects identified in the observational studies. Consequently, while WIC is a potentially compelling intervention given the evidence on

long-run effects of low birth weight from the twin studies discussed above, the lack of certainty about the magnitude and nature of any WIC effects precludes any strong conclusion about WIC's effect.

## III. Interventions Targeting Specific Health Conditions

### *A. Asthma*

Asthma is one of the most common chronic illnesses among children, with a national current prevalence rate of 8.9 percent in 2005 (Akinbami 2006). This level represents a historic high, with gradually increasing rates over the 1980s and 1990s. The disease is most prevalent among minority children, particularly non-Hispanic black children, who have prevalence rates that are nearly 50 percent higher than white prevalence rates (Akinbami 2006). It can be sufficiently severe as to result in disability (defined as an inability to conduct a major activity such as school or a limitation in the amount or kind of the activity performed, Newacheck and Halfon 2000). Asthma is, however, controllable for most children with medication and behavior modification. In assessing the importance of asthma-oriented interventions with the goal of poverty reduction in mind, there are two components to consider: first, can interventions successfully reduce symptom days among children with asthma, and second, are reductions in asthma symptom days likely to lead to improved long run outcomes.

From the extensive literature on treating asthma, the answer to the first question appears to be yes: even children in very difficult economic circumstances can have their asthma controlled. The Centers for Disease Control's website on Potentially Effective Interventions for Asthma[7] provides information on over 40 interventions that have been evaluated with

[7]http://www.cdc.gov/asthma/interventions/interventions_info.htm

randomized trials and shown indications of effectiveness. Based on these interventions, it is clear that access to health care (such as provided by Medicaid) is a necessary, but not sufficient, condition to ensure adequate asthma control; the interventions reviewed typically involve patient education, counseling, and possibly provision of additional resources. For example, as part of the National Cooperative Inner-City Asthma Study, Evans et al. (1999) incorporated a randomized trial of an asthma management program in an inner-city setting. This program included social workers as asthma counselors, asthma education, referrals to community resources (e.g. smoking cessation programs), pillow and mattress covers, and insecticide to reduce cockroaches in the home. Evans et al. find a reduction of 0.5 symptom days per 2-week period in treatment vs. control group and some reduction in unscheduled visits to physicians and emergency rooms by the second year follow-up. Moreover, these effects persist after the end of the intervention.

While effective interventions exist, the answer to the second question, whether interventions targeting asthma are likely to have long run impacts on poverty reduction, is much less clear. One issue is that while there is a clear link between asthma and days of school missed (see the comprehensive review by Milton et al. 2004), few studies have found effects of asthma on academic achievement. Milton et al. (2004) review 11 studies that compare the academic achievement of children with asthma to the achievement either of matched controls or the general population and find no evidence of differential academic performance between children with and without asthma. There are several problems with these studies, however. The measures of academic achievement are fairly limited, there is no experimental or even quasi-experimental variation available, and it is not always clear in the study whether the child's

asthma is well controlled or poorly controlled. Moreover, the studies typically do not focus on low income children, who suffer disproportionately from asthma and may be more at risk for differential academic performance. There is some evidence for worse labor market outcomes among individuals with childhood-onset asthma (see the review by Milton et al. (2004) and the citations therein), although the outcomes studied have been limited to employment (individuals with asthma are less likely to be employed and more likely to be out of the labor force) and the methods are limited to including (sometimes extensive) controls as there is no quasi-experimental variation available. Overall, this is an area in which additional research is necessary to determine convincingly the extent of the relationship between asthma in childhood, asthma control in childhood, and long run economic outcomes.

*B. Mental Health*

One area in which links between child health status and outcomes potentially affecting adult poverty have been fairly convincingly identified is mental health, and particularly attention deficit hyperactivity disorder (ADHD) (see Table 5). ADHD is a neurobehavioral disorder characterized by the presence of at least six symptoms of inattention or hyperactivity-impulsivity that are sufficiently severe and inconsistent with the child's level of development (American Academy of Pediatrics Committee on Quality Improvement, Subcommittee on Attention-Deficit/Hyperactivity Disorder 2000). Based on the 2003 National Survey of Children's Health, in which parents were asked about whether their child had been diagnosed with ADHD, the Centers for Disease Control estimate a prevalence rate of nearly 8 percent among children 4 to 17 years old in 2003 (CDC 2005a).

The most common approach to examining the effect of ADHD symptoms on various

outcomes is to control for family fixed effects. Using this approach, Currie and Stabile (2006) find that children with symptoms of ADHD are more likely to repeat a grade, score lower on math and reading tests, and are more likely to be placed in special education, with some evidence of stronger effects for boys than girls. Using the same methods, for comparison they examine the effects of the presence of chronic conditions and poor health and find no statistically significant relationship between physical conditions and their outcome measures. In an extension of this work, Currie and Stabile (2007) examine other behavioral problems symptoms and find qualitatively similar, though substantially smaller, effects for antisocial/aggressive symptoms and depressive symptoms.

In an examination of whether the Currie and Stabile results hold for longer term outcomes, Fletcher and Wolfe (2008) examine the effects of retrospectively reported ADHD symptoms among 18-28 year-olds on an array of high school outcomes, years of education, and whether the person attended college. Like Currie and Stabile, they find evidence of effects on short run outcomes, particularly increased probabilities of grade repetition and special education placement. They find little evidence of effects on longer run outcomes, however. Moreover, Fletcher and Wolfe find statistically significant effects in OLS models but no statistically significant effects in the family fixed effects models (with the exception of an increase in the probability an individual was suspended). They argue that this finding indicates the possible existence of spillover effects on long run outcomes for siblings of individuals with ADHD. While this is certainly a possibility, it does call into question the assumption underlying the use of sibling fixed effect models to eliminate family level unobservables.

Ding et al. (2007) and Fletcher and Lehrer (2008) use an alternative approach, using

variation in the presence of genetic markers (either across or within families) believed to be correlated with symptoms of ADHD, depression and obesity. Ding et al. (2007) find evidence of an approximately one standard deviation reduction in grade point average due to the presence of depression or obesity, but the effect is statistically significant only for girls and the combined sample. Unlike the previous studies, they find no statistically significant effect of ADHD. One concern with this study is its external validity, as the data come from five high schools from a single county in Northern Virginia. Finally, the study by Fletcher and Lehrer (2008) combines the use of genetic instruments with family fixed effects in data from the National Longitudinal Study of Adolescent Health. They find statistically significant effects of a diagnosis of attention deficit disorder on GPA, though no statistically significant effects for the combined ADHD diagnosis nor for depression nor obesity. Interestingly, the results for the family fixed effects only, instrumental variables only, and fixed effects and instrumental variables models differ substantially, raising concerns about the assumptions underlying the use of both models.

While the use of genetic markers as instruments is intriguing, it does raise the question of whether the presence of these health problems is manipulable by interventions. That is, while the use of genetic markers may help researchers establish a statistical relationship between mental health problems and later outcomes, they do less to help researchers or policy makers determine what an effective intervention targeting these health problems would be. This problem is not limited to the genetic markers studies, but holds for the studies using family fixed effects as well.[8]

[8]As noted by the Surgeon General's Report on Mental Health (U.S. Department of Health and Human Services (1999)), even mental health problems believed to have a significant genetic

Since drug treatments for ADHD exist (psychostimulant therapy), the effects of ADHD may plausibly be affected by public health insurance interventions, particularly the availability of health insurance through Medicaid and the State Children's Health Insurance Program. Indeed, Medicaid spending on stimulant drugs (most used to treat ADHD) increased fourteenfold between 1991 and 2000 (Frank, Goldman, and Hogan 2003). However, a review of studies of the impact of drug treatment for ADHD by Wigal et al. (1999) indicates that while there is convincing evidence of symptom reduction from randomized placebo-controlled trials, there is little to no evidence of improvement in academic achievement. Wigal et al. discuss three studies, two of which found no effect on academic achievement, and one which found an effect. All three of the studies have problems that limit their validity: the two studies finding no effect had very limited follow-up times (5-6 weeks of treatment), so it is possible that an effect could emerge over a longer period, while the study that found an effect had a longer follow-up period (15 months) but had a 72 percent drop-out rate among the placebo group, so that the results may be biased. The conclusion that there is little evidence of long-term academic benefits of stimulant therapy has been drawn in other reviews as well (see Pelham, Wheeler, and Chronis 1998 and the citations therein).

