Medicaid Eligibility and Obesity of Low-Income Children

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Abstract

This paper studies the effect of Medicaid expansions on childhood obesity and finds robust evidence of ex-ante moral hazard induced by public insurance. I establish this result by estimating two reduced-form models and a structural model. My reduced-form identification strategy exploits eligibility discontinuity created by the Omnibus Budget Reconciliation Act 1990, which extended Medicaid eligibility to children from families below the federal poverty threshold and born after October 1983. Drawing on the MEPS, I find offering low-income children public insurance leads to an approximately 10-percentage-point increase in the chances of obesity. Combining the MEPS and the SIPP, I am able to investigate the intermediate-term effects of insurance take-up. I estimate a fuzzy regression discontinuity design using Angrist-Krueger two-sample IV estimator (Angrist and Krueger 1992). The results suggest that early insurance take-up induced by the expansions of Medicaid leads to a roughly 3-percentage-point increase in chances of obesity. I also develop and estimate a two-period structural model that quantifies moral hazard, net-wealth effect, and risk preferences. I use the estimates to study the relative importance of net-wealth effect and moral hazard in the childhood obesity problem. The estimates of the choice model suggest that ex-ante moral hazard accounts for a greater share of the increase in childhood obesity rate than net-wealth effect.

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1 Introduction

This paper attempts to provide a full account of the effect of public insurance on childhood obesity. I provide new evidence to answer the question in three parts: (1) Does Medicaid eligibility affect childhood obesity? (2) Does insurance take-up affect childhood obesity? (3) Through what mechanism does Medicaid eligibility affect childhood obesity?

The first question focuses on the “intent-to-treat” effect: How does the “offering” of public insurance affect the obesity problem of low-income children? Without perfect compliance, that is, some eligible children would not take up the coverage, children who take up Medicaid can have a very different health status from those who do not. The well-known endogeneity problem of public insurance coverage makes it invalid to compare the outcomes between children under coverage and those who are not. On the other hand, Medicaid eligibility is likely exogenous. I exploit the eligibility discontinuity created by the Omnibus Budget Reconciliation Act (OBRA) 1990 that expanded the eligibility for Medicaid to children from families below the poverty line and born after October 1983. Applying a regression discontinuity design, I compare the obesity rate between the cohort born right before October 1983 and the cohort born right after. This comparison yields causal interpretation since the eligibility cutoff was exogenous. Parents do not have perfect control over children’s dates of birth. The circumstances bear a close resemblance to a randomized experiment, which motivates the causal interpretation of the RD design. Drawing on the Medical Expenditure Panel Survey 1996 - 2001, I found roughly a 10-percentage-point increase in childhood obesity caused by Medicaid eligibility in 1990.

To show the robustness of my estimates, I estimate a regression-adjusted difference-in-difference model using children from families of the 100% - 124% federal poverty level (FPL) as the control group. The estimates rely on an additional assumption that the control group has a similar obesity trend to the treatment group. My estimates suggest that a roughly 5-percentage-point increase in the obesity rate is attributable to Medicaid expansions. This result is robust against the functional form assumptions. As a placebo test, a graphical analysis that uses the subsample of children from families between 100% FPL and 199% FPL shows no discontinuity at the cutoff of October 1983. To formalize the analysis, I estimate a regression-discontinuity design that uses counterfactual birth-
month cutoffs. I find no consistent evidence that shows the false policy cutoff has any effect on the obesity rate.

A valid instrumental variable is necessary to answer the question of the effect of insurance take-up. The previous argument immediately implies that eligibility is a valid instrument for insurance coverage given eligibility affects taking up insurance, equivalent to a fuzzy RD design. In order to address the effect of early take-up on childhood obesity, I estimate the fuzzy design combining two data sets. I estimate the first stage using SIPP 1992, 1993, or 1996, and the second stage using MEPS 1996-2001. The estimates can be interpreted as the effect of early insurance take-up on later obesity. I found an approximately 5-percentage-point increase in prevalence of obesity.

My reduced-form findings suggest that Medicaid expansion has adverse effects on childhood obesity. A possible policy intervention to correct for ex-ante moral hazard is to impose a behavior-adjusted premiums while providing information for low-income households suffices to correct for net-wealth effect. Because the two potential causes of the effect have different policy implications, it is of interest to further investigate the relative importance of these two mechanisms. To accomplish the goal, I estimate a structural model, where food spendings on children are recovered from observed household income and household preference. The results suggest that ex-ante moral hazard has a more substantial effect on childhood obesity than net-wealth effect. A counterfactual simulation suggests that obesity prevalence is relatively responsive to changes in premiums. The results supports the use of behavior-adjusted premiums to correct for the adverse effect of Medicaid expansions.

2 Background

It is well-known in the economic literature that individuals have weaker incentives to engage in healthy behaviors that help prevent illness and injuries in the presence of health insurance. This scenario is the ex ante moral hazard problem (Rashad and Markowitz, 2007; Zweifel and Manning, 2000; Dave and Kaestner, 2009). For children who became eligible for public health insurance, either they or their parents may have weaker incentives to look after the child’s weight. Due to the recent expansion of public insurance, ex-ante moral hazard is no longer one’s own problem. Through
pooled health insurance without adequately adjusted premiums, a decrease in one’s preventive efforts lead to a negative externality (Bhattacharya et al., 2007). When one child becomes obese, he is at a greater risk of complications. Because of pooled health insurance, the increase in his medical costs are bore by many others. The existence of externalities justifies public policy interventions.

Along with the ex ante moral hazard, public insurance may also lead to a net-wealth effect: a more expensive private insurance package is replaced by a less costly public insurance package, which leaves the family with additional money for food. The relationship between income and obesity is well-documented in the literature. In general, the relationship between income and the BMI has an inverted U-shape (Chang and Lauderdale, 2005). Akee et. al (2010) exploit an exogenous government transfer to adult members of an American tribe. Because non-Indians in the community do not receive the disbursements, they can identify the causal effect of income on BMI using this exogenous policy change. Their findings are also congruent with the inverted U-shape curve. One of the main reasons that an increase in income raises obesity rates of low-income population is that low-income and non-white individuals have more convenient access to fast food and consume more fast-food (Block et. al, 2004). Easier access to fast-food in low-income neighborhoods decreases availability of healthy food. In the end, as their income increases, their BMI increases.

On the other hand, the recent reforms on Medicaid mandated the states to provide preventive care, such as the Early and Periodic Screening, Diagnostic and Treatment (EPSDT) benefits. If the child periodically attends preventive care, she may follow suggestions for a healthier lifestyle through changing her diet and exercise routines. These opposite factors lead to an a priori ambiguous outcome. It is of policy interest to identify the causal effect of public insurance expansion on childhood obesity.

Childhood obesity is an important problem in the United States. Obesity rates among U.S. children rapidly increased over the past few decades. In a 2008 nationwide survey, obesity was ranked as the number-one health problem for children. Comparing surveys conducted in 1971-1974 and in 1999-2000 revealed a stunning trend: obesity rates among two-to-five-year-olds rose

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1The EPSDT benefits require the states to provide preventive health services for children under age 21 who are enrolled in Medicaid based on the federal guidelines.
from 5% to 10.4%, from 4% to 15.3% among six-to-eleven-year-olds, and from 6.1% to 15.5% among twelve-to-nineteen-year-olds (Cawley, 2010; Ogden, Flegal, Carroll, and Johnson, 2010). As shown in Figures 1 and 2, the changes in prevalence of obesity and overweight for American children between 2003 and 2007 are visually detectable, even within a four-year window. In particular, the southern states suffer from the most severe childhood obesity problem. Childhood obesity imposes not only a direct cost through medical expenditure but also an indirect cost through potential adult obesity. Wang and Dietz (2002) estimated that annual costs of obesity-associated hospitalization for children ages 6 - 17 increased from $35 million in 1979 - 1981 to $127 million in 1997 - 1999. A more recent study by Trasande, Liu, Fryer, and Weitzman (2009) found that, between 2001 and 2005, obesity-associated hospitalization costs rose from $125.9 million to $237.6 million. Indirectly, childhood obesity is positively associated with adult obesity. Roughly a third of obese children became obese adults (Cawley, 2010). Adult obesity has been shown to be a risk factor for many illness, such as heart disease, Type 2 diabetes, etc.. It is also related to labor market outcomes: adult obesity is associated with lower wages and with higher job absenteeism.

An important motivation for expansions of public insurance in the United States is to address relatively poor health for children from low-income families. Particularly noticeable are the social inequalities in childhood obesity in the United States. Children from low-income families are roughly twice as likely to be obese as children from high-income families (Singh et al., 2010). Obesity may be an important factor that leads to the disparities in health. Under the Affordable Care Act, individuals under age 65, including parents and adults without dependent children and with incomes below 133% of the Federal Poverty Level will become eligible for Medicaid in every state in 2014. It means that we expect a great portion of Medicaid-eligible children to remain eligible for Medicaid from 2014. Considering Medicaid’s costs are higher than that of any other program, accounting for 24% of spending in 2012, and low-income children are at the highest risk of childhood obesity, it is important to know whether offering public insurance at an early age could lower the childhood obesity rate. It would in turn affect the prevalence of obesity among low-income adults and long-term Medicaid costs.

Expansions of Medicaid, including introduction of SCHIP, in the past two decades provided a
unique opportunity to make causal inferences. Many discontinuities in eligibility were created by the expansions. Before the Omnibus Budget Reconciliation Act (OBRA) of 1986, Medicaid primarily covered children who received assistance through Aid to Families with Dependent Children (AFDC). OBRA 1986 expanded the eligibility to children under age 6 in families with income less than 133% of the poverty threshold. This change created a discontinuity in eligibility: a five-year old child from a family between 100% and 133% of the poverty threshold was eligible for Medicaid while those more than six years old were not. OBRA 1990 extended the eligibility to cover children born after September 30, 1983 in families below the poverty line. The policy discontinuity occurred because children born in October 1983 with family incomes below the poverty line were eligible while those who were born one month earlier were not. The change brought by the Balanced Budget Act (BBA) of 1997 provided federal matching funds for states to expand coverage up to age 19 and to children in families with incomes beyond previous Medicaid eligibility levels (Card and Shore-Sheppard, 2004; Levine, McKnight, and Heep, 2011). This legislation created an age-discontinuity in eligibility at age 19 in 1999. Young adults who were under age 19 in 1999 were eligible for most state health insurance programs while those over age 19 were not.

3 Empirical Strategy

3.1 Identification

My identification strategy relies on the birth month cutoff for eligibility created by OBRA 1990. In order to address the importance of OBRA 1990, I created a simulation algorithm based on the key policy parameters for OBRA 1990 and the introduction of SCHIP in 1997 reported in Table 1 which summarizes the changes in public health insurance policies in each state. The algorithm simulates gains in years of eligibility for children born between 1975 and 1990. Figures (3) plot the simulated childhood eligibility gains with respect to birth date using the state policy parameters of the West Region. I added a vertical line to show the cutoff in October 1983. The other states show similar patterns.

As shown in all the graphs, the largest jump in eligibility gains for children below the poverty
line was created by OBRA 1990. Children born in October 1983 enjoyed more years of Medicaid eligibility than children born in September 1983. This fact holds for all the states even though each state has its own public health insurance programs. The graphs also show gains in eligibility for children from families of different poverty thresholds. We do not observe the same increase in gains in eligibility in September 1983 for other poverty levels. In addition, the magnitude of discontinuity in eligibility created by OBRA 1990 was unique in recent years, which justifies the use of OBRA 1990 to study the effects of public health insurance eligibility on low-income children. Meyer and Wherry (2012) also did a calculation on average years of eligibility gains by OBRA 1990 using the March Current Population Survey. They found OBRA 1990 could increase the years of eligibility for children who were born after September 30, 1983 by as much as 4.7 years.

