Does Health Insurance Make You Fat?

Jay Bhattacharya, M. Kate Bundorf, Noemi Poo, and Neeraj Sood

2.1 Introduction

Adult obesity is a thorny problem. Several studies document rising obesity prevalence in the United States (see Mokdad et al. 1999; Mokdad et al. 2003). Economists have argued that the primary cause of increasing obesity prevalence are: (a) a failing relative price of food; (b) a technologically induced shift away from physically demanding work; and (c) a decline in time spent on food production at home (see Lakdawalla and Philipson 2002; Cutter, Ghinser, and Shapiro 2003; and Anderson, Butcher, and Levine 2003). As most view these fundamental changes in the economy as desirable and would not want to undo them, developing public policy to address the root causes of rising obesity prevalence is difficult, if not entirely problematic.
Nevertheless, the health care and other costs associated with obesity are enormous. For example, Wolf and Colditz (1994) estimate that over $68 billion are lost annually in increased health care costs and job absenteeism as a result of obesity in the United States. The morbidity and accounting costs associated with obesity have led public health experts (such as Needle 2003; Brownell and Horgen 2003; and Sturm 2002) to advocate vigorous public intervention, including regulation of fast-food establishments and taxes on nutritionally questionable foods.

The economic justification for these sorts of policy interventions, such as taxes on food, favored by some of these authors, rests on the idea that when one person becomes obese, many other people pay the cost. In economic jargon, there are negative externalities from body weight decisions that lead to obesity. If external costs are high, then public welfare can be improved by interventions that change the incentives adults face when making decisions about body weight. If external costs are small, then adults pay fully for their body weight decisions and public interventions aimed at decreasing body weight can play only a limited role in improving public welfare.4

5 The main mechanism by which obesity imposes external costs is through pooled health insurance. In a health insurance pool with inadequately risk-adjusted premiums, one person’s increase in body weight really is everyone else’s business, since obesity often leads to higher medical expenditures. In this chapter, we describe a model of this negative obesity externality associated with health insurance.5 The main insight of this model is that measuring the obesity externality involves more than just measuring the subsidy to obese individuals induced by health insurance. The welfare loss due to the obesity externality depends upon both the size of the subsidy and upon the extent to which body weight decisions are distorted on the margin by the subsidy—that is, does coverage with pooled health insurance cause enrollees to gain weight? If the answer is no, and there is no moral hazard of this sort caused by insurance coverage, then the subsidy induced by one person’s obesity would simply represent a transfer from the thinner individuals in his insurance pool to the obese person, with no net effect on social welfare.

Despite the importance of this parameter—the health insurance elasticity of body weight—to the welfare economics of obesity, there has been scant work in the economics literature on the topic.6 The one exception is a paper by Rashad and Markowitz (2010) who find a zero elasticity of insurance coverage on body weight or obesity rates. These authors rely on the size of a firm where an individual works as an instrument for insurance coverage in their body weight regressions. We extend this work along three dimen-

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3. See Blattschuyer and Soold (2007) for a full description of this model.

4. In a related study, Davis and Kasey (2009) analyze the effect of insurance on smoking, drinking, and exercise in the elderly population. They find that obtaining health insurance reduces prevention and increases unhealthy behaviors among elderly men.

5. Some of the studies we reviewed, but arbitrarily do not discuss here, include Bumgarner et al. (2001), Meaith et al. (2004), Quinlithy, Jr., Caan, and Jacobson (1998); Thompson et al. (2001); and Weing et al. (2003).
of insuring these workers. Although theory predicts that employers would have incentives to do so (Rosen 1986), in practice, it is not clear that they would be able to make these adjustments.4 According to Gruber (2000), "... the problems of preference revelation in this context are daunting; it is difficult in reality to see how firms could appropriately set worker specific compensating differentials" (656).

As is the case with Medicare, however, there is little research on obesity-related payment differences in a private insurance setting. An important exception is Bhattacharya and Bundorf (2009), who find some evidence that obese workers receive lower pay than nonobese workers primarily at firms that provide health insurance.