*C. Dental Health*

Dental caries–a bacterial infection of the tooth–is the most prevalent chronic disease of childhood–five times more common than asthma (U.S. Department of Health and Human

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component are affected by environmental factors. The Surgeon General's Report suggests that early childhood interventions such as Head Start may work to prevent mental health problems from developing. See the chapter in this volume for an extensive review of these interventions.

Services 2000). As with many chronic diseases, it is even more prevalent among low-income and minority children. For example, the mean number of decayed and filled surfaces of primary (baby) teeth among 5-year-olds was approximately 2 among children with family incomes above twice the poverty level and over 6 among children with family incomes below the poverty level (Dye et al. 2007, Figure 2, p. 4). This disparity has been increasing for primary teeth, although other data in this report indicate that disparities in caries rates for permanent teeth have not widened substantially (Dye et al. 2007). Rates of treatment also differ substantially by income and minority status, with the odds of having at least one untreated decayed tooth nearly double among poor children (see Figure 5, from U.S. Department of Health and Human Services 2000, p. 63).

In addition to causing pain, absence from school, difficulty learning, playing, and eating, and poor appearance, untreated dental disease can have long-term economic consequences. While there is a wealth of anecdotal evidence that tooth loss may lead to greater difficulty finding a job or getting a promotion (see, e.g. Sered and Fernandopulle 2007 and Shipler 2005), recent research by Glied and Neidell (2008) provides compelling empirical evidence that this is indeed the case, at least for women. Using variation in dental health caused by variation in community water fluoridation levels during childhood, Glied and Neidell find that women whose childhood counties had fluoridated water earn approximately 4% more than women who did not. They find no evidence of a relationship for men. They show evidence that this relationship is most likely due to tooth loss, finding that residence in a fluoridated community is associated with approximately one-third of a tooth more in adulthood.

There are three types of interventions targeting dental health among children: fluoridation

(either at the community level or via provision of fluoride to individual children), provision of dental sealants (coating the teeth to make them more resistant to caries), and dental insurance through Medicaid or SCHIP. The first two interventions have received extensive study and review by the Task Force on Community Preventive Services at the Centers for Disease Control. Based on this review (Task Force on Community Preventive Services 2005, ch. 7) community water fluoridation has been shown to be very effective, reducing dental caries by 30 percent to 50 percent among children 4 to 17 years old in communities with fluoridated water. It is also a relatively inexpensive intervention, with a per capita cost of between $0.68 and $3.00 per person depending on the size of the population served (U.S. Department of Health and Human Services 2000, p. 161). Among communities without community water fluoridation, children may be prescribed dietary supplements for home use or there may be a school-based dietary supplement program. The evidence on the effectiveness of home use supplements is weak, with no well-designed clinical trials of home-based supplementation and difficulties with self-selection bias and compliance arising in observational studies (U.S. Department of Health and Human Services 2000, p. 164). School-based programs (again in communities without water fluoridation) have been shown to be more effective, with randomized controlled trials showing caries reductions of 20 to 28 percent over periods of 3 to 6 years, although optimal effectiveness is only attained when administration of the supplement is tightly controlled and cost-effectiveness is only attained in schools with children at high risk of dental caries (U.S. Department of Health and Human Services 2000, pp. 164-165).

Dental sealants are a somewhat more recently considered intervention. Sealants are plastic resinous materials that are applied to the molars and harden into a protective coating,

providing a physical barrier against bacteria and food particles. For the most part, sealants are provided by individual dentists, although school-based sealant programs also exist. Sealants have been shown to reduce the incidence of "pit-and-fissure caries" (caries on the chewing surfaces of the molars) by 52 percent after 15 years (Simonsen (1991) as cited in U.S. Department of Health and Human Services 2000, p. 167). Sealants have been shown to be cost-effective as well as effective when used on children at high risk for caries. Sealants are a required service under Medicaid's EPSDT rules, as is routine dental care from a dental professional. Medicaid is thus a potentially important dental health intervention for low-income children, as is SCHIP in many states.[9] However, compelling research on the effect of the Medicaid/SCHIP intervention on dental health is relatively scarce. Based on state reports on EPSDT compliance, it is clear that the Medicaid/SCHIP intervention is reaching few children– among children enrolled in Medicaid, states report that only 30 percent received any dental service in 2004 (Gehshan and Wyatt 2007). The U.S. Department of Health and Human Services Office of the Inspector General attributes this low level of service use primarily to low levels of dentist participation in the Medicaid program, most likely because of low reimbursement rates (less than half the private rates) and the hassles involved in dealing with the Medicaid program. Another commonly cited factor is that Medicaid families place a low priority on dental services, although this may be due in part to the difficulty such families have in finding a dentist willing to accept Medicaid. Nevertheless, researchers have found evidence that having Medicaid or SCHIP yields higher levels of dental care services than being uninsured. For example, Wang, Norton,

[9]Unlike Medicaid, states are not required to include dental services in their SCHIP plans, although many do include these services.

and Rozier (2007) show that children with Medicaid or SCHIP are less likely than uninsured children to report unmet dental care need and more likely to have visited a dentist in the past six months. Further research in this area is necessary to establish a definitive causal link and to examine the role of recent changes in public health insurance provision of dental care (such as increased use of private insurers to provide SCHIP dental coverage in some states).

*D. Childhood Overweight*

Perhaps the most discussed issue in children's health in recent years has been the sharp rise in the percentage of children overweight (defined for children as a Body Mass Index (BMI) at or above the 95$^{th}$ percentile for children of the same age and sex) or at risk of overweight (defined as being between the 85$^{th}$ and 95$^{th}$ percentiles). According to data from the National Health and Nutrition Examination Survey (NHANES), the fraction of overweight children in both age groups 6-11 and 12-19 has risen from roughly 5 percent in the 1976-1980 period to nearly 20 percent in the 2003-2004 period (see Figure 6). Children who are overweight have a higher risk of various health problems including diabetes and cardiovascular problems (Gidding et al. 1996). In addition, there is a strong association between being overweight in childhood and being overweight as an adult.

Although the research is conflicting, there is some evidence that being overweight as an adult leads to worse economic outcomes among women (see Table 6). The difficulty in establishing whether such a relationship exists and is causal is of course the endogeneity of obesity and labor market outcomes. While obese individuals tend to have worse labor market outcomes, this correlation could come from the reverse causality (low wages causing obesity), for which there is some empirical support. Or there may be an additional factor that causes both

obesity and lower wages. Studies have attempted to account for these possibilities largely in three ways: sibling fixed effects (thus assuming that any omitted factors vary only at the family level), using lagged values of weight instead of current values (eliminating the reverse causality issue but not dealing with the "third factor" possibility), and instrumental variables that are correlated with the individual's weight but not his or her labor market outcomes. The most convincing studies have tended to use a combination of these approaches. For example, Cawley (2004) uses sibling weight as an instrument as well as using lagged weight and sibling fixed effects and finds evidence in the NLSY of a negative effect of weight on labor market outcomes for women. However Norton and Han (2007) also use a combination of approaches including genetic markers as instruments (similar to the approach described earlier for mental health) and find no statistically significant effect of weight on labor market outcomes for any group. One issue with this study is its relatively small sample size–there are only 524 observations in the log wage regression, so the standard errors are fairly large.