Except for the fact that OBRA 1990 created the largest discontinuity in eligibility in the recent Medicaid expansions, another advantage lies in its external validity. An RD design delivers a credible treatment effect for the subpopulation at the threshold. In general, generalization of the local average treatment effect to individuals away from the threshold may not be externally valid. As Lee and Lemieux (2010) argues, depending on the context of the question, the discontinuity gap in an RD design can be interpreted as a weighted average treatment effect across all individuals, where the weights equal the relative ex ante probability that an individual is in the neighborhood of the threshold. This argument is very likely to hold in this context. If we believe that dates of birth are random and the threshold of September 30, 1983 was set exogenously – that is, it is equally likely for the policymakers to set the threshold on October 31, 1983 as well as January 31, 1983, then the effect would have been similar if the threshold were to set on another date. Therefore, using the cutoff defined by birthdate has an additional advantage as a clear possibility for the estimated effect to be externally valid. On the other hand, if one uses the cutoff defined by income levels, then it may be hardly persuasive that the effect at the 100% poverty threshold would have been similar if the threshold were to be set at the 300% poverty threshold. The same difficulty would arise for an age cutoff as well. In short, the RDD estimator in my context can be interpreted as the effect of the eligibility expansions averaged across the full sample of children.
weighted by the probability of being “around” the cutoff\textsuperscript{2}

The same identification strategy has been applied to answer a couple of different questions. Card and Shore-Sheppard (2004) is the path-breaking study that exploited the eligibility cutoff created by OBRA 1990 to study the crow-out effect of public insurance. A recent paper by Meyer and Wherry (2012) applied the same policy discontinuity to study the effect of public insurance on teenagers’ mortality rates.

3.2 Sharp Regression Discontinuity Design

The key idea behind the regression discontinuity design is to examine whether the discontinuity at the eligibility threshold, September 30, 1983, is mirrored by the discontinuity in obesity. Based on OBRA 1990, a significant increase in years of eligibility is a deterministic function of month of birth:

\[
\text{Eligible} = 1(\text{Birth Month} \geq \text{October 1983}).
\]

This rule implies the applicability of a sharp RD design. In the following analysis, I would normalize the eligibility cutoff to zero. That is, the previous equation can be written as

\[
D = 1(x \geq 0).
\]

The validity of the identification strategy crucially hinges on the assumption that individuals are not able to perfectly manipulate the forcing variable so that they get selected into the treatment group. It is highly unlikely in my case. OBRA 1990 was passed in 1990 and was applied to the children who were born after September 30, 1983. A histogram of the number of observations by month of birth (Figure 5) shows no sign of manipulation as expected.

To estimate the effect of Medicaid eligibility on childhood obesity, I mainly work with the following specification:

\[
Y = \beta_0 + \tau \cdot D + f(x; \theta) + g(x, D; \delta) + W\gamma + \epsilon. \tag{1}
\]

\textsuperscript{2}For the details about the weighted average treatment effect interpretation, see Lee and Lemieux (2010)
$D$ is a binary indicator for eligibility; $x$ is the month of birth, the forcing variable; and $W$ is other control covariates, in which I include dummies for age, sex, census region, race, hispanic, month of birth, and survey year. As noted in Card and Lee (2008), an RD design is not non-parametrically identified if the forcing variable is discrete. The insight is that when the support for the forcing variable is discrete, we cannot compare the cohort “right before” the cutoff with the cohort “right after” the cutoff. Therefore, we have to make a functional-form assumption on how the conditional expectation function is “approaching” the cutoff in the limit. The function $g(x, D; \delta)$ in Equation 1 is a lower-order polynomial of the forcing variable interacted with the treatment variable $D$. The function $f(x; \theta)$ is a lower-order polynomial of the forcing variable. This specification allows the conditional mean functions on the both sides of the cutoff to have different functional forms. It is equivalent to allowing the potential outcomes, $Y(0)$ and $Y(1)$, to have different functional forms.

3.3 Fuzzy Regression Discontinuity Design

As previously discussed, the RD design corresponds closely to a local randomized experiment. It is the reason why estimating Equation (1) yields a causal interpretation. Extending the previous argument, one notices that eligibility status is a valid instrument for insurance coverage given Medicaid eligibility affects insurance coverage.

Locally random assignment of Medicaid eligibility

3Let $Y(0)$ and $Y(1)$ denote the potential outcomes. $Y(0)$ is a binary indicator for whether a child would have been obese if he were not eligible for Medicaid; $Y(1)$ is a binary indicator for whether he would have been obese if he were eligible for Medicaid. Let $f(x, \theta)$ and $h(x, \alpha)$ denote flexible functions of the month of birth. Then the following specification motivates the model:

$$Y(1) = \beta_0 + \tau + h(x, \alpha) + \epsilon$$
$$Y(0) = \beta_0 + f(x, \theta) + \epsilon.$$ 

The revealed outcome follows:

$$Y = D \cdot Y(1) + (1 - D) \cdot Y(0)$$
$$= \beta_0 + \tau D + f(x, \theta) + g(x, D; \delta) + \epsilon$$

where $g(x, D; \delta)$ is essentially the difference between $h$ and $f$ function interacted with $D$ under the assumption that $f$ and $h$ are polynomials of the same degree.

The model with additional demographic covariates follows the same logic.

4The argument can be easily understood under the potential outcome framework. Let $Y(t), t \in \{0, 1\}$ denote the potential obesity propensity under coverage status; $T(d), d \in \{0, 1\}$ denote the potential coverage under eligibility status; $D$ denotes the eligibility status assigned by OBRA 1990. The following assumptions underly the validity of the IV estimator:

Exogeneity $\{Y(0), Y(1), T(0), T(1)\} \perp D$

Non-trivial Assignment $P(D) \in (0, 1)$
implies that eligibility is exogenous to insurance coverage. The assignment of eligibility is non-trivial because there are children born before and after October 1983. The fact that expansion of public insurance affects insurance coverage is supported by many previous studies, such as Card and Shore-Sheppard (2004), De la Mata (2012), to name a few. The monotonicity assumption, which is standard in an IV framework, states that children who were covered by any insurance would not become uncovered if they were offered public insurance. Even though there is evidence for public insurance crowding out private insurance, there is no evidence showing that people who are covered by their own private insurance give up both private and public insurance after becoming eligible for public insurance. Therefore, this assumption is likely to hold as well.

To estimate the effect of insurance take-up on childhood obesity, I primarily work with the following specification:

\[
Y = \beta_0 + \beta T + f(x, \theta) + \epsilon \\
T = \alpha_0 + \alpha D + g(x, \delta) + \nu
\]  

where \( T \) is a binary indicator for insurance coverage, \( D = 1(x \geq 0) \) denotes the eligibility status, and \( x \) is the number of months born before or after October 1983. The parameter of interest is \( \beta \), which is the effect of insurance coverage on childhood obesity. Because insurance coverage is determined by factors other than the month of birth, the model is a fuzzy regression discontinuity design:

\[
Pr(\text{Take-up} = 1|\text{Month of Birth} = x) = \begin{cases} 
  g_1(x) & \text{if } x \geq 0 \\
  g_0(x) & \text{if } x < 0 
\end{cases}
\]

where \( g_1(x) \) and \( g_0(x) \) are different functions of \( x \). The fuzzy RD design can be estimated by standard two-stage-least-squares.

As shown in Hahn, Van der Klauww, and Todd (2001), the fuzzy RD design has the same interpretation as the Wald estimator. The fuzzy RD design estimator estimates the average treatment

\[ \text{Relevance } P(T(1)) \neq P(T(0)) \]

\[ \text{Monotonicity } P(T(1) \geq T(0)) = 1. \]
effect of the “compliers” around the eligibility cutoff. In other words, the fuzzy design estimator estimates the average effect at the eligibility cutoff for children who became covered by either public or private insurance after being eligible for Medicaid in 1990. In short, the estimator has a direct interpretation as the effect of the insurance “take-up.”

Because obesity takes time to develop, it is more interesting to know the effect of insurance coverage some time after they took up the insurance. To address this problem, I combine the Survey of Income and Program Participation (SIPP) with the primary dataset. The SIPP has information on insurance coverage in 1992, 1993, and 1996, while the MEPS has information on obesity between 1996 and 2001. Utilizing the information in the two different datasets allows me to estimate the effect of insurance coverage for those who took up in the early 1990s. For example, the estimates using SIPP 1992 can be interpreted as the effect of insurance take-up in 1992 induced by OBRA 1990 on obesity in 1996 - 2001.

The fact that a fuzzy design can be estimated by TSLS motivates a simple two-sample IV estimator (TSIV). When TSLS is just-identified, it is equivalent to the IV estimator of the form

\[ \hat{\beta}_{IV} = (X'X)^{-1}X'Y \]

where \( Z \) is a N-by-K matrix of instrumental variables, \( X \) is a N-by-K matrix of covariates, and \( Y \) is a N-by-1 vector of the outcome variable. Then a two-sample IV estimator has a moment function, which can be estimated using GMM with the moment function:

\[ g(\theta) = \frac{Z'Y_1}{N_1} - \frac{Z'X_2\theta}{N_2}. \]

Using Medicaid eligibility to instrument for insurance coverage, the model is the just-identified case for the TSIV estimator firstly proposed by Angrist and Krueger (1992). The insight of Angrist and Krueger (1992) is to notice that the IV estimator can be written as a function of two sets of sample moments. Under some regularity conditions and the assumption that the sample size of both samples grows to infinity in a fixed ratio \( k \). That is, (1) Estimated moments of the two samples are independent and (2) \( \lim_{N_1 \to \infty} \frac{N_1}{N_2} = k \), where \( k \) is a constant. The two-sample IV estimator fits seamlessly into the GMM framework. The assumption required for the moment function to have zero mean is \( \text{cov}(Z_{1},X_{1}) = \text{cov}(Z_{2},X_{2}) \). Technically, the comparability assumption requires \( \text{plim}_{N_{h} \to \infty} \left( \frac{Z_{h}'X_{h}}{N_{h}} \right) = \Sigma_{ZX} \) for \( h=1,2 \). In other words, even though I do not observe \( X_{1} \), the population covariance of \( Z_{1} \) and \( X_{1} \) would be the same as the population covariance of \( Z_{2} \) and \( X_{2} \) if I have observed \( X_{1} \).

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Suppose I wanted to estimate the effect of insurance take-up for the compliers in year $t$. The two-sample IV estimator requires the assumption that insurance coverage in year $t$ is correlated with eligibility in the same way as in the SIPP as if I had observed insurance coverage in year $t$ in the MEPS. This assumption is trivially satisfied because both the SIPP and the MEPS are designed to construct a representative sample of the United States. The only data requirement for this approach to work is to have the same instrumental variable in the both datasets. I restrict the samples to children born 60 months before and after the cutoff, October 1983. This choice guarantees the same set of instrumental variables is observed in both samples. Then the two-sample IV estimator can be applied to estimate the fuzzy design.

### 3.4 Choice Model

Without imposing a specific model, the previous analysis using the regression discontinuity design helps us understand the “effects of causes.” (Holland, 1986) In this section, I present a model that jointly quantifies the choices of food consumption and medical expenses. Through the model-based estimation results, we are able to understand the “causes of effects.” (Heckman, 2008)

The goal of the structural model is to separate the moral hazard effect from the net-wealth effect. A model-based approach is a natural tool for this purpose. Firstly, the household’s food expenditure is not directly observed in the data, similar to the case of Berry (1992), where he does not observe the profits of the firms. Secondly, a structural model can be used to evaluate the relative importance of different channels of the effects. An example is Eckstein and Lifshitz (2011). Using a dynamic discrete choice model, they evaluate the relative importance of the proposed explanations why the female labor force participation rate and employment rate have changed dramatically in the past century.

To model the trade-offs between food expenditure and medical expenses faced by the low-income households, I develop a model that builds on the ideas by Einav et al. (2013), Bajari et al. (2010), and Cardon and Hendel (2001).

I incorporate the joint decisions of food expenditure and insurance coverage choice in a two-period model. In the first period, a household chooses the type of insurance coverage for their
children. They choose between private coverage, public coverage, and no coverage. In the second period, the households choose the optimal food expenditure and the medical expenses for their children.

I assume the second-period utility function for the households is additively separable in food consumption and medical expenses:

\[
U(m, B, c) = \frac{1}{1 - \gamma_1} (m - \mu)^{1 - \gamma_1} + \frac{\gamma_2}{1 - \gamma_1} (B - \eta)^{1 - \gamma_1} + \frac{\gamma_3}{1 - \gamma_1} c^{1 - \gamma_1},
\]

where \( m \) denotes medical expenses, \( B \) denotes food expenditure, and \( c \) denotes the consumption of composite goods. The utility function is a special case of the functional form employed by Bajari et al. (2010). They show that a general version of the utility function is non-parametrically identified.