In related work, Keeler et al. (1989) and Manning et al. (1991), using data from the RAND Health Insurance Experiment (RAND HIE) and from the National Health Interview Survey (NHIS), report estimates of lifetime medical costs attributable to physical inactivity (rather than obesity). "At a 5 percent rate of discount, the lifetime subsidy from others to those with a sedentary life style is $1,900" (Keeler et al. 1989). Though they label this estimate the “external cost of physical inactivity,” like the rest of the literature they focus on physical inactivity-related medical expenditure differences, while ignoring payment differences that occur outside experimental settings in their calculation of the subsidy.

2.3 A Model of the Social Costs of Obesity

The timeline in figure 2.1 illustrates the basic setup of the model. Each consumer starts with an initial endowment of weight $W$. This endowment might be seen as reflecting the consumer’s genetic propensity to be overweight or obese, and in any case it cannot be chosen by the consumer. In the first stage, consumers decide how much weight to lose, $s$. Weight loss (exercising, dieting) gives consumers some disutility but has two associated benefits: (a) it increases productivity, consequently raising consumer income and (b) it improves health (more precisely, it decreases the probability of

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6. However, when employers offer multiple health plans, obese workers may tend to select into a different set of plans than their thinner colleagues. In that case, premiums may differ.

7. The literature on medical expenditure-associated obesity costs has a parallel and often interesting literature on the labor market productivity costs associated with obesity (often these latter costs are called “indirect” costs of obesity). The theory of compensating wage differentials has important implications for whether these labor market costs are internal; that is, whether obese individuals pay for lower productivity levels (such as through more sick days) associated with their body weight, or someone else pays. This theory suggests that obese workers will pay for lower productivity through reduced wages. The economics literature on obesity-related wage differences—for example, Reiter and Williams (1998), Pigeon and Dordis (1997), and Casey (2004)—unanimously finds that obese workers earn lower wages than their thinner colleagues, and that these differences are equal to or greater than the wage differences that would arise from measurable productivity differences. Hence, both theory and evidence suggest that these “indirect” costs of obesity are not external.
receiving severe and costly health shocks. Since consumers are insured, they are reimbursed for all of these additional medical care expenditures associated with health shocks. In the second stage, nature reveals a health shock with \( i = 1 \ldots N \) points of support. Each type of health shock entails additional medical expenses, \( M_i \). Essentially, weight gain shifts the health shock distribution so that expected medical care expenditures increase with weight. Consumers first observe this health shock, and then decide how much to consume. The consumers' problem is to maximize expected utility by jointly choosing weight change \((\omega)\) and a consumption plan \((C)\) for each of the \( N \) possible health states:

\[
(1) \quad \max_{\omega, C} EU = \sum_{i=1}^{N} \pi_i \left(W_i - \omega \right) U(C_i) - \Phi(\omega),
\]

where \( U(C_i) \) represents utility from consumption; \( \pi_i(W_i - \omega) \) is the probability of health state \( i \) given weight \((W_i - \omega)\); \( C_i \) is the consumption in health state \( i \); and, \( \Phi(\omega) \) is the disutility from weight loss.

We divide our analysis into two cases: (a) health insurance pools risk across people with heterogeneous risk (so that premiums do not change with body weight); and (b) people pay the risk-adjusted premiums for their own body weight. The primary difference between these cases manifests itself in consumer budget constraints.

2.3.1 Risk Pooling

In this case, health risk is pooled across people of different body weight. As long as the pool size is large enough, a single individual's medical expenditures will have a negligible effect on the common premium, \( \bar{p} \), charged to everyone in the pool. Hence, from the point of view of each individual, premiums are taken as fixed, and the budget constraint is:

\[
(2) \quad (W_i - \omega) = C_i + \bar{P} \forall i.
\]

In equation \( (2) \), \( (W_i - \omega) \) is the income earned by an individual who weighs \( W_i \). By allowing income to depend upon weight, we are modeling the effect of health on labor market productivity. We assume that \( F(\cdot) > 0 \).