Given that there is some evidence for labor market impacts of adult overweight and evidence for a relationship between childhood overweight and adult overweight, the next question is whether successful interventions to target overweight in children exist. While there is research that shows that it is possible to reduce weight among overweight children in the short run (see, e.g. Savoye et al. 2007 for a well-evaluated weight management program targeting inner city minority children that shows evidence of successful weight management sustained for up to a year), the more difficult question is whether these short run interventions have long run effects. The American Academy of Pediatrics' Committee on Nutrition takes a fairly pessimistic view about this possibility, stating "Prevention of overweight is critical, because long-term

outcome data for successful treatment approaches are limited" (American Academy of Pediatrics Committee on Nutrition 2003). However, there is as yet little evidence of convincingly evaluated interventions targeting prevention that have long-term success–for example, none of the 88 citations in the Committee on Nutrition's Policy Statement describes such a study. Consequently, while it is possible that a causal link exists between childhood overweight and adult poverty, further research on possible interventions is needed before any recommendations can be made.

*E. Exposure to Environmental Toxins*

The final set of child health interventions I consider in this chapter are interventions targeting reduction in exposure to environmental toxins. I focus on two interventions in particular: improvements in air quality and reductions in lead exposure. According to findings from the Environmental Protection Agency cited by the American Academy of Pediatrics Committee on Environmental Health (2004), in 2002 roughly half of the American population was living in areas where monitored air did not meet air quality standards for at least one of 6 key pollutants. There are biological reasons to believe that children, and particularly infants, may be more susceptible to health problems as a consequence of air pollution. Lung development continues after birth and through adolescence, and children's recommended activities (such as outdoor play and exercise) tend to increase their exposure to pollution (American Academy of Pediatrics Committee on Environmental Health 2004). Associations between most air pollutants and health problems in children (particularly respiratory problems such as asthma) have been well documented (see American Academy of Pediatrics Committee on Environmental Health 2004 and the citations therein). Recent research has shown that there is

a causal link (and that the associations are not solely due to the fact that areas with higher pollution also tend to have other characteristics such as higher population densities that may have effects on health). Moreover, this research has shown that there are effects on children at moderate levels of pollution (see Table 7). Pollution, particularly carbon monoxide, ozone, and particulate matter, has been shown to cause increased hospitalizations for asthma and other respiratory illness (Ransom and Pope 1995, Friedman et al. 2001, Neidell 2004); increased absenteeism (Ransom and Pope 1992, Gilliland et al. 2001, Currie et al 2007); and increased infant mortality (Chay and Greenstone 2003, Currie and Neidell 2005). Thus interventions to reduce air pollution appear to be effective in improving children's health. Unfortunately, this is an area for which there is little to no information on long term effects. Detailed data over a long period of time is necessary to determine an individual's exposure to pollution in order to relate that exposure to adult outcomes. Recognizing this need, the Children's Health Act of 2000 established the National Children's Study to examine long-term (birth to age 21) environmental effects on the health and development of more than 100,000 children (http://www.nationalchildrensstudy.gov).

Another environmental hazard that has historically been significant for children's health is lead. Lead is a potent neurotoxin, causing effects on brain development and functioning even at doses originally believed to be safe. Over the 20$^{th}$ century, scientists and clinicians gradually realized that lead had negative effects on children's brain development even at levels below that causing acute lead poisoning, but because of lead's usefulness in various materials (including pipes, paint, and most notably, gasoline) there was great unwillingness to discontinue its use (Silbergeld 1997). (The decision to permit lead additives in fuel has been described as a public

health catastrophe, while the banning of lead additives in fuel has been widely recognized as a triumph of public health intervention.) Importantly for considering interventions to combat lead damage, lead damage is long lasting and difficult to treat after it has occurred (Silbergeld 1997). However, it is entirely preventable by limiting exposure. Between 1960 and 1990 the blood lead level at which the CDC recommended individual intervention in children was lowered from 60 μg/dL to 25 μg/dL, and in 1991 it was lowered further, to 15 μg/dL, with a "level of concern" at 10 μg/dL (Centers for Disease Control and Prevention 2005b). While there is evidence of lead toxicity at levels below 10 μg/dL, this evidence is all based on observational studies. Since children disadvantaged for other reasons are also more likely to have higher lead levels, it is difficult to determine whether the relationship is causal at levels below 10 μg/dL (Centers for Disease Control and Prevention 2005b). The evidence for a causal relationship between higher levels of lead exposure and both cognitive functioning and behavioral change is more widely accepted, although still not definitive at moderate levels of lead (see Silbergeld 1997 and Rhoads et al. 1999 and the citations therein). The magnitude of the effect of increasing blood lead from 10 to 20 μg/dL has been shown to be associated with a mean deficit in full scale IQ of around 1-2 IQ points based on a systematic review of the literature (Pocock et al. 1994). A compelling causal relationship between reduction in childhood lead exposure and crime has been shown by Reyes (2007), who finds that the reduction in childhood lead exposure in the late 1970s and early 1980s was responsible for significant declines in violent crime in the 1990s. Reyes (2005) also shows that the phaseout of leaded gasoline led to 3-4 percent reductions in infant mortality and low birth weight.

Nationwide, blood lead levels among children have dropped precipitously as bans on lead

in various uses have been instituted. According to data from the National Health and Nutrition Examination Surveys (NHANES), the percentage of children ages 1 to 5 with blood lead levels exceeding 10 μg/dL fell from 88.2 percent in the 1976-1980 wave to 8.6 percent in the 1988-1991 wave, to 4.4 percent in the 1991-1994 wave, and to 2.2 percent in the 1999-2000 wave (Centers for Disease Control and Prevention (undated), Table 1). However, this nationwide fall masks an increasing spread in the distribution of elevated blood lead levels, with minority children and low-income children at much higher risk (Silbergeld 1997, Table 5). Low-income minority children living in areas with older housing stocks are the most at risk, as lead paint in deteriorating housing is the most significant source of lead exposure remaining for children. Interventions targeting these children include blood lead screening (not sufficient by itself to reduce lead exposure), removal to lead-free housing, lead abatement, and effective cleaning methods. Widespread removal to lead-free housing is effective, but has not been tested as a policy due to its cost. Abatement has been shown to be effective (Charney et al. 1983) but it is also quite expensive. Rhoads et al. (1999) conducted a randomized controlled trial of a maternal education and cleaning intervention and showed that blood lead fell 17 percent as a result, with higher reductions for children whose homes were cleaned more frequently. This decrease is modest, but it does show that cleaning is an effective intervention that may be more economically feasible.

## IV. Discussion and Extensions

This chapter has surveyed a wide range of child health-related interventions and the links between them and long run outcomes. There is fairly clear evidence that several of these

interventions "work" in the sense of improving children's health, notably interventions targeting dental health, childhood asthma, and exposure to environmental toxins. However, the evidence on links between children's health and adult poverty is much weaker. While it exists, researchers have faced some important challenges in estimating the magnitude of these links. These challenges include data availability (particularly the availability of data spanning long time periods); the almost complete lack of true experiments; the limited availability of quasi-experiments affecting long-run outcomes; and the intrinsic difficulty of measurement of the outcomes of interest. Consequently, long-term effects of many types of health-related outcomes are for the most part not yet established. While enough evidence exists to indicate that at least some child health measures are causally related to long run outcomes, it remains an open question whether the interventions investigated have long run effects at levels of child health currently existing (a good example is blood lead: while it is clear that reducing blood lead from the previous high levels had significantly positive long run effects, it is less clear whether further reductions in blood lead would have a sizeable impact on the probability of adult poverty). Despite this fairly pessimistic assessment of the state of knowledge about the poverty-related benefits of health interventions, it would be irresponsible not to consider the non-poverty related benefits when assessing such interventions. It is undisputed that health is an intrinsic part of individual well-being, and the reduction in pain and suffering (both physical and in some cases, financial) offered by the health interventions surveyed here is in some cases substantial. The fact that poor children suffer from worse health gives further impetus not only to an effort to improve the research environment for determining the long-run effects of child health, but also to public policies to ameliorate poor health among poor children if only for its short run benefits.