Following the ideas by Einav et al. (2013), I introduce the non-additive shocks to the utility functions. The parameters \( \mu \) and \( \eta \) are the shifters of the choice parameters in the utility function. The parameter \( \eta \) captures the shocks to the baseline food consumption, while the parameter \( \mu \) captures the health shocks. I assume that these two random variables are jointly normally distributed,

\[
\begin{pmatrix}
\eta_i \\
\log \mu_i
\end{pmatrix} \mid X_i \sim \mathcal{N}\left(\begin{pmatrix} X_{1i} \beta \\ X_{2i} \alpha \end{pmatrix}, \Sigma \right) 1(\eta_i \geq 0),
\]

where the random shock \( \eta_i \) is truncated above 0.

The correlation between the two random variables captures the fact that obesity is correlated with one’s health status. Obese children are likely to have higher medical utilization. However, I am concerned that the level of medical utilization may not be correlated to obesity in a linear fashion. In this regard, it is important to allow individual heterogeneity in the model. The stochastic structure permits the random variables, \( \eta \) and \( \mu \), to vary in the population conditional on a set of observed demographic characteristics. Furthermore, the random variable \( \eta \) is a shock to the sustainable food expenditure. Conditional on \( \log \mu \), \( \eta \) follows a truncated normal distribution. This feature allows the optimal food expenditure to be normally distributed and forbids a negative shock on food expenditure. The random variable \( \mu \), on the other hand, shifts the optimal medical
expenses. The log normal distribution assumption imposed on the random variable $\mu$ allows the optimal medical expenses to resemble the data: a great portion of observations have zero medical expenditure while a small portion have large expenditure.

In addition, the stochastic structure allows for an exclusion restriction that shifts the mean of $\eta$ but not of $\log \mu$. In the estimation, I assume the employment status of the household head affects the shocks on food but not the shocks on medical expenses.

The variance-covariance matrix is parametrized as:

$$
\Sigma = \begin{pmatrix} 
\sigma^2 + \delta^2 & \delta \\
\delta & 1 
\end{pmatrix}.
$$

The parametrization significantly expedites convergence of the search routine. It also implies that the variance-covariance is positive definite without restrictions on the parameter space.

The average shock on the medical expenditure is determined by $\delta$ and $X_i\beta$:

$$
\log \mu_i|\eta \sim N\left(X_2\alpha + \frac{\delta}{\sigma^2 + \delta^2}(\eta_i - X_1\beta), \frac{\sigma^2}{\sigma^2 + \delta^2}\right)
$$

This result implies that

$$
E[\mu_i|\eta] = \exp\left(X_2\alpha + \frac{\delta}{\sigma^2 + \delta^2}(\eta_i - X_1\beta) + \frac{\sigma^2}{2(\sigma^2 + \delta^2)}\right)
$$

By the iterated expectation, $E[\mu_i] = E[E(\mu_i|\eta_i)]$ can be obtained through integrating out the marginal distribution of $\eta_i$.

The households face a constrained maximization problem:

$$
\max_{m, B, c} \quad U(m, B, c) \text{ subject to } y_i - p_j = \phi_j m + B + c, \quad B \geq \lambda, \quad c \geq 0, \quad \text{and } m \geq \mu,
$$

where $m$ is the level of medical utilization, $\phi_j$ is the co-insurance rate for the health insurance $j$, and $p_j$ is the premium. $\phi_j m$ represents the out-of-pocket expenditure. Because I do not observe the health insurance premiums for each observation in the MEPS, I combine the data with the
information provided in the insurance component table of the MEPS. I use the regional weighted average of the employee contribution distributions (in dollars) for private-sector employees enrolled in family coverage to proxy for premiums for the private insurance coverage. I use the weighted average co-insurance rate of the private insurance coverage from the MEPS as the proxy for the co-insurance rate for the private insurance. The co-insurance rate for the public insurance, on the other hand, is proxied by the maximum nominal co-insurance rate mandated by the Federal government.

The constrained maximization problem states that the household maximizes its utility by choosing medical expenses and food consumption for their children given the budget constraint, where the household income can be separated into medical expenses, food expenditure, other consumption, and fixed costs of insurance packages.

Consider the internal solution:

\[
B^* = \frac{y - p_j - \phi_j \mu + \left[ \phi_j \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} + \left( \frac{\gamma_2}{\gamma_1} \right)^{\frac{1}{\gamma_1}} \right] \eta}{1 + \phi_j \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} + \left( \frac{\gamma_2}{\gamma_1} \right)^{\frac{1}{\gamma_1}}},
\]

\[
m^* = \mu + \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} \left( \frac{y - p_j - \phi_j \mu + \left[ \phi_j \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} + \left( \frac{\gamma_2}{\gamma_1} \right)^{\frac{1}{\gamma_1}} \right] \eta}{1 + \phi_j \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} + \left( \frac{\gamma_2}{\gamma_1} \right)^{\frac{1}{\gamma_1}}} - \eta \right),
\]

\[
c^* = \left( \frac{\gamma_3}{\gamma_2} \right)^{\frac{1}{\gamma_1}} \left( \frac{y - p_j - \phi_j \mu + \left[ \phi_j \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} + \left( \frac{\gamma_2}{\gamma_1} \right)^{\frac{1}{\gamma_1}} \right] \eta}{1 + \phi_j \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} + \left( \frac{\gamma_2}{\gamma_1} \right)^{\frac{1}{\gamma_1}}} - \eta \right).
\]

The internal optimal solution shows some interesting patterns: First, the joint distribution of the “shocks” leads to a trade-off between food expenditure and medical expenses. The larger the shock \(\eta\), the larger the optimal food consumption \(B^*\), at the same time, the smaller the optimal medical expenses \(m^*\). The larger the shock \(\mu\), the smaller the optimal food consumption \(B^*\), but the larger the optimal medical expenses. Second, the moral hazard effect of public insurance comes into the model through the role that the co-insurance rate \(\phi_j\) plays in the model. The closer the co-insurance rate \(\phi_j\) is to 1, the greater the “surplus” medical spending is. On the other hand,
the net-wealth effect goes through the insurance premium \( p_j \). Because the states are only allow to charge limited premiums on specific groups of the enrollees, in particular with family income above 150% FPL, the cohort under study is not required to pay any premiums. When a household switches from their original private insurance to Medicaid, its disposable income increases, which, in turns, leads to an increase in their optimal medical expenses, food expenditure, and composite consumption, \textit{ceteris paribus}.

I also calculate the Kuhn-Tucker conditions for boundary solutions. The second-period optimal choice is determined by comparing the utility level under the internal solution and the boundary solutions.

If the optimal food expenditure exceeds a certain threshold as a function of \( Z \), the child falls in the obesity category:

\[
B^*(\mu_i, \eta_i) > h(Z_i).
\]

Specifically, I assume that the cutoff is a linear function of the socioeconomic characteristics \( h(Z_i) = Z_i \gamma \). This specification implicitly assumes that an increase in food spending leads to an increase in chances of obesity. For the low-income population, that obesity rates increase as income increases is well-documented in the literature (Akee et al., 2010; Chang et al., 2005) I also observe a similar pattern in my dataset. Table 15 shows an descriptive evidence that chances of having a obese child are greater in a household below the 100% Federal poverty threshold with higher household income per capita. Figure 11 visualizes the pattern. The point is the mean obesity rates for each income category, and the line range shows one standard error away from the mean. The model allows the obesity cutoff to vary based on the demographic information.

We can estimate the probability of obesity given insurance choice:

\[
\Pr(\text{obesity} = 1|\text{insurance} = j) = \int 1\{B^*(\mu_i, \eta_i) > h(Z_i)\} dF(\mu_i, \eta_i).
\]

In the first period, the households face a standard insurance coverage choice. Consider the set of coverage choice \( J \in \{\text{private, public, no insurance } \} \). Each household will evaluate its expected
utility from each option:

\[ \nu_j(\epsilon_{ij}; y_i, X_i) = v_j(y_i, X_i) + \epsilon_{ij} = -\int \exp \left( -\psi U^*(\mu_i, \eta_i; y_i, X_i) \right) dF(\mu_i, \eta_i) + \epsilon_{ij}. \]

The indirect utility is transformed by the CARA utility function. The random variable \( \epsilon_{ij} \) is the policy-specific shocks, which are assumed to follow an independent Type 1 Extreme Value distribution. The optimal coverage choice is

\[ j^*(\mu_i, \eta_i; y_i, X_i) = \arg \max_{j \in J} \nu_j(\mu_i, \eta_i; y_i, X_i). \]

The Type 1 Extreme Value assumption leads to the standard logit form for the probability of insurance choice. The probability of individual \( i \) chooses insurance \( j \) can be calculated by

\[ Pr(\text{coverage} = j|\mu_i, \eta_i) = \int 1(\nu_j > \nu_k, \forall k \neq j) g(\epsilon_{ij}) d\epsilon_{ij} = \frac{\exp \{v_j(y_i, X_i)\}}{\sum_{k \in J} \exp \{v_j(y_i, X_i)\}}. \]

The joint probability of obesity and insurance choice \( j \) is given by

\[ Pr(\text{coverage} = j \text{ and obesity} = k) = Pr(\text{obesity} = k|\text{coverage} = j) \cdot Pr(\text{coverage} = j). \]

For each individual \( i \), I calculate the predicted errors in terms of choice probability and expected medical expenses simulated from the model:

\[
\begin{pmatrix}
\Pr_i(\text{obesity} = 0, \text{insurance} = 1|\theta) - 1_i(\text{obesity} = 0, \text{insurance} = 1) \\
\vdots \\
\Pr_i(\text{obesity} = 1, \text{insurance} = J|\theta) - 1_i(\text{obesity} = 1, \text{insurance} = J) \\
\Pr_i(\text{obesity} = 0, \text{insurance} = 1|\theta) \cdot m^*_i(\theta) - 1_i(\text{obesity} = 0, \text{insurance} = 1) \cdot m_i \\
\vdots \\
\Pr_i(\text{obesity} = 1, \text{insurance} = J|\theta) \cdot m^*_i(\theta) - 1_i(\text{obesity} = 1, \text{insurance} = J) \cdot m_i
\end{pmatrix},
\]

where \( 1_i(\cdot) \) is an indicator function, \( \theta \) is the vector of parameters of interest, \( m^* \) denotes the simulated medical expenses, and \( m \) is the observed medical expenses.
The moment function $G(\theta_0)$ can be defined as

$$G(\theta_0) = E[W_i \otimes u_i(\theta_0|y_i, X_i, Z_i)] = 0$$

Following Cardon and Hendel (2001), I use the demographic information as instruments, $W_i$. In addition, considering the fact that the employment status of the household head is exogenously determined given the demographics, the employment status can be used as an instrument as well. I have six instruments: age, age squared, sex, hispanics, unemployment status, and a constant\(^6\).

Given the predicted errors, the algorithm minimizes the objective function:

$$Q_N(\theta|y, X, Z, W) = \left[ \frac{1}{N} \sum_{i=1}^{N} W_i \otimes u_i(\theta|y_i, X_i, Z_i) \right]' \left[ \frac{1}{N} \sum_{i=1}^{N} W_i \otimes u_i(\theta|y_i, X_i, Z_i) \right].$$

Overall, the model is estimated using the Method of Simulated Moments. I estimate four structural parameters, two vectors of the mean shifters, four covariance parameters, and a vector of the obesity cutoff.

4 Data

To assess the impact of Medicaid eligibility on childhood obesity, I draw my conclusion on data from the Medical Expenditure Panel Survey. It is a set of large-scale surveys of families and individuals, their medical providers (doctors, hospitals, pharmacies, etc.), and employers across the United States. The MEPS contains detailed information on children’s height and weight, the poverty levels of their families, and their birth date. I use the datasets from 1996 to 2001. Because obesity takes time to develop, it is reasonable to look at the policy effect some years after implementation.

A good measure of childhood obesity is based on the Body Mass Index (BMI). Overweight and obesity are defined using Centers for Disease Control and Prevention (CDC) age- and sex-specific growth charts for BMI. An example of the 2000 CDC growth chart is shown in Figure

\(^6\)I do not use the region dummies as instruments because of the data restriction. In order to obtain the approximate health premiums and the co-insurance rates, the premium and the co-insurance rate data are averaged to the region level and then matched to the MEPS. Therefore, the lack of household variation in the premium data prevents me from using region dummies as instruments.
These growth charts are based on data from sequential evaluations of nationally representative samples of children in the United States in the past four decades (the National Health and Nutrition Examination Surveys (NHANES) and the National Household Education Surveys (NHES)). The NHES II and the NHES III were conducted in 1963 - 1965 and 1966 - 1970. The NHANES I, II, and III were conducted in 1971 - 1974, 1976 - 1980, and 1988 - 1994. Because of concern of the under classification of overweight, the children greater than or equal to six years of age in the NHANES III are excluded from the BMI growth charts. A child is overweight if his BMI is above 85th percentile among children of the same sex and age of the reference group. A child is obese if his BMI is above 95th percentile. For example, a 10-year-old boy with a BMI of 21 would be categorized as overweight while a 8-year-old boy with the same BMI would be in the obese category. By late adolescence, the 95 percentiles approach the commonly used measure for adult obesity: $\text{BMI} \approx 30$. The 2000 CDC growth charts provide a more accurate measure of obesity of children and adolescents\(^7\).