The budget constraint specifies that in each health state \( i \), income equals expenditures on consumption and health insurance premiums. An immediate consequence of equation \( (2) \) is that consumption is identical in each health state, which makes sense since consumers are fully insured against medical expenditures.

The consumer's problem is to maximize expected utility, equation \( (1) \), subject to the budget constraint, equation \( (2) \). We solve the consumer's problem using standard discrete numerical programming methods. In the first step, taking the amount of weight as given, we calculate the optimal demand for consumption in each health state. Inputting the optimal consumption plan in the utility function gives the maximum utility attainable in each health state. In the second stage, we choose weight to maximize expected utility given optimal consumption in each health state.

Plugging the budget constraint into equation \( (1) \), we reformulate the consumers' problem in the second stage:

\[
(3) \quad \max_{\omega} EU = U((W_i - \omega) - \bar{P}) - \Phi(\omega),
\]

The first-order condition for the consumer's maximization problem is:

\[
(4) \quad -F(W_i - \omega^\star)U'(F(W_i - \omega^\star) - \bar{P}) - \Phi'(\omega^\star) = 0.
\]

Here, \( \omega^\star \) is the consumer's optimal weight in the pooling case. The first term in equation \( (4) \) is the marginal gain from weight loss, it is entirely due to the marginal increase in income from increased productivity arising from weight loss (scaled by the marginal utility of consumption). In equilibrium, consumers will lose weight until the marginal gain from weight loss equals the marginal disutility from weight loss.

If the insurance market is in competitive equilibrium, then premiums will be actuarially fair. They will equal the expected medical expenses for individuals in the insurance pool:

\[
(5) \quad \bar{F} = \sum_{i=1}^{N} \pi_i (W_i - \omega^\star) M_i.
\]

Equation \( (4) \) also shows that since consumers are fully insured against medical expenses, the only incentive for weight loss is the increase in income due to weight loss. Thus, when insurance premiums do not depend on weight, consumers do not view the reduction in medical expenditures as an additional benefit of weight loss when making decisions about body weight. Insurance induces a form of moral hazard with respect to weight loss incentives since the benefits of weight loss are not fully internalized by
the consumer. As a consequence, weight loss creates a positive externality for everyone else in the insurance pool, since it lowers the health insurance premiums. Because this benefit is not fully captured by the consumer losing the weight, insured people will tend to lose less weight than would be optimal. By contrast, the productivity benefits of weight loss are fully internalized as changes in productivity lead to an increase in consumer income.

### 2.3.2 Risk-Adjusted Insurance

We now turn to the case where health insurance premiums adjust to reflect the weight choice of consumers. In contrast to the previous case, where the premium is taken as fixed, consumers now face a risk-adjusted schedule of health insurance premiums that depends upon their own body weight. In the context of employer provided insurance, this could be achieved by wage reductions for obese employees, or simply by offering premium rebates to individuals who lose weight. In this case, the budget constraint is given by:

\[
R(W_e - w) = C_i + P(W_e - w) \forall i.
\]

Here, \(P(W_e - w)\) is the health insurance premium for an individual who weighs \(W_e - w\). Again, if the insurance market is competitive, premiums will be actuarially fair. Hence, they will be an increasing function of weight, reflecting the increase in expected medical expenses:

\[
P(W_e - w) = \sum_i p_i(W_e - w) M_i.
\]

The consumers’ problem in this case can be reformulated as:

\[
\max_{w} EU = \mathcal{U}(R(W_e - w) - P(W_e - w)) - \Phi(w).
\]

The first-order condition for the consumer’s maximization problem is:

\[
-\mathcal{U}'(W_e - w^{**}) - P'(W_e - w^{**}) U'(R(W_e - w^{**}) - P(W_e - w^{**})) = 0.
\]

Here, \(w^{**}\) is the consumer’s optimal weight in the risk-adjusted case. Clearly, equation (9) is necessary for \(w^{**}\) to be individually optimal, but whether it is also socially optimal depends upon what is meant by social optimality. Suppose \(EU\) is the expected utility of the representative consumer in the economy, and all individuals start with the same initial weight, \(W_e\). In that (unrealistic) case, \(w^{**}\) can be said to be socially optimal, since the full social costs of body weight decisions are internalized. In the appendix, we consider a more realistic case where \(W_e\) differs across individuals in the population. We show that, aside from transfers that do not depend upon final weight, \(W_e - w^{**}\), equation (9) is a necessary condition for the social optimum.