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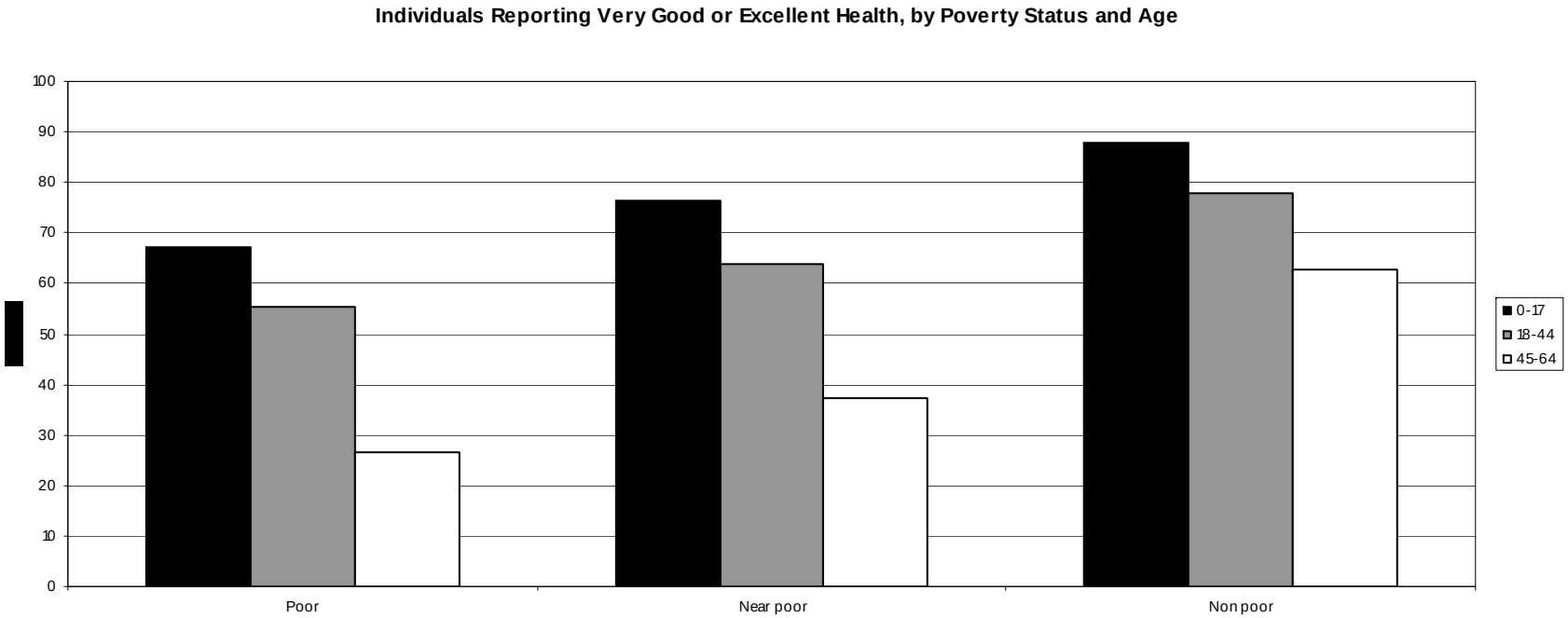
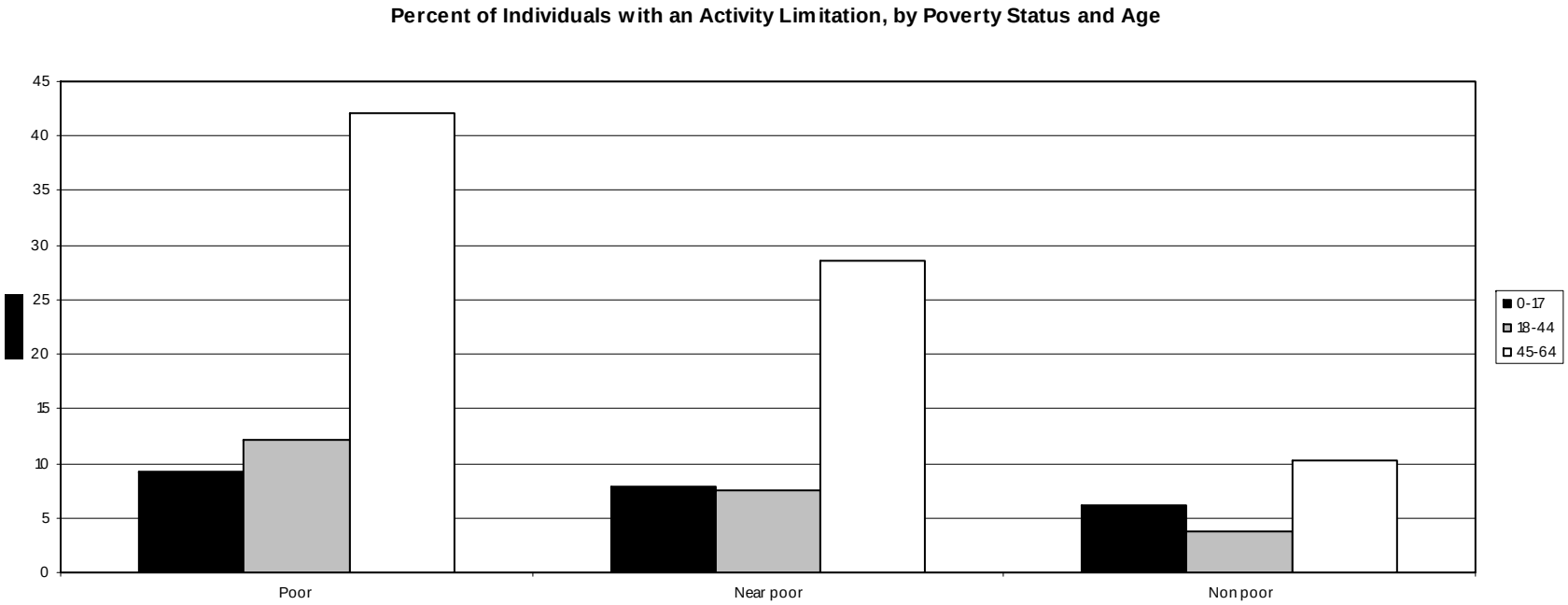
Figure 1




Source: Centers for Disease Control Health Data Interactive calculation from National Health Interview Survey 2004-2006

Figure 2




Source: Centers for Disease Control Health Data Interactive calculation from National Health Interview Survey 2004-2006