Table 2 shows the characteristics of the children in the MEPS samples. Samples are restricted to those between age 3 and age 19. Because the expansions of SCHIP up to 1999 extended eligibility to age 19 (See Table 1), the selection guarantees the individuals were eligible when observed in the data. From 1996 to 2001, there was a gradual increase in the obesity rate. It increased from 19.6% to 22.2% in 5 years. The obesity rate for whites is the lowest compared with hispanics or blacks. The hispanics had the largest growth in obesity prevalence from 25% to almost 30%. A similar pattern is observed for the prevalence of overweight as well.

To estimate the impact of insurance take-up on childhood obesity, I combine information contained in two different datasets. I use the information of obesity in the MEPS and the information of insurance coverage in the Survey of Income and Program Participation 1992, 1993, and 1996. SIPP provides detailed information on insurance coverage of children in 1992, 1993, or 1996. Because I also observe children’s month of birth in SIPP, the same set of instruments — eligibility status and a flexible function of the month of birth — is observed in both SIPP and MEPS. Combining the obesity status in the late 1990s in the MEPS with the information of insurance coverage in the

\(^7\)For details about the CDC BMI-for-Age growth charts, see Kuczmarski et al. (2002)
early 1990s identifies the long-term impact of health insurance for the early take-up cohort.

Table 3 shows the summary statistics of the panels in the SIPP. The samples are restricted to those aged between 3 and 19 for the same reason described before. Medicaid coverage increased by 3 percentage points between 1992 and 1993 while it remained at a similar level from 1993 to 1996. Insurance coverage, on the other hand, is approximately 2 percentage points higher in 1996 than in 1992.

To estimate the choice model, I use the main dataset from the MEPS 1997 to 2000. I use the subsample of children who are eligible for Medicaid. In order to estimate the full choice model, I need the information on the household income. I construct the household income from the household dataset. Some interviewees did not provide credible information on their income. I compare household income with the Federal Poverty Level for each year. The observations that do not have income information that match the FPL requirements are dropped. After data attrition and sample selection, I am left with 1959 observations for the estimation of the choice model.

5 The Effect of Medicaid Eligibility on Childhood Obesity

5.1 Graphical Analysis

An effective and diaphanous way to examine whether there is a policy effect is to plot the conditional obesity rates given months born before/after October 1983. In the graph, I fit the conditional obesity rates with three local polynomials: one polynomial is fitted using the data points to the left of the cutoff, the second one is fitted using the data points to the right of the cutoff, and the third polynomial is fitted using the full sample. The limit at the boundary is the effect of a RD design. This method is preliminary because local polynomials do not fit well at the “boundary.” A more rigorous method is required to provide a credible answer. I fit the data points with different degrees of polynomials. In general, the literature prefers using under-smoothing functions to fit the conditional mean function (Lee and Lemieux, 2010). Shown in Figure 6, I use a linear fit, squared polynomial, and a cubic polynomial to fit the conditional means. The figures suggest preliminarily that the eligibility cutoff has a positive effect on obesity rate.
The conditional means show some seasonality pattern of the prevalence of obesity. I define the outcomes as the 12-month-difference of the obesity rates. For example,

\[
\text{Differenced obesity rates in October 1983} = \text{obesity rate in October 1983} - \text{obesity rate in October 1982}.
\]

Because of the upward trend in obesity rates, I expect to see the differenced obesity rates to fluctuate around a small positive number. In Figure 7, I fit the local averages of the observations born one before the cutoff, one year after the cutoff, and more than one year after the cutoff. Consistent with the previous finding, the local average estimated by observations between October 1983 and September 1984 is higher than the local averages estimated by other observations. The cohort born between October 1983 and September 1984 shows the difference between the eligible and the ineligible. The pattern that the local average of this cohort is larger suggests a positive policy effect at October 1983.

5.2 Sharp Regression Discontinuity Design

In Table 4 are the estimates of the effect of Medicaid eligibility on childhood obesity using the sharp RD design. I estimate the effect on two measures of outcomes: obesity and overweight. In order to show the results are robust against different parametric assumptions on the conditional moment functions, I estimate the model using lower-order polynomials of different degrees. In order to account for possible misspecification of the functional forms, I follow Lee and Card (2008) to cluster the standard errors at each value of month of birth. This simple remedy comes with the cost of an additional assumption that the specification errors of the conditional mean functions on the both sides of the cutoff are the same.

For the obesity model, I find the effects range from 6 percentage points to 17 percentage points and are mostly statistically significant. Except for the squared polynomial, I find statistically positive effects. The results suggest ex-ante moral hazard is the dominant factor that affects childhood obesity. Because the RD designs imitate local randomized experiments, inclusion of conditioning covariates is irrelevant (Lee and Lemieux (2010)). Conditioning on demographic covariates is likely to alleviate small-sample bias (Imbens and Lemieux, 2008). I also report estimates conditioning on
a set of dummies for age, sex, census regions, race, hispanic, month of birth, and survey years. In
general, the estimates would not differ much. If it does, the estimate conditional on demographics
would be the preferred estimate. In the obesity model, I find no conspicuous differences. Except
for the specifications of polynomial degree of two and six, the difference between the estimates is
around 0.01.

The second column of Table 4 reports the results of Medicaid eligibility on overweight prevalence.
The effect of Medicaid eligibility is much milder for this outcome and susceptible to the functional-
form assumption. The statistically significant estimates range from 10 percentage points to 17
percentage points. Except for the specification of polynomial of degree 2, the other estimates
suggest a positive effect. Again, the results suggest the ex-ante moral hazard is the dominant factor.
Comparing the estimates for the both outcomes shows that offering public insurance to low-income
children has a stronger effect on obesity (BMI greater than 95 percentile) than overweight (BMI
greater than 85 percentile).

5.3 Regression-Adjusted Difference-in-Difference

In order to show my main results are robust against small perturbation of econometric assumptions,
I estimate the effect using Difference-in-difference. I essentially estimate the following regression:

\[ Y = \beta_0 + \beta_1 D_1 + \beta_2 D_2 + \beta_3 D_1 \cdot D_2 + g(x; \theta) + W \gamma + \epsilon \]  

(7)

where \( D_1 \) is an indicator for below the poverty threshold and \( D_2 \) is an indicator for born after
October 1983. \( \beta_3 \) is the treatment effect of interest. \( g(x) \) is a flexible function of the number of
months born before/after October 1983. The idea of this \( g(x) \) function, following Card and Shore-
Sheppard (2004), is to shift the source of identification from a global diff-in-diff to a local one. In
the estimated model, \( g(x) \) is specified as a polynomial of degree 3.

Table 5 shows the DD estimates. I cluster the standard errors at the level of Census regions
under the assumption that prevalence of obesity is correlated over time within each region. The

\[ I also use different orders of polynomials. The estimates are quite stable around 5 percentage points for obesity
and 1 percentage points for overweight.\]
results suggest the Medicaid expansions have a roughly 6-percentage-point positive effect on obesity and a 2-percentage-point positive effect on overweight. While the effect of public insurance is still statistically significant for obesity, it is not statistically for overweight. The estimates are smaller than the RD estimates for both obesity and overweight models. Compared with the RD design, an advantage for the Diff-in-Diff is that it utilizes a larger sample. But it also requires an additional assumption that the outcomes for the control group (100% - 133% FPL) and the treatment group (below 100% FPL) have a similar trend. Both Diff-in-Diff estimates and RDD estimates suggest a statistically significant positive effect of Medicaid eligibility on childhood obesity rates.

5.4 Specification Test

An alternative approach to estimate the RD designs is local linear regressions. An advantage of this approach is that it restricts the source of identification around the cutoff. On the other hand, the polynomial regressions allow observations farther away from the cutoff to have small weights. Its disadvantage is that relatively few data points are used in estimation. The problem of small sample bias is likely to arise. I work with the following specifications:

\[ Y = \beta_0 + \tau \cdot D + \beta_1 D \cdot x + \beta_2 x + \epsilon, \quad -h \leq x \leq h. \]

This specification allows the model to have different slopes on both sides of the cutoff. Small sample size also leads to lack of power. As suggested in statistics literature (Fan and Gijbels, 1996; Cheng, Fan, and Marron, 1997), the triangular kernel-weighted regressions are boundary-optimal. I also estimate the local linear regressions weighted by the triangle kernel.

The results are shown in Table 6. The left panel shows the results weighted by rectangle kernel. The estimates generally suggest positive association between eligibility and obesity. The right panel shows the estimates weighted by the triangular kernel. The magnitude of the estimates does not change much, but the standard errors fall greatly. The results support the main result using polynomial regressions.
5.5 Placebo Test

A potential concern of the result is that, rather than picking up the “true” effect of Medicaid, the estimates pick up the upward trend of childhood obesity. In order to address this concern, I apply the graphical analysis to children from families between 100% and 199% FPL. Figures ?? show that the jump between the two polynomials on the both sides of the cutoff becomes less and less evident when the fitted polynomials under-smooth the data points. This pattern suggests that it is likely that there is no discontinuity at the cutoff for children from families between 100% and 199% FPL.

The second placebo test I implement is a counterfactual cut-off test. I assume a counterfactual policy that sets the eligibility cutoff six months and one year before the actual policy. That is, I apply the same sharp RD design to the cutoff in May 1983 and October 1982. The results are reported in Table 7. For the estimates using May 1983 as the cutoff, the only statistically significant estimates are the models without conditioning covariates. They are only statistically significant at 10% level. For the estimates using October 1982 as the cutoff, some specifications show statistical significance. However, the estimates are rather sensitive to the polynomials that are used to approximate the obesity trend. In general, I do not observe consistent evidence that there is a policy effect at the counterfactual cutoffs.

5.6 Estimating Obesity-induced Cost Increase

Combined with previous findings, my estimates are informative of the expected increase in future medical expenditure caused by Medicaid expansions through the channel of increasing obesity rates. A review of international medical literature by Serdula et al. (1993) concludes that 42 - 63% of obese school age children were obese as adults. In particular, a study follows school-aged children in the United States to their forties and early fifties finds that 63 percent of obese children become obese adults. In addition, Cawley and Meyerhoefer (2012) use an instrumental variable approach to estimate the effect of obesity on medical expenditure. Their estimates suggest that obesity causes an average increase of $3674 for the Medicaid subsample and an average increase of $2741 for the full sample. Their estimates for the Medicaid subsample ranges from $857 (least squares estimate)
to $3674 (Two-part model estimate). My estimates of the effect of Medicaid offering on obesity rates ranges from 5 percentage points (square polynomial with controls) to 16.6 percentage points (quartic polynomial without controls).

Combining the aforementioned estimates, I can calculate how Medicaid expansion may affect future medical expenses through obesity. A simple formula as follows gives an approximate quantity:

\[
\text{Obesity-induced cost increase} = \text{Percentage-Point Increase in Childhood Obesity Rate} \\
\times \text{Proportion of Obese Children Remaining Obese in Adulthood} \\
\times \text{Increase in Medical Care Costs associated with Adult Obesity}
\]

Lower Bound = 5 percentage points \times 63\% \times $857 \approx $27

Upper Bound = 16.6 percentage points \times 63\% \times $3674 \approx $384.

The simple back-of-the-envelope calculation shows that Medicaid expansions increase the expected Medicaid costs by as much as $384 per person annually through the channel of increasing childhood obesity rates. Considering the per capita health care spending in 2001 is $3298 on average (Thorpe et. al, 2004), the expected annual health care spending could increase by as much as 12%.