It is instructive to compare the first order condition in equation (9) with the analogous condition in equation (4), when there was a single risk pool. Both equations have a single term reflecting the marginal costs of weight loss: \(\Phi'(\cdot)\). However, equation (9) has two terms, \(P'(\cdot)\) and \(U'(\cdot)\), reflecting the marginal benefit of weight loss accruing from an increase in productivity and a decrease in the health insurance premium. By contrast, equation (4) has only a single term reflecting the marginal productivity benefit of weight loss: \(U'(\cdot)\). Thus, when premiums reflect individual health risk, consumers have two incentives for weight loss—productivity gains and lower health insurance premiums. In this case, there is no moral hazard induced by health insurance and consumer body weight decisions.

### 2.3.3 Deadweight Loss From The Obesity Externality

In this section, we show that the size of the loss in social welfare from the obesity externality under-pooled premiums depends upon both the fact that expected health expenditures are higher for the obese, and also upon how responsive people would be in their weight loss decisions to a switch from pooled to risk-adjusted premiums. This calculation is important because, while there is a lot of empirical evidence that obese people are more likely to have higher medical care expenditures than nonobese people, there is no empirical evidence on whether pooled insurance causes obesity or weight gain. Whether the rise in obesity prevalence is a public health crisis, or merely a private crisis for many people, depends on the evidence on both quantities.

We start with the expression for expected utility, evaluated at the optimum under risk-adjusted insurance:

\[
EU(w^{**}) = \mathcal{U}(R(W_e - w^{**}) - P(W_e - w^{**})) - \Phi(w^{**}).
\]

We have imposed the condition that consumption does not vary with health outcome since consumers are fully insured under both cases.

Next, we consider a first-order Taylor series approximation of equation (10) around \(w^{*}\), which is optimal weight loss under pooled insurance:

\[
EU(w^{*}) = EU(w^{**}) + \frac{\partial EU}{\partial w} (w^{**} - w^{*}) \Delta w.
\]

The deadweight loss (DWL) from the obesity externality is the change in expected utility resulting from pooling. Equation (11) suggests an approximation to this quantity:

\[
DWL = EU(w^{**}) - EU(w^{*}) = \frac{\partial EU}{\partial w} \Delta w.
\]

11. This argument is developed in more detail in the appendix.
Here, \( \Delta u = u^* - u^\circ \) is the difference between optimal weight under risk-adjusted and pooled risk cases. Since weight is socially optimal in the risk-adjusted case, \( \Delta u \) also reflects the degree to which weight choice differs from socially optimal when pooling pertains.

Using a first-order Taylor series approximation, the deadweight loss (DWL) in expected utility terms due to the obesity externality is:

\[
\text{DWL} = U'(W_c - u^*) - U'(W_c - u^\circ) \\
\times \left[ -\left[ \frac{dU'}{du} \right] (W_c - u^*) + \frac{dU'}{du} (W_c - u^\circ) \right] \Delta u.
\]

Substituting the first order condition in equation (4) in equation (13) yields a simple expression for the deadweight loss from the obesity externality:

\[
\text{DWL} = U'(W_c - u^*) \Delta u.
\]

Equation (14) shows that the deadweight loss is proportional to two crucial factors: the extent to which body weight deviates from the optimal due to pooled health insurance when individuals do not bear the full medical care costs of obesity, \( \Delta u \), and the responsiveness of medical care expenditures to changes in weight, \( P(W_c - u^*) \). The deadweight loss from the obesity externality is zero if individual weight choice does not respond to subsidies for obesity, or if medical expenditures do not change with body weight.