**Figure 3**

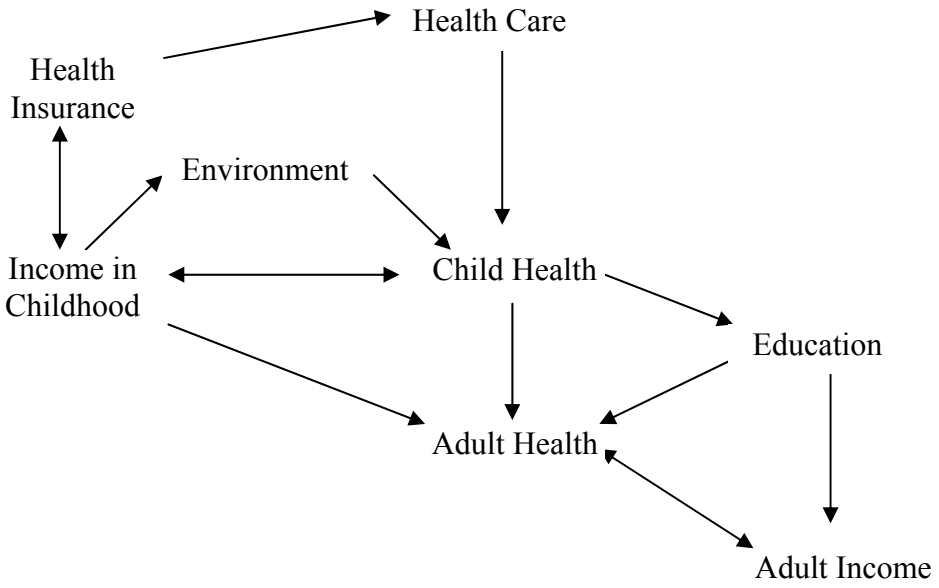
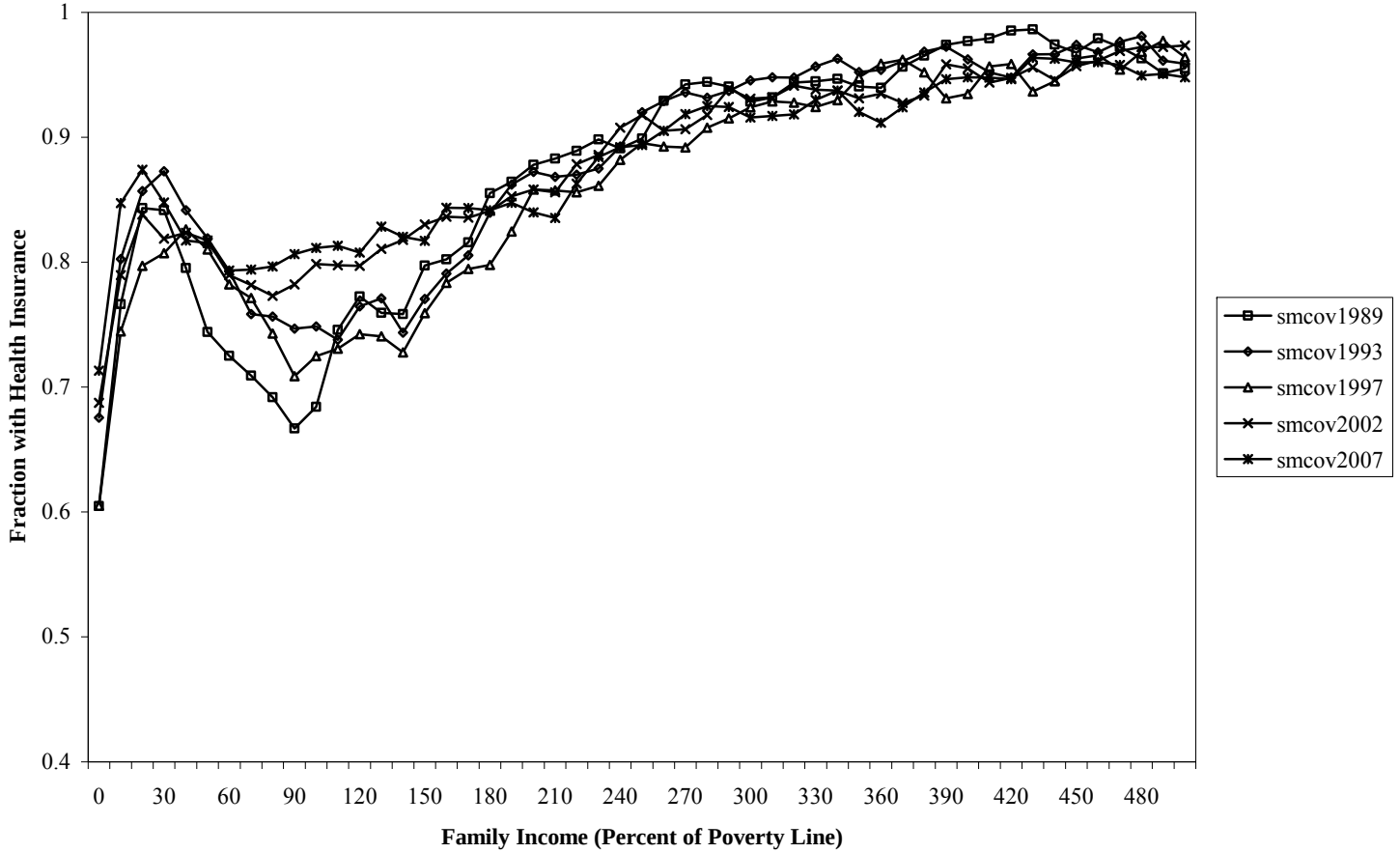

Health Care
Health Insurance
Environment
Income in Childhood
Child Health
Education
Adult Health
Adult Income