5.7 Alternative Outcome Measure: Excess BMI

In this section, I use an alternative outcome measure for the effect of obesity. I define an excess BMI as the BMI points above obesity cutoff given age and gender:

\[
\text{Excess BMI} = \max\{\text{BMI} - \text{Obesity Cutoff BMI}, 0\}
\]

Most of the children have a zero excess BMI while there are a few morbidly obese children who have a large excess BMI. The quantile analysis, which is robust against outliers, is a natural tool in this context. I use the same specification as the RD design and estimate the effect at 85-, 90-, and
95-percentiles. The results are reported in Table 8. The results suggest that Medicaid eligibility increases excess BMI by roughly 1 to 3 points at 90- and 95- percentiles. This finding is consistent with the previous finding that uses the obesity indicator as the outcome variable.

5.8 Heterogeneous Effects

A straightforward extension of the previous model is to investigate heterogeneity of the policy effects. Firstly, I apply the sharp RD design specifically to each race cohort. The estimates can be interpreted as the race-specific effects. This question is important because obesity and overweight prevalence in the U.S. show significant disparities between race/ethnicity groups. Table 9 is excerpted from Singh et al. (2010), which summarizes data from the National Survey of Child’s Health 2003 and 2007. They find that the chances of obesity for blacks and hispanics are 10 percentage points higher than the whites. The disparities grew significantly from 2003 to 2007. The inequalities lead to an immediate interest for how the previous results can be decomposed to different race/ethnicity groups.

The estimates shown in Table 10 are estimated by a sharp RD design. Because the estimation is restricted to each race/ethnicity group, each regression has smaller number of observations. It makes sense to be more cautious about the small sample bias. Therefore, I include demographic covariates in each regression. The results show that the effect is the most severe among low-income non-hispanic whites. The effect can be as large as 25 percentage points. For the non-hispanic blacks, I also observe strong positive effects. Being eligible for Medicaid increased the chances of obesity by roughly 20 percentage points. A modest positive effect is observed for hispanic children. Consistent with the regressions of obesity, the models of overweight also show that the policy effect is the strongest among the whites. But the results are rather sensitive to the functional-form assumption. That the policy effect is the least severe among hispanic children may be explained by the fact that they have the highest obesity prevalence to begin with. Public insurance eligibility induces a relatively small behavioral change.

Alternatively, I estimate the sharp RD design using male and female subsamples. As shown in Table 11 I find Medicaid eligibility induces an increase in prevalence of obesity for both genders.
But the effect is much stronger for male than female. Consistent with previous results, the effect of Medicaid eligibility on prevalence of overweight is smaller than the effect on obesity rate and is more sensitive to parametric assumptions.

Comparing the results in the obesity and the overweight models, I conjecture that the policy effects are also heterogeneous across different levels of the BMI. To test this hypothesis, I apply a quantile regression to estimate the sharp RD design, where the dependent variable is the logarithm of the BMI. The quantile regression RD design estimates the distributional treatment effect of Medicaid eligibility on each percentile of the BMI. The results are plotted in Figure 8. The shaded area is the 90% confidence interval. The standard errors are estimated using kernel estimates. Two vertical lines are added to the plot. The vertical line to the left represents the 62 percentile of the log-BMI. Because the rate of overweight is roughly 38%, the vertical line approximates the overweight cutoff. The vertical line to the right represents 81 percentile of the log-BMI. The line approximately represents the obesity cutoff. As shown in the graph, the effect of Medicaid eligibility is in general positive over the BMI distribution. The effect is larger for children with larger BMI. The effect results in shifting the distribution of BMI in a skewed fashion: the heaviest children would become heavier. This fact is well-documented in physiology literature (Ebbeling et al., 2002). The result indirectly supports the argument that the adverse effect of Medicaid on obesity is greater for children with unhealthy diet.

Table 12 shows the corresponding results at specified points in the distribution of log-BMI. I also estimate the quantile regression using different subsamples. Except for the female subsample, of which all the estimates are statistically insignificant, I observe a similar pattern for both male and hispanic subsamples.

6 The Effect of Insurance Take-up on Childhood Obesity

The effect of eligibility on childhood obesity has the “intent-to-treat” interpretation. However, in general, a more relevant question is the treatment effect of insurance coverage. This section provides
the first set of the direct estimates on the effect of insurance take-up on childhood obesity. The existing literature that estimates the effect of public insurance on obesity or BMI all focuses on estimating the “intent-to-treat” effect.

There are a great number of studies that estimate the effect of eligibility on insurance coverage. The effect of eligibility on coverage is not the focus of this paper. Table 13 shows raw estimates of the effect of eligibility on insurance coverage using SIPP 1996. They suggest a positive effect of eligibility on coverage. It can be considered as a follow-up study of Card and Shore-Sheppard 2004, which finds a modest positive impact of Medicaid eligibility on insurance coverage using SIPP 1992 and 1993. The estimates suggest that Medicaid eligibility is a relevant instrument for insurance coverage. As discussed before, the RD design imitates a local randomized experiment, which implies Medicaid eligibility is exogenous. Because insurance coverage is determined by other factors than the eligibility cutoff, the model is a fuzzy regression discontinuity design.

I estimate the fuzzy design using Angrist-Krueger TSIV approach to utilize information contained in two different datasets, which is a GMM estimator with the moment function:

\[
g(\theta) = \frac{Z'Y_1}{N_1} - \frac{Z'_2X_2\theta}{N_2}.
\]

The outcome vector \(Y\) is a vector of binary indicators for either obesity or overweight. The matrix of the instrument \(Z\) contains variables of eligibility status and a flexible function of number of months born before/after October 1983. The covariate matrix \(X\) contains variables of insurance coverage and a flexible function of the forcing variable. The main treatment variable here is insurance coverage, including both public and private packages. The reason to use this set of treatment variables is convenience of interpretation. The estimate for the treatment has a local average treatment effect interpretation, the same as the Wald estimator. The estimate for the effect of insurance coverage from the fuzzy design is therefore interpreted as the average effect of insurance coverage on those who switched from uncovered to covered because of eligibility gain. The TSIV requires the dimensionality of \(Z_1\) and \(Z_2\) are compatible. Therefore, I do not include any conditioning covariates in the model. By irrelevance of inclusion of conditioning covariates

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9 The existing literature that estimates the effect of public insurance on obesity or BMI all focuses on estimating the "intent-to-treat" effect.

10 For example, see Card and Shore-Sheppard (2004); Card, Dobkin, and Maestas (2008); De la Mata (2012)

11 Notice that this model can be extended to samples with sample weights by using weighted averages for each sample moment.
in the RD design, this step would not bias the estimates. Also, in order to guarantee the two samples have the same set of instruments, I restrict the sample to children born within a certain number of months. Intuitively, the sample selection would not bias the estimates because the source of identification of the RD designs is the observations around the cutoff. This selection can be considered as setting the weights for observations far from the cutoff to zero instead of giving a very small weight.

The estimates from TSIV is reported in Table 14. I implement the method using data from different years of SIPP. Each row shows the estimates from data of insurance coverage in SIPP 1992, 1993, or 1996. The second stage is estimated using obesity information in MEPS 1996 - 2001. Therefore, the interpretation of each row changes slightly. For example, the first row is the effect on those who took up insurance coverage by 1992. The last row is the effect on those who took up insurance coverage by 1996. The effects range from 1 percentage point to 6 percentage points, depending on the panel year and the parametric assumption. In general, the square polynomial regression models find the smallest effect. But all results are consistent with a positive effect of insurance take-up.

A similar pattern is found in the overweight models as well. All estimates suggest a positive effect of insurance take-up on prevalence of overweight. But the estimates are less stable. The estimates are fairly sensitive to functional-form specifications.

The main findings in this section suggest that public insurance coverage indeed induces obesity. The effect is more severe for children at risk of obesity than those at risk of overweight. It is consistent with the eligibility results. The difference may be explained by the reason that children of BMI right below 85 percentile have a better diet and exercise routine than those of BMI right below 95 percentile.

7 The Results of the Choice Model

The estimates for the choice model, obtained by estimating the joint decision of medical expenses, food expenditure, and composite consumption, are shown in Table 16. The model uses 500 simulation draws for each individual. The estimates for the structural parameters suggest that individuals
value composite consumption the most, roughly three times of the food spending and eight times of the medical expenses. The effects of demographics on health care consumption and food expenditure are calculated through their effects on the joint distribution of $\eta$ and $\mu$. The fact that most of the estimates are statistically significant suggests a great extent of heterogeneity. The estimates also suggest that hispanic women who are unemployed are more likely to received a large shock on food spending and that hispanic females are more likely to be obese.

As expected, the estimates for the variance-covariance matrix reveal a positive correlation between the shocks on medical expenses and the shocks on food consumption. Conditional on the demographic information, children who have higher sustainable level of food consumption are more likely to utilize health cares.

The main purpose of the choice model is to evaluate the relative importance of the net-wealth effect and the ex-ante moral hazard effect. These effects are not represented by a single parameter in the model. To answer this policy-relevant question, I use the model estimates to calculate the following average partial effects:

\[
\delta_{\phi_j} = \frac{1}{N} \sum_{i=1}^{N} \frac{\partial Pr^*(\text{obesity} = 1|\hat{\theta}; y_i, \phi_j)}{\partial \phi_j};
\]

\[
\delta_{p_2} = \frac{1}{N} \sum_{i=1}^{N} \frac{\partial Pr^*(\text{obesity} = 1|\hat{\theta}; y_i, p_2)}{\partial p_2};
\]

\[
\delta_y = \frac{1}{N} \sum_{i=1}^{N} \frac{\partial Pr^*(\text{obesity} = 1|\hat{\theta}; y_i, \phi_j)}{\partial y_i}.
\]

Analogous to Cardon and Hendel (2001), where they interpret moral hazard as the elasticity of demand for medical utilization with respect to co-insurance rates, the moral hazard in the context of my question can be interpreted as the effect of a small change in co-insurance rate on the probability of obesity. I measure the moral hazard effect by the quantity $\delta_{\phi_j}$, the average partial effect of the co-insurance rates on the probability of obesity. I also estimate the average partial effect of change in public insurance premiums. The estimate suggests that the effect of public insurance premium is more substantial than the change in co-insurance rate; however, the estimate is still not statistically
significant. Following the same logic, the quantity $\delta y$ is a measure of net-wealth effect.

Because the magnitude of the average partial effects depend on the measurements, I measure how responsive the chances of obesity are to the change in income and the generosity of insurance following the idea similar to elasticities:

$$
\epsilon_\phi = \frac{1}{N} \sum_{i=1}^{N} \left| \frac{\partial Pr^*(obesity = 1|\hat{\theta}; y_i, \phi_j, p_2)}{\partial \phi_j} \cdot \frac{\phi_j}{Pr^*(obesity = 1|\hat{\theta}; y_i, \phi_j, p_2)} \right|
$$

$$
\epsilon_{p_2} = \frac{1}{N} \sum_{i=1}^{N} \left| \frac{\partial Pr^*(obesity = 1|\hat{\theta}; y_i, \phi_j, p_2)}{\partial p_2} \cdot \frac{p_2}{Pr^*(obesity = 1|\hat{\theta}; y_i, \phi_j, p_2)} \right|
$$

$$
\epsilon_y = \frac{1}{N} \sum_{i=1}^{N} \left| \frac{\partial Pr^*(obesity = 1|\hat{\theta}; y_i, \phi_j, p_2)}{\partial y_i} \cdot \frac{y_i}{Pr^*(obesity = 1|\hat{\theta}; y_i, \phi_j, p_2)} \right|
$$

I simulate the average “elasticities” of chances of obesity with respect to a 10% change in co-insurance rates and income. Also, I simulate the change in chances of obesity with respect to a $1000 increase in public insurance premium.

The results of the estimates are reported in Table 17. The average partial effect of a slight increase in the generosity of insurance is negative on the prevalence of obesity; however, the effect is not statistically significant. The result is congruent with the previous findings by Bhattacharya et al. (2011). Their findings drawn on data from RAND Health Insurance Experiment do not suggest statistically significant effect of change in generosity of insurance on either chances of becoming obesity or BMI. On the other hand, a slight increase in household income raises the chances of obesity, and the effect is also statistically insignificant. However, we observe that the average partial effect of generosity of coverage is stronger in magnitude compared with the one of income.

The simulation results show that prevalence of childhood obesity is not responsive to either income or generosity of insurance coverage. The simulation results are also congruent with previous reduced-form findings (Rashad and Markowitz, 2007) that obesity rate is inelastic in response to insurance coverage. The results are also consistent with physiology literature (Ebbeling et al., 2002) that the childhood obesity epidemic can be primarily attributed to adverse environmental factors.
at home and in school.