While several estimates of \( P(W_c - u^*) \) are available from the public health and economics literatures, there is no work that quantifies \( \Delta u \). To estimate \( \Delta u \), ideally, we would like to know (a) body weight under pooled insurance when the consumer is shielded from the medical care costs of obesity and (b) under risk-adjusted premiums when the individual faces the full medical care costs of obesity. To answer whether obesity creates a negative externality and lost social welfare through the health insurance mechanism, we need to know whether risk-rating insurance premiums affects body weight. Unfortunately, there are no real world data that we are aware of that would permit us to ascertain the effect of risk rating on body weight.

Instead, we aim at answering a related question—whether insurance coverage expansions along both extensive and intensive margins cause body weight to change. It is our conjecture that if insurance coverage does not influence body weight choices, it is unlikely that risk rating would influence body weight choices. Conversely, if health insurance expansion (along either intensive or extensive margin) does influence body weight, it is likely, depending on the mechanism by which risk rating is implemented, that risk rating would influence body weight. We start with an empirical consideration of the intensive margin—expansions in health insurance generosity. Next, we examine the effect of insurance status on the extensive margin, that is, whether the uninsured, who face the full medical care costs of obesity, weigh less than the insured.

2.4 The Intensive Margin: Increasing Generosity of Coverage

Using data from the RAND Health Insurance Experiment (HIIE), we are able to examine the effect of health insurance on body weight when people are randomly assigned to different levels of insurance coverage (the intensive margin). In the HIIE, which was conducted in six areas of the country during the late 1970s and early 1980s, approximately 2,000 nonelderly families were assigned to differing levels of insurance coverage. The purpose of the HIIE was to determine the effects of patient cost sharing on medical care utilization and health. The participants were assigned to different fee-for-service plans that varied along two dimensions: the coinsurance rate (the fraction of billed charges paid by patients), and the maximum dollar expenditure (the maximum amount a family would spend on covered expenditures during a twelve-month period). The coverage was comprehensive in the sense that it included nearly all types of medical care. Participants remained enrolled in their assigned plan and were followed for either three (70 percent) or five years.

The plans were characterized by four different coinsurance percentages—0 (often referred to as "free care"), 25 percent, 50 percent, and 95 percent—and three levels of maximum out-of-pocket spending—5 percent, 10 percent, and 15 percent of family income up to a maximum of $1,000. In one plan, the maximum dollar expenditure (MDE)—also known as maximum out-of-pocket expenditures—was set at $150 per individual, and $450 per family (often referred to as the "individual deductible plan"). In this plan, the coinsurance rate was 95 percent. In our empirical work, we categorized plans based on their coinsurance rate and control for the MDE. We categorized the individual deductible plan separately due to the more complicated structure of the MDE.

In order to minimize participation bias, the investigators offered a participation incentive. The participation incentive for a given family was defined as "the maximum loss risk by changing to the experimental plan from existing coverage," and was intended to ensure that families were equally likely to participate independent of their prior health insurance status and the plan to which they were assigned.

The study collected data on demographic and socioeconomic character-
Table 2.1  Study population characteristics by plan assignment