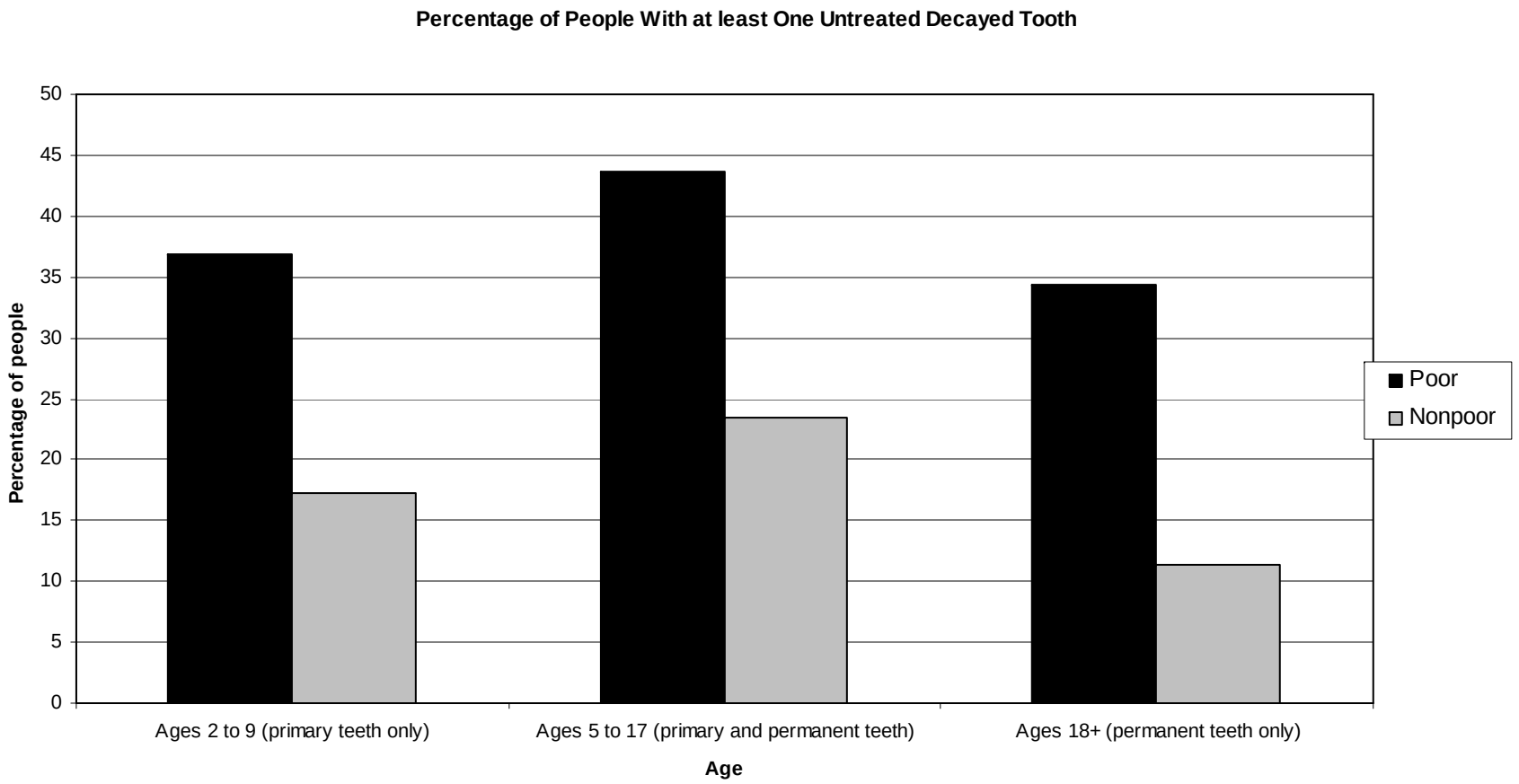
Figure 4

**Health Insurance Coverage Rates by Family Income (smoothed)**



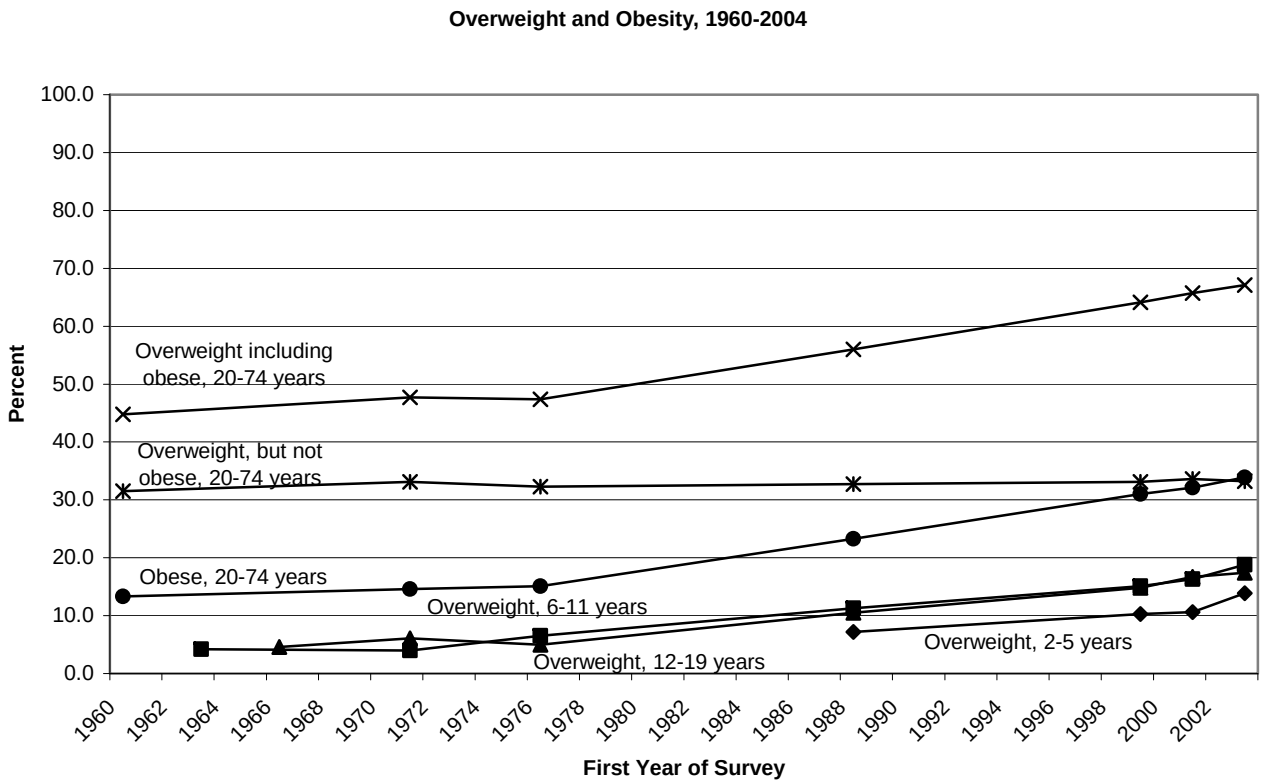
Source: Author's calculation from March Current Population Surveys, 1989, 1993, 1997, 2002, 2007

Figure 5




Source: U.S. Department of Health and Human Services (2000), p. 63.

Figure 6




Source: National Center for Health Statistics (2007), p. 41

Table 1: Selected Studies of Medicaid Expansions and State Children's Health Insurance Implementation

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Cutler and Gruber (1996) | Medicaid expansion | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | March CPS 1988-1993, children 0-18, women 15-44 | Any coverage, Medicaid coverage, private coverage | Children: Prob. uninsured reduced 8-12 pp.; Women: No stat. sig. changes in covg |
| Card and Shore-Sheppard (2004) | Medicaid expansion | Regression discontinuity; children born before and after 9/30/1983 cutoff and children older and younger than 6 | SIPP 1990-1993, March CPS 1990-1996, NHIS 1992-1996 children 0-18 | Any coverage, Medicaid coverage, private coverage, any doctor visit last year | Prob. uninsured reduced 1-9 pp.(more for OBRA 1990 expansion, less for OBRA 1989 expansion); prob. doctor visit/year increased 1-4 pp. |
| Ham and Shore-Sheppard (2005) | Medicaid expansion | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | SIPP 1986-1993, children 0-15 | Any coverage, Medicaid coverage, private coverage | Prob. uninsured reduced 10-12 pp. |
| Shore-Sheppard (2008) | Medicaid expansion | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups, controlling for coverage trends | March CPS 1988-1996, children 0-18 | Any coverage, Medicaid coverage, private coverage | Prob. uninsured reduced 8-10 pp. |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| LoSasso and Buchmueller (2004) | SCHIP implementation | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | March CPS 1996-2000 | Any coverage, public coverage, private coverage | Prob. uninsured reduced by 5-8 pp. |
| Hudson, Selden, and Banthin (2005) | SCHIP implementation | Quasi-experimental; instrumental variables and diff. in trends based on exogeneity of state coverage levels for different groups | MEPS 1996-2002 | Any coverage, public coverage, private coverage | Prob. uninsured reduced by 6-10 pp. |
| Gruber and Simon (2008) | SCHIP implementation | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | SIPP 1996, 2001 (partial) | Any coverage, public coverage, private coverage | Prob. uninsured reduced by 5 pp. |
| Currie and Gruber (1996a) | Medicaid expansion | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | NHIS 1984-1992 | Any doctor visit last year, any visit last two weeks, hospital admission last year, child mortality rate | Prob. doctor visit/year increased by 10 pp.; prob. hospital visit/year increased by 4 pp.; child mortality rate red. by 0.13 pp. for each 10 pp. increase in Medicaid eligibility |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Banthin and Selden (2003) | Medicaid expansion | Quasi-experimental; difference in differences (eligible vs. ineligible children, where ineligible consisted of two groups: children who eventually became eligible, and those who were never eligible) | NMES 1987, MEPS 1996 | Any doctor visit last year, any dentist visit last year, usual source of care, any visit to ER | Prob. doctor visit/year increased by 8-9 pp., prob. dentist visit/year inc. by 5-6 pp., no stat. sig. effect on usual source of care or ER |
| Dafny and Gruber (2005) | Medicaid expansion | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | NHDS 1983-1996, discharges for children<16, grouped into age-state-year cells | Hosp. rate, unavoidable hosp. rate, avoidable hosp. rate | 10 pp. increase in elig. ➔ 8.4% increase in hosp., 8% increase in unavoidable hosp., stat. insig. increase in avoidable hosp. |
| Kaestner, Joyce, and Racine (2001) | Medicaid expansion | Quasi-experimental; difference in differences: hosp. of children from low income zip codes vs. higher income zip codes before and after exp. | HCUP-3 HIS 1988, 1992 | Incidence of ambulatory care sensitive (ACS) hospitalizations | Decline in ACS hospitalizations (except asthma) for 2-6 year olds; little change for 7-9 year olds |
| Aizer (2007) | Medicaid outreach effort | Quasi-experimental; instrumental variables based on exogeneity of outreach program at zip code level | California hospital discharge data 1996-2000 | Incidence of ambulatory care sensitive hospitalizations | 10% increase in enrollment ➔ 2-3% red. in ACS hospitalizations |