A potential bias of the simulation results is that the structural model is agnostic about the supply side of the insurance market. The results are merely a partial equilibrium in which the current market structure of the insurance market is assumed to be fixed. In other words, the simulation does not take into account the possibility that private insurance companies will adjust their policies in response to the change of public insurance.

8 Concluding Remarks

This paper focuses on estimating the effect of public insurance on childhood obesity. The causal effect is identified by utilizing the federal Medicaid expansion in 1990. OBRA 1990 extended eligibility to children born after October 1983. By comparing children whose birth dates are close to the eligibility cutoff, I find strong evidence that public insurance expansions lead to significant ex-ante moral hazard problem. The results estimated from a sharp regression discontinuity design suggests eligibility for Medicaid in 1990 increases the odds of obesity by approximately 10 percentage points. It also leads to an approximately 7 percentage-point increase in the odds of being overweight.

By applying the sharp regression discontinuity design to gender and ethnic subsamples, I find conspicuous heterogeneous effects. The quantile regression estimates provide evidence that Medicaid eligibility induces obesity in a skewed fashion: greater effects on heavier children.

Following the argument that a fuzzy regression discontinuity design has a Wald-estimator interpretation, I apply Angrist-Krueger two-sample IV estimator to estimate the effect of early insurance take-up on obesity. I combine information of insurance coverage from SIPP 1992, 1993, and 1996 with information of obesity in MEPS 1996 - 2001. I find early insurance take-up has a positive effect on obesity. Early insurance take-up induced by Medicaid expansion in 1990 leads to approximately 3-percentage-point increase in later obesity rates. A similar but smaller effect is also found to the outcome of overweight. But the result is sensitive to the functional-form assumption. I summarize the estimates under different parametric assumptions in a box plot shown in Figure 10. Each point in the box plot is a point estimate using polynomials of different degrees to approximate the cutoff. The figure provides an easy comparison of different estimates.
To further investigate the question, I develop and estimate a choice model that quantifies risk preference, insurance coverage choice, and decisions on food expenditure and medical expenses. I use the model estimates to infer the relative importance of the channels through which the public insurance offerings lead to childhood obesity. The model suggests that moral hazard effect is more substantial than net-wealth effect.

My results have important policy implications. It suggests the existence of moral hazard and net-wealth effect. When the cost of being ill is lower, a child or his parent has less incentive for preventive efforts. In addition, when a more expensive private insurance package is replaced by public insurance, it increases disposable income for families with eligible children. It may lead to an increase in food consumption. Without improvement in diet or exercise routines, the net-wealth effect on food consumption worsens the obesity problem. My simulation suggests that obesity prevalence is relatively responsive to the change in premiums. It is suggestive of imposition of behavior-adjusted premiums in Medicaid. Besides, adult obesity is a strong predictor for a number of chronic illnesses. Because childhood obesity is highly correlated with adult obesity, my result implies potentially higher future health costs. A simple back-of-the-envelope calculation based on my estimates suggests that childhood obesity associated with Medicaid expansion could potentially increase medical costs by $384 annually. I also find evidence that the primary source of the increase in utilization of preventive care comes from increasing accessibility through Medicaid expansion. There is little change in utilization of those who already had access. This result suggests that it may be necessary to improve the preventive care mandated by the federal Medicaid guidelines to facilitate information flow to low-income households.

A caution about interpreting the result is its external validity. Specifically, all estimates provided in this paper focus on the effects on low-income children. Children from low-income families may be different from an average children in the U.S. society. Children from low-income families tend to have less educated parents and are more likely to be obese to begin with. A significant ex-ante moral hazard effect to this cohort may not suggest the existence of ex-ante moral hazard to other subpopulations. However, while recent health policy reforms primarily focus on increasing access to medical care for low-income families, ex-ante moral hazard indeed imposes an external cost on
all tax-payers and raises a concern about rising long-term health costs.

References


[26] John Geweke. Efficient simulation from the multivariate normal and student-t distributions subject to linear constraints and the evaluation of constraint probabilities, —1998—.


A Kuhn-Tucker Conditions

In this section, I present the solutions to the Kuhn-Tucker conditions for the choice model.

\[
\max_{m, B, c} U(m, B, c) = \frac{1}{1 - \gamma_1} (m - \mu)^{1 - \gamma_1} + \frac{\gamma_2}{1 - \gamma_1} (B - \eta)^{1 - \gamma_1} + \frac{\gamma_3}{1 - \gamma_1} c^{1 - \gamma_1}
\]  
subject to \[ y_i - p_j = \phi_j m + B + c \]  
\[ B \geq \eta \]  
\[ c \geq 0 \]  
\[ m \geq \mu. \]  

Note that, by the “no-cash-left-on-the-table” principle, the budget constraint \( (9) \) should always be binding:

1. Constraints \( (10), (11), (12) \) are non-binding:

\[
B^* = \frac{y - p_j - \phi_j \mu + \left[ \phi_j \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} + \left( \frac{\gamma_1}{\gamma_2} \right)^{\frac{1}{\gamma_1}} \right] \eta}{1 + \phi_j \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} + \left( \frac{\gamma_1}{\gamma_2} \right)^{\frac{1}{\gamma_1}}},
\]  
m^* = \mu + \left( \frac{1}{\phi_j \gamma_2} \right)^{\frac{1}{\gamma_1}} (B^* - \eta),
\]  
c^* = \left( \frac{\gamma_3}{\gamma_2} \right)^{\frac{1}{\gamma_1}} (B^* - \eta).
2. Constraints (11) and (12) are non-binding:

\[ m^* = \frac{y - p_j - \eta + (\phi_j \gamma_3)^{\frac{1}{\gamma_1}} \mu}{\phi_j + (\phi_j \gamma_3)^{\frac{1}{\gamma_1}}}, \]
\[ B^* = \eta, \]
\[ c^* = (\phi_j \gamma_3)^{\frac{1}{\gamma_1}} (m^* - \mu). \]

3. Constraints (10) and (12) are non-binding:

\[ m^* = \frac{y - p_j - \eta + (\gamma_2 \phi_j)^{\frac{1}{\gamma_1}} \theta}{\phi_j + (\gamma_2 \phi_j)}, \]
\[ B^* = \eta + (\gamma_2 \phi_j)^{\frac{1}{\gamma_1}} (m^* - \mu), \]
\[ c^* = 0. \]

4. Constraints (10) and (11) are non-binding:

\[ m^* = \mu, \]
\[ B^* = \frac{y - p_j - \phi_j \mu + (\gamma_2 \phi_j)^{\frac{1}{\gamma_1}} \eta}{1 + (\gamma_2 \phi_j)^{\frac{1}{\gamma_1}}}, \]
\[ c^* = \left(\frac{\gamma_3}{\gamma_2}\right)^{\frac{1}{\gamma_1}} (B^* - \eta). \]

5. Constraint (10) is non-binding:

\[ m^* = \mu, \]
\[ B^* = y - p_j - \phi_j \mu, \]
\[ c^* = 0. \]

6. Constraint (11) is non-binding:

\[ m^* = \mu, \]
\[ B^* = \eta, \]
\[ c^* = y - p_j - \eta - \phi_j \mu. \]

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7. Constraint \( (12) \) is non-binding:

\[
\begin{align*}
m^* &= y - p_j - \eta \frac{\phi_j}{\phi_j}, \\
B^* &= \eta, \\
c^* &= 0.
\end{align*}
\]

8. All constraints are binding:

\[
\begin{align*}
m^* &= \mu, \\
B^* &= \eta, \\
c^* &= 0.
\end{align*}
\]

B Estimation Algorithm

The choice model is estimated in the following steps:

**Step 1** Given the parameters, generate 500 draws for \( \eta \) and \( \log \mu \) are generated from a truncated bivariate normal distribution using Gibbs Sampling algorithm (Geweke, 1998):

\[
\begin{pmatrix}
\eta_i \\
\log \mu_i
\end{pmatrix} \mid X_i \sim \mathcal{N} \left( \begin{pmatrix} X_{1i} \beta \\ X_{2i} \alpha \end{pmatrix}, \Sigma \right) 1(\eta_i \geq 0),
\]

**Step 2** Given the random draws for \( \eta \) and \( \mu \), solve for the optimal solutions to the constrained utility maximization problem:

\[
\begin{align*}
\max_{m, B, c} & \quad U(m, B, c) = \frac{1}{1 - \gamma_1} (m - \mu)^{1 - \gamma_1} + \frac{\gamma_2}{1 - \gamma_1} (B - \eta)^{1 - \gamma_1} + \frac{\gamma_3}{1 - \gamma_1} c^{1 - \gamma_1} \\
\text{subject to} & \quad y_i - p_j = \phi_j m + B + c \\
& \quad B \geq \eta \\
& \quad c \geq 0 \\
& \quad m \geq \mu.
\end{align*}
\]

The optimal behaviors are solved by comparing the utility level among the interior solutions and the boundary solutions.

**Step 3** Calculate the obesity probability given the optimal food expenditure:

\[
\Pr(\text{obesity} = 1 | \text{insurance} = j) = \int 1 \{ B^*(\mu_i, \eta_i) > h(Z_i) \} \, dF(\mu_i, \eta_i).
\]

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The random draws from the truncated bivariate normal distribution are used to compute the Monte Carlo Integration.

**Step 4** Calculate the second-stage expected utility under alternative insurance coverages:

\[
\nu_j(\epsilon_{ij}; y_i, X_i) = v_j(y_i, X_i) + \epsilon_{ij} = - \int \exp \left( -\psi U^*(\mu_i, \eta_i; y_i, X_i) \right) dF(\mu_i, \eta_i) + \epsilon_{ij},
\]

and the marginal probability for each insurance coverage:

\[
Pr(\text{coverage} = j| \mu_i, \eta_i) = \int \left( \nu_j(y_i, X_i) > \nu_k, \forall k \neq j \right) g(\epsilon_{ij}) d\epsilon_{ij} = \frac{\exp\{v_j(y_i, X_i)\}}{\sum_{k \in J} \exp\{v_k(y_i, X_i)\}}.
\]

**Step 5** Calculate the joint probability for insurance coverage and obesity status:

\[
Pr(\text{coverage} = j \text{ and obesity} = k) = Pr(\text{obesity} = k| \text{coverage} = j) \cdot Pr(\text{coverage} = j).
\]

**Step 6** Calculate the predicted errors:

\[
u_i(\theta|y_i, X_i, Z_i) = \begin{pmatrix}
Pr_i(\text{obesity} = 0, \text{insurance} = 1|\theta) - 1_i(\text{obesity} = 0, \text{insurance} = 1) \\
\vdots \\
Pr_i(\text{obesity} = 1, \text{insurance} = J|\theta) - 1_i(\text{obesity} = 1, \text{insurance} = J)
\end{pmatrix},
\]

and the simulated moments:

\[
G_i(\theta) = \frac{1}{N} \sum_{i=1}^N W_i \otimes u_i(\theta|y_i, X_i, Z_i)
\]

**Step 7** The outer loop searches over the parameter space to minimize the objective function:

\[
Q_N(\theta|y, X, Z, W) = \left[ \frac{1}{N} \sum_{i=1}^N W_i \otimes u_i(\theta|y_i, X_i, Z_i) \right]' \left[ \frac{1}{N} \sum_{i=1}^N W_i \otimes u_i(\theta|y_i, X_i, Z_i) \right].
\]

This step is implemented by the Nelder-Mead algorithm with box constraints (See Kelley, 1999; Hausman and Woutersen, 2013).

After obtaining the estimates, the standard errors are calculated using the standard sandwich formula for the MSM standard errors:

\[
\hat{J} = \frac{1}{N} \sum_{i=1}^N \nabla^2 \theta \left[ W_i \otimes u_i(\theta|y_i, X_i, Z_i) \right]
\]

\[
\hat{\Lambda} = \frac{1}{N} \sum_{i=1}^N \left[ W_i \otimes u_i(\theta|y_i, X_i, Z_i) \right]' \left[ W_i \otimes u_i(\theta|y_i, X_i, Z_i) \right]'
\]
Then the asymptotic variance for the MSM estimates is estimated as:

\[ \text{Avar}(\hat{\theta}) = (\hat{J}' \hat{J})^{-1} (\hat{J}' \hat{\Lambda} \hat{J})(\hat{J}' \hat{J})^{-1} / N \]

The Jacobian, \( \hat{J} \), is calculated by the Richardson’s extrapolation.
Figure 1: Rates of Childhood Overweight (Age 10 - 17)

State-level data are retrieved from the National Survey of Children’s Health 2003 and 2007. The graphs show the rates of being overweight or obese (85th percentile or above).
State-level data are retrieved from the National Survey of Children’s Health 2003 and 2007. The graphs show the rates of being obese (95th percentile or above).