<table>
<thead>
<tr>
<th></th>
<th>All</th>
<th>Free</th>
<th>25%</th>
<th>30%</th>
<th>95%</th>
<th>Individual</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>2,461</td>
<td>824</td>
<td>492</td>
<td>167</td>
<td>46</td>
<td>556</td>
</tr>
<tr>
<td>BMI at entry</td>
<td>24.79</td>
<td>24.80</td>
<td>24.79</td>
<td>24.89</td>
<td>24.72</td>
<td>24.89</td>
</tr>
<tr>
<td>(4.53)</td>
<td>(4.78)</td>
<td>(4.78)</td>
<td>(4.78)</td>
<td>(4.78)</td>
<td>(4.78)</td>
<td>(4.78)</td>
</tr>
<tr>
<td>BMI at exit</td>
<td>25.51</td>
<td>25.59</td>
<td>25.50</td>
<td>25.45</td>
<td>25.51</td>
<td>25.40</td>
</tr>
<tr>
<td>(4.35)</td>
<td>(4.65)</td>
<td>(4.65)</td>
<td>(4.65)</td>
<td>(4.65)</td>
<td>(4.65)</td>
<td>(4.65)</td>
</tr>
<tr>
<td>BMI change</td>
<td>0.52</td>
<td>0.39</td>
<td>0.33</td>
<td>0.35</td>
<td>0.31</td>
<td>0.31</td>
</tr>
<tr>
<td>(2.45)</td>
<td>(2.16)</td>
<td>(2.16)</td>
<td>(2.16)</td>
<td>(2.16)</td>
<td>(2.16)</td>
<td>(2.16)</td>
</tr>
<tr>
<td>BMI change per year</td>
<td>0.15</td>
<td>0.16</td>
<td>0.10</td>
<td>0.15</td>
<td>0.17</td>
<td>0.15</td>
</tr>
<tr>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
</tr>
<tr>
<td>Obese at entry</td>
<td>0.12</td>
<td>0.12</td>
<td>0.13</td>
<td>0.15</td>
<td>0.11</td>
<td>0.13</td>
</tr>
<tr>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
</tr>
<tr>
<td>Obese at exit</td>
<td>0.14</td>
<td>0.14</td>
<td>0.13</td>
<td>0.16</td>
<td>0.12</td>
<td>0.15</td>
</tr>
<tr>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.24)</td>
</tr>
<tr>
<td>Became obese</td>
<td>0.06</td>
<td>0.04</td>
<td>0.03</td>
<td>0.06</td>
<td>0.00</td>
<td>0.02</td>
</tr>
<tr>
<td>(0.18)</td>
<td>(0.18)</td>
<td>(0.18)</td>
<td>(0.18)</td>
<td>(0.18)</td>
<td>(0.18)</td>
<td>(0.18)</td>
</tr>
</tbody>
</table>

Note: Used chi-square test for categorical variables and t-test of mean relative to free for continuous variables. No differences are statistically significant.

Statistics of enrollees, as well as health status and medical care utilization, both at baseline and during the experiment. This information included enrollee height and weight, both at baseline and at exit, and we use these measures to calculate body mass index (BMI) at each point in time for each enrollee. We limit our analysis to adults (age ≥ 21), and drop observations with missing data for key control variables (age, education, family income, race, gender, marital status, and self-reported health status).

Table 2.1 presents data on body weight by plan type. We find no evidence of statistically significant differences by plan type in body weight, as measured by either BMI or obesity status, either at entry or at exit. In addition, we find no evidence of differences across the plans in changes in these measures. The differences of the differences between plans in changes in BMI, however, are consistent with the hypothesized effect. In other words, enrollees in the free plan experienced the largest change in BMI over the study period (0.59) and the difference in change in BMI between the free plan (0.59) and the 25 percent coinsurance plan (0.33) is statistically significant at p ≤ 0.06. The results are less consistent for the indicator of becoming obese. In this case, the differences across plans based on their level of cost-sharing are not consistent across plans.

In appendix table A.1, we document some differences across the plans in enrollee characteristics despite random assignment. In particular, average family income varies across the plans, and correspondingly, the participation incentive as well. In addition, enrollee assignment to plans is not balanced by site.

In table 2.2, we determine whether the estimates of the effects of plan cost-sharing are influenced by these differences by controlling for them in multivariate models. The multivariate models also allow us to control for the enrollees’ maximum dollar expenditure. We estimate models with two different dependent variables: the BMI change per year (to control for differences across enrollees in their enrollment period), and an indicator of whether an individual became obese during the study period. We estimate three versions of each model. In the first, we control for plan characteristics only (the dummy variable indicating the coinsurance rate and the MDE). In the second, we add the controls for individual characteristics presented in appendix table A.1. In the third, we control for both individual characteristics and aspects of study design including the site and the enrollee’s participation incentive. We estimate the models using least squares. The results from the model of the probability of becoming obese show when we estimate a maximum likelihood logit model.