Table 2: Selected Studies of Infant Health Care and Outcomes

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Currie and Gruber (1996b) | Medicaid expansion | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | NLSY (prenatal care); aggregate Vital Statistics (health outcomes) | Delay prenatal care; incidence of LBW and infant mortality by state and year | Becoming elig. ➔ 50% reduction in prob. delay (large s.e.); 1 p.p. increase in eligibility rate due to exp. to very low-income pregnant women ➔ 17 p.p. reduction in LBW incidence, and 3 p.p. reduction in infant mortality. No effect for later exp. |
| Piper, Ray, and Griffin (1990) | TN Medicaid exp. to married women in 1985 | Observational: outcomes in groups with large enrollment increases | TN birth certificates linked to Medicaid enrollment records | Use of early prenatal care; BW; neonatal mortality | No effect for any outcome |
| Haas et al. (1993) | MA Healthy Start | Quasi-experimental; difference in differences (before/after and elig. for new program/Medicaid eligible or private) | MA birth certificates | Satisfactory prenatal care; prenatal care before third trimester; adverse infant outcomes | No effect on either prenatal care or infant outcomes |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Piper, Mitchel, and Ray (1994) | TN Medicaid exp. in 1987 | Observational: difference over time | TN birth certificates, death certificates, Medicaid enrollment files | No 1st trimester prenatal care; no care or third trimester care; inadequate care; LBW rate; perinatal, neonatal, infant mortality rates. | Reduction in probability received no or third trimester care; no effect on any other outcome |
| Ray, Mitchel, and Piper (1997) | TN Medicaid exp. 1983-1991 | Observational: outcomes in groups with large enrollment increases | TN birth certificates linked to Medicaid enrollment records | Enrolled in first trimester; adequate prenatal care; preterm birth | 27 p.p. increase in first trimester enrollment; 6.4 p.p. red. in inadequate prenatal care; no effect on preterm birth |
| Long and Marquis (1998) | FL Medicaid exp. of 1989 | Quasi-experimental; difference in differences (before/after and enrollees/privately insured) | FL birth, death certificates, hospital discharge records, Medicaid enrollment and claims files | No prenatal care; third trimester care only; inadequate care; LBW; infant mortality | Improvements in all measures, but not all statistically significantly different from 0 |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Currie and Grogger (2002) | Medicaid expansions | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | Vital statistics detailed natality and fetal death data 1990-1996 | Prenatal care begun in first trimester; adequate prenatal care | No eff. on first trim. care start; doubling income eligibility cutoff (100-200%) inc. prob. adequate care by 0.4% for whites; no eff. on bweight; red. in fetal death for blacks |
| Hanratty (1996) | Canadian National Health Insurance implementation | Quasi-experimental; based on exogeneity of province-level adoption of national health insurance | Vital statistics natality data, county-level infant mortality data | Infant mortality rate; incidence of LBW | NHI in province ➔ 4% decline on IMR; 1.3% decline in LBW; 8.9% decline in LBW for single mothers |
| Behrman and Rosenzweig (2004) | NA (Link study) | Regressions of adult outcomes on fetal growth controlling for twin fixed effects | 804 twins from Minnesota Twins Registry | Years of schooling; BMI; height; log(wage) | 1 lb. inc. in BW ➔ 1/3 more years of educ.; 0.6 inches in height; ~7% higher wages; no eff. on BMI |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Black, Devereux, and Salvanes (2007) | NA (Link study) | Regressions of adult outcomes on ln(bweight) controlling for twin fixed effects | Data on twins from Norwegian administrative data, (1862-13106 twins, dep. on outcome; some outcomes are only for different genders) | Height; BMI; IQ score; >=12 years educ.; earnings; BW of 1st child | 10% inc. in BW (~250g) ➔ 0.57 cm in height; 0.11 inc. in BMI; 1/20 stanine inc. in IQ; 1 p.p. inc. in prob. finish high school; 1% inc. in FT earnings; 1.5% inc. in BW of 1st child |
| Oreopoulos et al. (2008) | NA (Link study) | Regressions of adult outcomes on bweight in categories or APGAR score controlling for twin or sibling fixed effects | Data on siblings from administrative data from Manitoba province (880-40514 dep. on outcome and whether twins or siblings) | Language arts test score; Pr(reach grade 12 by age 17); social assistance take-up and length | Worse infant health ➔ red. pr(reach grade 12 by 17); inc. social assistance take-up and length; little consistent effect on test score |
| Royer (2009) | NA (Link study) | Regressions of adult outcomes on bweight controlling for twin fixed effects | Data on twins from intergenerationally linked CA birth records (5670 twins) | Education; BW of child; public payment for delivery of child; zip code characteristics | 500 g. inc. in BW (~1 lb.) ➔ .06 to .08 of a year inc. in educ.; 30 g. inc. in BW of child; no eff. on public payment, char. of zip code |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Sikorski et al. (1996) | Reduced schedule of prenatal visits | Randomized controlled trial | 2794 women w/low risk pregnancies randomly assigned to standard care or reduced # visits | Measures of fetal and maternal morbidity; health service use; satisfaction | No effect on morbidity; reduced health service use; reduced satisfaction |
| McDuffie et al. (1997) | Reduced schedule of prenatal visits | Randomized controlled trial | 2764 women w/low risk pregnancies randomly assigned to standard care or reduced # visits | Preterm delivery; preeclampsia; cesarean delivery; LBW; satisfaction | No effect on any outcome |
| Clement et al. (1999) | Reduced schedule of prenatal visits | Randomized controlled trial | 1117 women w/low risk pregnancies randomly assigned to standard care or reduced # visits | Post-birth maternal and child well-being, health service use, health-related behavior | No effect on any outcome |
| Villar et al. (2001) | Reduced schedule of prenatal visits | Randomized controlled trial | Sample of women obtaining prenatal care at clinics randomized to receive standard care or fewer visits | Referral rates; hospital admissions; LBW; measures of maternal and fetal morbidity | Higher referral rates in new model; no effect on any other outcome |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Evans and Lien (2005) | Reduced access to prenatal care | Quasi-experimental; instrumental variables based on public transit strike as exogenous variation in access to care | Women in Allegheny County (women in other Pittsburgh area counties and other similar city counties as controls) | Number of prenatal visits; BW; gestation length; maternal weight gain; smoking behavior | ½ visit reduction for black inner-city residents, 1/3 visit reduction for black suburban residents; no statistically significant effect on BW, gestation length, or maternal weight gain; increase in maternal smoking prevalence |

Table 3: Financial Effects of Expanded Public Health Insurance Eligibility

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Gruber and Yelowitz (1999) | Medicaid expansion | Quasi-experimental; instrumental variables based on exogeneity of state coverage levels for different groups | SIPP 1984-1993, CEX 1983-1993 | Pr(positive net worth); net worth; annual consumption | Red. pr(positive net worth); red. wealth holdings by $567-$722; inc. consumption by $538 |
| Banthin and Selden (2003) | Medicaid expansion | Quasi-experimental; difference in differences (eligible vs. ineligible children, where ineligible consisted of two groups: children who eventually became eligible, and those who were never eligible) | NMES 1987, MEPS 1996 | Family out of pocket spending; financial burden | Red. in out of pocket spending of ~$600; 7-8 pp. red. in prob. health care exp. >10% of disp. family income |

Table 4: Selected Studies of the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC)