Figure 2: Rates of Childhood Obesity (Age 10 - 17)
The graphs show how the years of childhood eligibility changed over different birth cohorts due to Medicaid/SCHIP expansions. The vertical line represents September 1983. The graphs show that children from families below the federal poverty line born before and after October 1983 have the largest difference in gains in eligibility years among all the recent Medicaid expansions. The graphs imply the importance of using OBRA 1990 as the source of identification. The other states in other Census regions all show a similar pattern.
Figure 4: An Example of BMI-for-Age growth chart

† Source: Centers for Disease Control and Prevention
Each bin represents the density given each birth month weighted by poverty-adjusted person weight. As expected, the histogram of the forcing variable shows no sign of manipulation.
The graphs show the obesity trend by month of birth of children from families below the 100% FPL. Each dot represents the obesity rates given the month of birth. October 1983 is normalized to 0. Using different degrees of polynomials, the fitted lines are plotted using the observations born before October 1983, observations born after October 1983, and all observations. The graphs provide a primitive evidence that the policy cutoff may lead to an increase in obesity rates.
Figure 7: 12-Month-Differenced Obesity Rates

The graph plots the differences in the prevalence of obesity between children who were born around 1983 and those who were born 12 months earlier. The graph shows evidence that the obesity rate of children increased as Medicaid eligibility expanded. We observe no difference for the first 12 months since the differences are taken between ineligibles. 12 months after the Medicaid expansion, the difference disappears since every cohort has the Medicaid eligibility dummy equal to one so that the policy effects are differenced out.
Figure 8: Distributional Effects of Eligibility on BMI Quantiles

The quantile regressions are run against eligibility status, a cubic polynomials of months born before/after the eligibility cutoffs, and dummies for Census region, race, hispanic, month of birth, and survey year, from 15 percentile to 95 percentile. All quantile regressions are weighted by poverty adjusted person weights. The shaded area represents the 90% confidence interval; the standard errors are estimated using a kernel estimator.
Figure 9: Obesity Rates by Birth Months (FPL 100% - 199%)

The graphs show the obesity trend by month of birth of children from families between 100% FPL and 199%FPL. Each dot represents the obesity rates given the month of birth. October 1983 is normalized to 0. Using different degrees of polynomials, the fitted lines are plotted using the observations born before October 1983, observations born after October 1983, and all observations. The graphs suggest that the cohorts who are not affected by Medicaid expansions do not show a significant increase obesity rates.
Figure 10: Box Plot of the Point Estimates

The figure shows the point estimates for obesity and overweight under different parametric assumptions. Each dot is an point estimate using different degrees of polynomials. The upper and lower hinges corresponds to the first and the third quartiles.
Figure 11: Proportion of Obese Children

The figure shows the preliminary evidence that the obesity rate is increasing in household income per capita.
Table 1: Changes in Income and Age Eligibility Limits by Medicaid Expansions and the SCHIP

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† Sources: Table 1 in Levine, McKnight, and Heep (2011)
### Table 2: Characteristics of Children in MEPS 1996-2001

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**Obesity**

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<th>Hispanics</th>
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<td>16.03</td>
<td>28.56</td>
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<td>20.71</td>
<td>17.86</td>
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**Overweight**

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<th>Non-hispanic Blacks</th>
<th>Hispanics</th>
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</thead>
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<td>43.89</td>
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<td>1997</td>
<td>35.51</td>
<td>31.29</td>
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<td>29.98</td>
<td>43.60</td>
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<td>36.79</td>
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<td>2001</td>
<td>37.01</td>
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**Number of Observations**

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The table reports the summary statistics of the sample of all observations between age 3 and age 19 in the MEPS panels from 1996 to 2001. All averages are weighted by poverty-adjusted person weights.

### Table 3: Characteristics of Children in SIPP 1992, 1993, and 1996

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<th>Proportions (%)</th>
<th>1992</th>
<th>1993</th>
<th>1996</th>
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<tr>
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<td>32.73</td>
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<td>Eligible for Medicaid</td>
<td>7.98</td>
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<td>15.39</td>
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<td>Below 100% FPL</td>
<td>21.70</td>
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<td>Medicaid Coverage</td>
<td>14.56</td>
<td>17.61</td>
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<td>Any Insurance Coverage</td>
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</table>

The table reports the summary statistics of the sample of all observations between age 3 and age 19 in the SIPP panels 1992, 1993, and 1996. Means are weighted by the first-year weights.
Table 4: Main Results: Polynomial Regressions

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<td>Est.</td>
<td>S.E.</td>
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<td>0.051</td>
<td>0.059</td>
<td>0.045</td>
</tr>
<tr>
<td>Cubic</td>
<td>0.103 **</td>
<td>0.053</td>
<td>0.093 *</td>
<td>0.053</td>
</tr>
<tr>
<td>Quartic</td>
<td>0.166 ***</td>
<td>0.055</td>
<td>0.171 ***</td>
<td>0.064</td>
</tr>
<tr>
<td>Quintic</td>
<td>0.159 ***</td>
<td>0.050</td>
<td>0.163 **</td>
<td>0.068</td>
</tr>
<tr>
<td>Sextic</td>
<td>0.127 **</td>
<td>0.057</td>
<td>0.138 *</td>
<td>0.078</td>
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<tr>
<td>Controls</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
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</tbody>
</table>

The table reports the polynomial regression estimates using lower-order polynomials of the months born before/after the eligibility cutoff. The estimates are interpreted as the effect of Medicaid eligibility on childhood obesity. The sample includes children born between October 1978 and October 1988. All standard errors are clustered at month of birth. Controls include dummies for Census region, race, hispanic, month of birth, and survey year. All regressions are weighted by poverty-adjusted person weights. The estimates suggest that offering Medicaid to low-income families increases the chances of childhood obesity.

Table 5: Regression-Adjusted Diff-in-Diff Estimates

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<td>0.054 ***</td>
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<td>0.008</td>
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<td>Control</td>
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<td>$\chi^2 = 157$</td>
<td>–</td>
<td>$\chi^2 = 168$</td>
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<tr>
<td>Square Polynomial</td>
<td>0.057 ***</td>
<td>0.054 ***</td>
<td>0.015</td>
<td>0.007</td>
</tr>
<tr>
<td>Controls</td>
<td>–</td>
<td>$\chi^2 = 157$</td>
<td>–</td>
<td>$\chi^2 = 168$</td>
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<tr>
<td>Cubic Polynomial</td>
<td>0.057 ***</td>
<td>0.054 ***</td>
<td>0.015</td>
<td>0.007</td>
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<tr>
<td>Control</td>
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<td>–</td>
<td>$\chi^2 = 167$</td>
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<tr>
<td>Quartic Polynomial</td>
<td>0.058 ***</td>
<td>0.055 ***</td>
<td>0.015</td>
<td>0.007</td>
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<tr>
<td>Control</td>
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The table reports the regression-adjusted difference-in-difference estimates. The difference-in-difference estimator relies on using children from families between 100% and 124% FPL as the control group. The model uses a polynomials of the months born before/after October 1983 to shift the source of identification to the observations around the Medicaid eligibility cutoff. The standard errors are clustered at Census regions. The controls include dummies for Census regions, races, hispanics, month of birth, and survey years. All models are weighted by poverty-adjusted person weights. The estimates are robust against the choice of the functional forms. The results are consistent with the estimates of the sharp regression discontinuity design.
Table 6: Specification Test: Local Linear Regressions

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<td>±6</td>
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The table reports local linear regression estimates. All models use observation born with a certain number of months around the eligibility cutoff. The left panel reports estimates weighted by rectangle kernels, while the right panel reports estimates weighted by triangle kernels. All the models are weighted by poverty-adjusted person weights. Controls include dummies for Census regions, race, and hispanic. The standard errors are clustered at the months of birth.
<table>
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<td>0.085</td>
<td>0.063</td>
<td>0.081</td>
<td>0.146</td>
<td>0.064</td>
<td>0.131</td>
<td></td>
</tr>
<tr>
<td>Sextic</td>
<td>0.074</td>
<td>0.077</td>
<td>0.034</td>
<td>0.080</td>
<td>0.078</td>
<td>0.175</td>
<td>0.057</td>
<td>0.159</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>False Cutoff in October 1982</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Square</td>
<td>-0.091  **</td>
<td>0.037</td>
<td>-0.041</td>
<td>0.040</td>
<td>-0.108 **</td>
<td>0.039</td>
<td>-0.085 **</td>
<td>0.040</td>
<td></td>
</tr>
<tr>
<td>Cubic</td>
<td>-0.048</td>
<td>0.056</td>
<td>-0.029</td>
<td>0.055</td>
<td>-0.144 **</td>
<td>0.051</td>
<td>-0.099 **</td>
<td>0.047</td>
<td></td>
</tr>
<tr>
<td>Quartic</td>
<td>-0.059</td>
<td>0.072</td>
<td>-0.018</td>
<td>0.069</td>
<td>-0.058</td>
<td>0.061</td>
<td>-0.001</td>
<td>0.057</td>
<td></td>
</tr>
<tr>
<td>Quintic</td>
<td>-0.040</td>
<td>0.086</td>
<td>0.032</td>
<td>0.083</td>
<td>-0.072</td>
<td>0.073</td>
<td>0.019</td>
<td>0.068</td>
<td></td>
</tr>
<tr>
<td>Sextic</td>
<td>0.004</td>
<td>0.092</td>
<td>0.053</td>
<td>0.093</td>
<td>-0.063</td>
<td>0.088</td>
<td>-0.018</td>
<td>0.091</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The table reports the estimates using counterfactual eligibility cutoff. All estimates are estimated by polynomial regressions. The top panel uses the counterfactual cutoff in May 1983 while the bottom panel uses the counterfactual cutoff in October 1982. Controls include dummies for Census region, race, hispanic, month of birth, and survey year. All models are weighted by poverty-adjusted person weights.
The table reports the quantile regression estimates of excess BMI. All models are estimated with a flexible function of the months born before/after the policy cutoff, conditional on covariates including dummies for race, Census regions, hispanics, survey years, and birth months. To avoid the influence of few morbidly obese children with large excess BMI, I estimate the effect on 85 percentile, 90 percentile, and 95 percentile. The standard errors are reported in the parentheses and are estimated by bootstrapping.

<table>
<thead>
<tr>
<th>Percentile</th>
<th>Squared</th>
<th>Cubic</th>
<th>Quartic</th>
</tr>
</thead>
<tbody>
<tr>
<td>85-Percentile</td>
<td>0.653 (0.611)</td>
<td>0.176 (0.557)</td>
<td>0.516 (0.824)</td>
</tr>
<tr>
<td>90-Percentile</td>
<td>1.670* (0.898)</td>
<td>1.409 (1.009)</td>
<td>2.491** (1.257)</td>
</tr>
<tr>
<td>95-Percentile</td>
<td>2.352** (1.107)</td>
<td>3.127* (1.737)</td>
<td>2.906 (1.894)</td>
</tr>
</tbody>
</table>
Table 9: Obesity and Overweight Prevalence among U.S. Children 10-17 Years of Age

<table>
<thead>
<tr>
<th>Race/Ethnicity</th>
<th>% in Sample 2007</th>
<th>% in Sample 2003</th>
<th>% Change</th>
<th>Obesity Mean 2007</th>
<th>Obesity Mean 2003</th>
<th>%Change</th>
<th>Overweight Mean 2007</th>
<th>Overweight Mean 2003</th>
<th>%Change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.E.</td>
<td>Mean</td>
<td>S.E.</td>
<td>Mean</td>
<td>S.E.</td>
<td>Mean</td>
<td>S.E.</td>
<td>Mean</td>
</tr>
<tr>
<td>Non-Hispanic Whites</td>
<td>58.82</td>
<td>0.49</td>
<td>12.86</td>
<td>0.29</td>
<td>7.17</td>
<td>0.60</td>
<td>26.84</td>
<td>0.40</td>
<td>0.75</td>
</tr>
<tr>
<td>Non-Hispanic Blacks</td>
<td>15.18</td>
<td>0.28</td>
<td>23.86</td>
<td>1.00</td>
<td>1.75</td>
<td>1.46</td>
<td>41.12</td>
<td>1.17</td>
<td>0.15</td>
</tr>
<tr>
<td>Hispanic</td>
<td>16.76</td>
<td>0.98</td>
<td>23.42</td>
<td>1.86</td>
<td>24.24</td>
<td>2.13</td>
<td>30.81</td>
<td>2.07</td>
<td>8.62</td>
</tr>
</tbody>
</table>

The table is excerpted from Table 1 in Singh et al. (2010). Their data source is the National Survey of Children’s Health 2003 and 2007. NSCH 2003 has 46707 observations, and NSCH 2007 has 44101 observations.