Table 2.2  The effect of insurance coverage on body weight and obesity

<table>
<thead>
<tr>
<th></th>
<th>BMI change per year</th>
<th>Became obese</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
</tr>
<tr>
<td>25% coinsurance rate</td>
<td>-0.109</td>
<td>-0.053</td>
</tr>
<tr>
<td>(0.48)</td>
<td>(0.48)</td>
<td>(0.48)</td>
</tr>
<tr>
<td>50% coinsurance rate</td>
<td>-0.062</td>
<td>0.025</td>
</tr>
<tr>
<td>(0.64)</td>
<td>(0.64)</td>
<td>(0.64)</td>
</tr>
<tr>
<td>95% coinsurance rate</td>
<td>-0.052</td>
<td>0.06</td>
</tr>
<tr>
<td>(0.48)</td>
<td>(0.48)</td>
<td>(0.48)</td>
</tr>
<tr>
<td>Individual deductible</td>
<td>-0.077</td>
<td>0.024</td>
</tr>
<tr>
<td>(0.72)</td>
<td>(0.72)</td>
<td>(0.72)</td>
</tr>
<tr>
<td>Maximum $ expenditure</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>(0.79)</td>
<td>(0.79)</td>
<td>(0.79)</td>
</tr>
<tr>
<td>Constant</td>
<td>0.161</td>
<td>0.051</td>
</tr>
<tr>
<td>(6.32)**</td>
<td>(6.32)**</td>
<td>(6.32)**</td>
</tr>
<tr>
<td>Includes individual controls: X X X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Includes site effects X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Includes participation incentive X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R-squared</td>
<td>2.441</td>
<td>2.441</td>
</tr>
<tr>
<td>0.01 0.01</td>
<td>2.441</td>
<td>2.441</td>
</tr>
</tbody>
</table>

Note: Absolute value of t statistics in brackets. "*" means that the regression includes either individual controls and controls for site effects and the participation incentive (as the case may be).

**Significant at the 5 percent level.
*Significant at the 10 percent level.
The results from the multivariate models are substantively similar to those from the unadjusted comparisons (table 2.2). While people randomly assigned to plans with cost-sharing experienced a smaller annual change in BMI during the experiment relative to those assigned to the free plan, the effect is statistically significant only in the case of the plan with the 25 percent coinsurance rate. And in this case, the effect is quite small. A 0.175 reduction in BMI represents less than 1 percent of BMI at entry among this group. Correspondingly, we do not find consistent evidence of differences by plan type in the probability of becoming obese during the study period. The direction of the effect varies by plan and none of the estimates are statistically significant.

2.5 The Extensive Margin: Insured vs. Uninsured

While the RAND data allow us to examine the responsiveness of body weight to a change in the generosity of coverage, the fact that everyone in the experiment had health insurance coverage leaves open the possibility of an effect along the extensive margin. In other words, the responsiveness of body weight to any insurance relative to none may be greater than the responsiveness to changes in the generosity of that coverage.

2.5.1 Methods

We use instrumental variables (IV) regressions to estimate the causal effects of private and public insurance coverage on body weight as measured by BMI and obesity status. If our instruments are valid, IV methods purge the estimates of confounding due to observable and unobservable characteristics. We first estimate a linear instrumental variables model estimated via two stage least squares. These models are widely used and a powerful tool in such contexts. However, for nonlinear and limited dependent variable models in general, the linear IV model may either be inappropriate or not work well in practice. Specifically, in our case, although the outcomes of interest are either binary or linear, the endogenous regressors (dummy variables for private and public insurance) are limited dependent variables. A linear IV model would treat the endogenous regressors as if they were linear and unrelated, when, in fact, the insurance choices are mutually exclusive and exhaustive.