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Kowaleski-Jones and Duncan (2002) | WIC | Family fixed effects (sibs w/ and w/o maternal prenatal WIC participation) | NLSY children born 1990-1996 (1984 children, 969 sibs, 71 discordant sib groups) | ln(BW) | WIC part. assoc. w/ stat. insig. inc. in BW at the mean |
| Bitler and Currie (2005) | WIC | Observational (Medicaid deliveries w/ and w/o WIC) | PRAMS deliveries pd. by Medicaid, 1992-1999 (60,731 women from 19 states) | BW; gestation length, P(prenatal care beg. 1st trimester); P(preterm); P(LBW); P(VLBW) | WIC part. assoc. w/ 63.7 g. inc. in BW; 0.28 of a week inc. in gest. length; 44% inc. in P(1st trimester care); 29% dec. in P(preterm); 27% dec. in P(LBW); 54% dec. in P(VLBW) |
| Joyce, Gibson, and Colman (2005) | WIC | Observational (Medicaid deliveries w/ and w/o WIC) | New York City birth cert., 1988-2001 (811,190 births) | BW; gestation length; BW for gestational age | WIC part. assoc. w/ 25.5 g. inc. in BW; 0.12 of a week inc. in gest. length; 7.3 g. inc. in BW for gestational age; results not consistent over time |

| | | | | | |
|---|---|---|---|---|---|
| Joyce, Racine, and Yunzal-Butler (2008) | WIC | Observational (women who enrolled in WIC during vs. after pregnancy) | PNSS deliveries ever enrolled in WIC, 1995-2004 (3,311,976 women from 9 states) | BW; P(LBW); P(VLBW): P(preterm); BW for gestational age; P(small for gestational age); P(term LBW) | prenatal WIC assoc. w/ 63 g. inc. in BW; 2.7 pp. dec. in LBW; 0.9 pp. dec. in VLBW; 2.7 pp. dec. in P(preterm); 39.5 g. inc. in BW for gest. age; 1.7 pp. dec. in P(small for gest. age); 0.7 pp. dec. in P(term LBW) |
| Figlio, Hamersma, and Roth (2009) | WIC | Quasi-experimental; instrumental variables based on differential impact of new documentation requirement in marginally eligible and marginally ineligible groups | Florida linked admin. data: births to women with a child already in school, 1997-2001 (4190 women) | BW; P(LBW); gestational age; P(preterm) | WIC assoc. w/ stat. insig. inc. in BW; 12.9 pp. dec. in LBW; stat. insig. eff. on gest. age and P(preterm) |

Table 5: Selected Studies of Child Mental Health

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Currie and Stabile (2006) | NA (Link study) | Family fixed effects (sibs w/ and w/o ADHD symptoms) | NLSY (3969 children ages 5-11, 2406 sibs) NLSCY (3925 children ages 4-11, 1540 sibs) | Math, reading test scores; probability of grade repetition, special ed. placement | (US): Median → 90th pctile in ADHD symptom scale ➔ 10 point red. (on mean of ~50) in math and reading scores; increase prob. of grade rep. and special ed. placement |
| Currie and Stabile (2007) | NA (Link study) | Family fixed effects (sibs w/ and w/o reported behavioral problems) | NLSY (3758 children ages 5-11, 2358 sibs) NLSCY (5604 children ages 4-11, 2374 sibs) | Math, reading test scores; probability of grade repetition, special ed. placement | (US): Median → 90th pctile in hyperactivity symptom scale ➔ 0.2 s.d. red. in math and reading scores, inc. prob. grade rep., special ed.; Median → 90th pctile in antisocial/aggressive symptom scale ➔ 0.1 s.d. red. in math and reading scores, inc. prob. grade rep.; Median → 90th pctile in depressive symptom scale ➔ no significant rel. to math and reading scores, inc. prob. grade rep. |
| Fletcher and Wolfe (2008) | NA (Link study) | Family fixed effects (sibs w/ and w/o ADHD symptoms) | Add Health (retrospective survey of ~14000 indiv. ages 18-28, ~2900 sibs) | GPA; probability repeat grade, special ed. placement, suspended, expelled, drop out; years of | Inc. prob. repeat grade, placed in special ed., drop out; no other statistically significant results in FE models |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| | | | | education, probability attend college | |
| Ding et al. (2007) | NA (Link study) | Instrumental variables (genetic markers as instruments) | Georgetown Adolescent Tobacco Research study (2576 adolescents from a county in N. VA) | GPA | Depression and obesity ➔ ~1 s.d. reduction in GPA, stat. sig. for combined sample and girls only; No stat. sig. effect for ADHD |
| Fletcher and Lehrer (2008) | NA (Link study) | Instrumental variables (genetic markers as instruments) and family fixed effects | Add Health (1684 individuals with genetic information, 1068 siblings) | Peabody Picture Vocabulary Test (Revised) | ~2 s.d. red. from ADD; ~1 s.d. red. from ADD when sample limited to same gender twins |
| Wigal et al. (1999) | Drug treatment for ADHD | Review of 3 placebo-controlled randomized studies | | | No effect on test scores in short term; possible long run effect but study inconclusive |

Table 6: Selected Studies of Overweight and Obesity

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Averett and Korenman (1996) | NA (Link study) | Sibling fixed effects, lagged values of BMI | NLSY (1990) | Family income; pr(marriage); spouse's income; hourly wages | Higher BMI category ➔ red. in family income, pr(marriage), only for women |
| Cawley (2004) | NA (Link study) | Individual and sibling fixed effects, sibling weight as instrument for own weight | NLSY (13 years) | Log(wage) | IV est: 1 s.d. inc. in weight 36 lbs) assoc. w/ 10 percent dec. in wages for white women; no sig. effect for other groups |
| Norton and Han (2007) | NA (Link study) | Instrumental variables (genetic markers as instruments) | Add Health | Pr(employed); log(wage) | No stat. sig. effect on either outcome for women or men |

Table 7: Selected Studies of Air Pollution

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Ransom and Pope (1992) | Reduction in pollution due to steel mill closure | Quasi-experimental; mill closed/opened due to labor strike, compared to adjacent valley | Utah Valley school districts weekly or daily attendance records, 1985-1990 | Absenteeism | ~1% of students absent each day as a result of particulate pollution exposure; effect of high pollution on absenteeism persisted for 3 to 4 weeks |
| Ransom and Pope (1995) | Reduction in pollution due to steel mill closure | Quasi-experimental; mill closed/opened due to labor strike, compared to adjacent valley | Hospital administrative records, vital statistics | Hospital admissions; mortality rate | 120% inc. in bronchial and asthma adm. for preschool children; 17% inc. in pneumonia adm. for preschool children; no stat. sig. eff. on mortality |
| Gilliland et al. (2001) | Daily variation in pollution levels | Observational; variation across 12 CA communities | Children's Health Study: 4th graders in 12 comm. w/in 200 mi. of LA; School records and survey data | Absenteeism | 20 ppb of ozone inc. assoc. w/62.9% increase for illness-related absence rates, esp. resp. illness |
| Chay and | Reduction in | Quasi-experimental; | US Vital Statistics | Infant mortality | 1 $\mu g/m^3$ dec. in |

| Study | Intervention | Design | Sample | Outcomes | Effects |
|---|---|---|---|---|---|
| Greenstone (2003) | pollution due to recession | variation across counties and over time in pollution levels | and pollution data from the Environmental Protection Agency | | particulates assoc. w/ 5 fewer infant deaths/100,000 live births |
| Neidell (2004) | Seasonal reduction in pollution at the zip code level | Quasi-experimental; seasonal variation in pollution by zip code and year | CA hospitalization data at zip code-month level | Hospitalization for asthma | CO inc. asthma hosp. for children over 1 |
| Currie and Neidell (2005) | Seasonal reduction in pollution at the zip code level | Quasi-experimental; seasonal variation in pollution by zip code and year | CA vital statistics data, 1989-2000 | Infant mortality | One-unit red. in CO dec. inf. mort. by 34 per 100,000 live births |
| Currie et al. (2007) | Seasonal reduction in pollution at the school level | Quasi-experimental; seasonal variation in pollution by school, attendance period, and year | School-level average absentee rates by 6-wk att. period, TX, 1996-2001 | Absenteeism | Absentee rates inc. with increasing days CO is within 75% of EPA threshold |

Appendix: List of Data Source Names

Add Health=National Longitudinal Study of Adolescent Health
CEX=Consumer Expenditure Survey
CPS=Current Population Survey
HCUP-3 HIS=Healthcare Cost and Utilization Project Nationwide Inpatient Sample
NHDS=National Hospital Discharge Survey
NHIS=National Health Interview Survey
NLSY=National Longitudinal Survey of Youth
NLSCY=Canadian National Longitudinal Survey of Children and Youth
NMES=National Medical Expenditure Survey
MEPS=Medical Expenditure Panel Survey
PNSS=Pregnancy Nutritional Surveillance System
PRAMS=Pregnancy Risk Assessment Monitoring System
SIPP=Survey of Income and Program Participation