Table 10: Race/Ethnicity Specific Effects

<table>
<thead>
<tr>
<th>Race/Ethnicity</th>
<th>Obesity Square</th>
<th>Obesity Cubic</th>
<th>Obesity Quartic</th>
<th>Obesity Quintic</th>
<th>Overweight Square</th>
<th>Overweight Cubic</th>
<th>Overweight Quartic</th>
<th>Overweight Quintic</th>
<th>Obs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Hispanic White</td>
<td>0.019</td>
<td>0.107*</td>
<td>0.210**</td>
<td>0.266**</td>
<td>-0.036</td>
<td>0.097</td>
<td>0.275**</td>
<td>0.277*</td>
<td>1991</td>
</tr>
<tr>
<td></td>
<td>(0.069)</td>
<td>(0.085)</td>
<td>(0.090)</td>
<td>(0.107)</td>
<td>(0.114)</td>
<td>(0.128)</td>
<td>(0.131)</td>
<td>(0.143)</td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic Black</td>
<td>0.061</td>
<td>0.185**</td>
<td>0.193**</td>
<td>0.227**</td>
<td>-0.079</td>
<td>0.028</td>
<td>0.125</td>
<td>-0.018</td>
<td>1806</td>
</tr>
<tr>
<td></td>
<td>(0.064)</td>
<td>(0.073)</td>
<td>(0.084)</td>
<td>(0.095)</td>
<td>(0.082)</td>
<td>(0.100)</td>
<td>(0.135)</td>
<td>(0.131)</td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>0.017</td>
<td>0.082</td>
<td>0.165**</td>
<td>0.199**</td>
<td>-0.010</td>
<td>-0.061</td>
<td>-0.032</td>
<td>0.049</td>
<td>2648</td>
</tr>
<tr>
<td></td>
<td>(0.050)</td>
<td>(0.060)</td>
<td>(0.076)</td>
<td>(0.083)</td>
<td>(0.063)</td>
<td>(0.083)</td>
<td>(0.104)</td>
<td>(0.114)</td>
<td></td>
</tr>
</tbody>
</table>

The table reports the effect of Medicaid expansions on subsamples. Controls include dummies for Census region, month of birth, and survey year. The standard errors are clustered at each value of the month of birth.
The table reports the estimates of the effect of Medicaid eligibility on male and female subsamples. Since the sample size is small, all models include dummies for Census regions, month of birth, and survey year to minimize small-sample bias. The standard errors are clustered at each value of the month of birth. All models are weighted by poverty-adjusted person weights.

The table reports the quantile regression estimates. The model estimates the distributional effect of the Medicaid expansions on the quantiles of log-BMI. The standard errors are estimated using a kernel estimator. The model is weighted by poverty-adjusted person weights. The estimates suggest that the effect of Medicaid expansions is highly skewed to children with higher BMI.

<table>
<thead>
<tr>
<th></th>
<th>Est.</th>
<th>S.E.</th>
<th>Est.</th>
<th>S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Square</td>
<td>0.100</td>
<td>***</td>
<td>0.036</td>
<td>0.053</td>
</tr>
<tr>
<td>Cubic</td>
<td>0.114</td>
<td>**</td>
<td>0.052</td>
<td>0.097</td>
</tr>
<tr>
<td>Quartic</td>
<td>0.066</td>
<td></td>
<td>0.069</td>
<td>0.103</td>
</tr>
<tr>
<td>Quintic</td>
<td>0.089</td>
<td></td>
<td>0.072</td>
<td>0.152</td>
</tr>
<tr>
<td>Sextic</td>
<td>0.144</td>
<td>**</td>
<td>0.072</td>
<td>0.165</td>
</tr>
<tr>
<td>Septic</td>
<td>0.199</td>
<td>***</td>
<td>0.070</td>
<td>0.157</td>
</tr>
<tr>
<td>Octic</td>
<td>0.196</td>
<td>***</td>
<td>0.074</td>
<td>0.087</td>
</tr>
<tr>
<td>Controls</td>
<td>No</td>
<td></td>
<td>Yes</td>
<td></td>
</tr>
</tbody>
</table>

The table reports estimates of the effect of Medicaid eligibility on insurance coverage using SIPP 1996. The estimates are obtained by sharp regression discontinuity designs using different degrees of polynomials. All models are estimated conditional on age, sex, and state fixed effects. The results show that Medicaid expansions lead to an increase in insurance coverage, which is consistent with most findings in the literature.

Table 14: Two-Sample IV Estimates for Fuzzy RD Design

<table>
<thead>
<tr>
<th>First-Stage</th>
<th>Obesity</th>
<th>Overweight</th>
</tr>
</thead>
<tbody>
<tr>
<td>SIPP 1992</td>
<td>Square</td>
<td>Cubic</td>
</tr>
<tr>
<td></td>
<td>0.010***</td>
<td>0.050***</td>
</tr>
<tr>
<td></td>
<td>(0.001)</td>
<td>(0.002)</td>
</tr>
<tr>
<td>SIPP 1993</td>
<td>0.015***</td>
<td>0.065***</td>
</tr>
<tr>
<td></td>
<td>(0.001)</td>
<td>(0.002)</td>
</tr>
<tr>
<td>SIPP 1996</td>
<td>0.025***</td>
<td>0.047***</td>
</tr>
<tr>
<td></td>
<td>(0.001)</td>
<td>(0.001)</td>
</tr>
</tbody>
</table>

The table reports two-sample IV estimates for the effect of insurance take-up induced by Medicaid eligibility. The main reason to combine two samples is that obesity takes time to develop. The two-sample estimates are interpreted as an intermediate-term effect. For example, the estimates using SIPP 1992 estimate the effect of insurance take-up in 1992 induced by eligibility starting in 1990 on obesity rate in 1996 - 2001. All standard errors are clustered at month of birth. The sample includes individuals born after October 1978 and before October 1988. The estimates are weighted by the sample weights.
Table 15: Proportion of Obese Children by Income per Capita

<table>
<thead>
<tr>
<th>HH Income per Capita</th>
<th>Probability of Obesity</th>
<th>Std. Error</th>
<th>Number of Obs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 1000</td>
<td>0.28</td>
<td>0.03</td>
<td>172.00</td>
</tr>
<tr>
<td>1001 - 1500</td>
<td>0.33</td>
<td>0.02</td>
<td>473.00</td>
</tr>
<tr>
<td>1501 - 2000</td>
<td>0.31</td>
<td>0.02</td>
<td>505.00</td>
</tr>
<tr>
<td>2001 - 2500</td>
<td>0.31</td>
<td>0.02</td>
<td>442.00</td>
</tr>
<tr>
<td>2501 - 3000</td>
<td>0.30</td>
<td>0.02</td>
<td>479.00</td>
</tr>
<tr>
<td>3001 - 3500</td>
<td>0.33</td>
<td>0.02</td>
<td>453.00</td>
</tr>
<tr>
<td>≥ 3501</td>
<td>0.40</td>
<td>0.04</td>
<td>167.00</td>
</tr>
</tbody>
</table>

The data contains the observations that are eligible after OBRA 1990 was in effect. This is the subsample used for the structural estimation. The summary statistics show a source of identification for the structural model, which relies on the assumption that higher food expenditure leads to higher chances of obesity.
Table 16: Method of Simulated Moments Estimation Results (500 draws)

<table>
<thead>
<tr>
<th></th>
<th>Est.</th>
<th>Std. Error</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Structural Parameters</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\gamma_1$ (Risk Aversion)</td>
<td>0.096</td>
<td>0.038</td>
</tr>
<tr>
<td>$\gamma_2$ (Food Expenditure)</td>
<td>8.440</td>
<td>6.317</td>
</tr>
<tr>
<td>$\gamma_3$ (Composite Consumption)</td>
<td>30.429</td>
<td>9.095</td>
</tr>
<tr>
<td>$\psi$ (CARA)</td>
<td>0.132</td>
<td>0.027</td>
</tr>
<tr>
<td><strong>Food Expenditure Shifter</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>-0.170</td>
<td>0.057</td>
</tr>
<tr>
<td>Age</td>
<td>1.698</td>
<td>1.697</td>
</tr>
<tr>
<td>Age$^2$</td>
<td>0.325</td>
<td>0.151</td>
</tr>
<tr>
<td>Hispanic</td>
<td>1.714</td>
<td>0.746</td>
</tr>
<tr>
<td>Female</td>
<td>0.045</td>
<td>0.047</td>
</tr>
<tr>
<td>Unemployed</td>
<td>0.484</td>
<td>0.147</td>
</tr>
<tr>
<td><strong>Medical Expense Shifter</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>-0.434</td>
<td>0.246</td>
</tr>
<tr>
<td>Age</td>
<td>1.748</td>
<td>2.174</td>
</tr>
<tr>
<td>Age$^2$</td>
<td>-0.394</td>
<td>0.072</td>
</tr>
<tr>
<td>Hispanic</td>
<td>-0.193</td>
<td>0.180</td>
</tr>
<tr>
<td>Female</td>
<td>-0.437</td>
<td>0.106</td>
</tr>
<tr>
<td><strong>Obesity Cutoff</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>-1.557</td>
<td>0.553</td>
</tr>
<tr>
<td>Age</td>
<td>6.670</td>
<td>2.459</td>
</tr>
<tr>
<td>Age$^2$</td>
<td>-0.016</td>
<td>0.009</td>
</tr>
<tr>
<td>Hispanic</td>
<td>-1.668</td>
<td>1.316</td>
</tr>
<tr>
<td>Female</td>
<td>-0.806</td>
<td>0.415</td>
</tr>
<tr>
<td>Year=1998</td>
<td>1.418</td>
<td>0.697</td>
</tr>
<tr>
<td>Year=1999</td>
<td>-2.103</td>
<td>1.986</td>
</tr>
<tr>
<td>Year=2000</td>
<td>0.576</td>
<td>0.219</td>
</tr>
<tr>
<td><strong>Variance-Covariance Matrix</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\eta$</td>
<td>0.719</td>
<td>1.375</td>
</tr>
<tr>
<td>$\log \mu$</td>
<td>(0.267)</td>
<td>(1.573)</td>
</tr>
<tr>
<td>$\log \mu$</td>
<td>.</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>.</td>
<td>(—)</td>
</tr>
<tr>
<td><strong>Value of Obj. Fn.</strong></td>
<td>1303</td>
<td></td>
</tr>
</tbody>
</table>

The table reports the structural estimates using the Method of Simulated Moments. Instruments include a constant, age, age squared, hispanics, female, unemployment status, and survey year dummies.
Table 17: Average Partial Effects and Counterfactual Simulations

<table>
<thead>
<tr>
<th></th>
<th>Average Partial Effects</th>
<th>Simulated Elasticities</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\phi_j$ (All Insurance)</td>
<td>-0.401</td>
<td>0.525</td>
</tr>
<tr>
<td></td>
<td>(0.853)</td>
<td>—</td>
</tr>
<tr>
<td>$p_2$ (Public Insurance Premium)</td>
<td>-0.802</td>
<td>0.831</td>
</tr>
<tr>
<td></td>
<td>(1.700)</td>
<td>—</td>
</tr>
<tr>
<td>$y$ (Income)</td>
<td>0.027</td>
<td>0.316</td>
</tr>
<tr>
<td></td>
<td>(0.751)</td>
<td>—</td>
</tr>
</tbody>
</table>

The table reports the estimates of the average partial effects and the simulations using the structural estimates. The simulation results suggest that (1) ex-ante moral hazard (how responsive obesity rates are to the change in the co-insurance rate) is more important in leading to childhood obesity than net-wealth effect (how responsive obesity rates are to the change in income) and (2) households’ behaviors are more responsive to insurance premiums.