To address the discrete nature of our data, we next estimate a nonlinear instrumental variables model using latent factors to account for selection on unobservables. Our model respects the multinomial nature of the endogenous regressors as well as the binary or linear nature of the outcome. Specifically, we assume that the endogenous regressors have a multinomial logit form, while the outcome equations have logit and normal (linear) forms respectively. Then, latent factors are incorporated into the equations to allow for unobserved influences on insurance choice to affect outcomes, and their joint distribution specified (Deb and Trivedi 1997).

The main computational problem is that the joint distribution, which involves a multidimensional integral, does not have a closed form solution. This difficulty can be addressed using simulation-based estimation. Using normally distributed random draws for the latent variables, a simulated likelihood function for the data is defined and its parameters estimated using a Maximum Simulated Likelihood Estimator. Because of the complexity of our model and the large sample size, standard simulation methods are quite slow. Therefore, we adapt an acceleration technique that uses quasi-random draws based on Halton sequences. The formulation, estimation methods, and exposition borrow heavily from Deb and Trivedi (2006).

The model is represented by two sets of equations. In the first set of equations, the insurance choices (private, public, or uninsured) are represented by a multinomial logit model. The second equation, representing the outcome, is modeled as an ordinary least squares (OLS) (BMI as outcome) or logit (obese status as outcome) model. In this model, the choice of insurance and outcome are linked because insurance choices are regressors in the outcome module and because there are common unobservable (latent) factors.

Let Pvt and Pub be binary variables representing private and public insurance coverage. For BMI, we specify the outcome equation as follows:

\[
BMI = x'B + \gamma_iX + \gamma_pPvt + \gamma_Pub + \lambda_iPvt + \lambda_Pub + \epsilon
\]

where x is a set of exogenous covariates and the \(\gamma, \lambda_i, \) and \(\lambda_Pub\) are parameters associated with the exogenous covariates and the endogenous insurance variables. The error term is partitioned into \(\epsilon\), an independently distributed random error, and latent factors \(\lambda_i\) and \(\lambda_Pub\) which denote unobserved characteristics common to an individual’s choice of insurance and the outcome of that individual. The \(\lambda_i\) and \(\lambda_Pub\) are factor loadings or parameters associated with the latent factors that capture the degree of correlation between unobserved determinants of insurance choice and outcomes. If \(\epsilon\) is normally distributed, then

\[
Pr(BMI = BMI) = \phi(x'B + \gamma_pPvt + \gamma_Pub + \lambda_iPvt + \lambda_Pub) = \phi(BMI) = (1 + \exp(x'B + \gamma_pPvt + \gamma_Pub + \lambda_iPvt + \lambda_Pub)^{-1})^{-1}
\]

We estimate a separate version of this model with an indicator of obesity, which we define as BMI greater than 30, as the outcome variable. In this second version of the model, we assume a logit functional form.

\[
Pr(obese = 1 | x, z, Pvt, Pub; \lambda_iPvt + \lambda_Pub) = (1 + \exp(x'B + \gamma_iPvt + \gamma_Pub + \lambda_iPvt + \lambda_Pub)^{-1})^{-1}
\]
Following the multinomial logit framework (McFadden 1980, S15), we formulate the probability of choosing in private insurance, public insurance or remaining uninsured as:

\[
\text{Pr}(\text{Pvt} = 1 | x, f_{\text{Pvt}}, f_{\text{Pub}}) = \frac{\exp(x\alpha_{\text{Pvt}} + \delta_f f_{\text{Pvt}})}{1 + \exp(x\alpha_{\text{Pvt}} + \delta_f f_{\text{Pvt}}) + \exp(x\alpha_{\text{Pub}} + \delta_f f_{\text{Pub}})}
\]

\[
\text{Pr}(\text{Pub} = 1 | x, f_{\text{Pvt}}, f_{\text{Pub}}) = \frac{\exp(x\alpha_{\text{Pub}} + \delta_f f_{\text{Pub}})}{1 + \exp(x\alpha_{\text{Pvt}} + \delta_f f_{\text{Pvt}}) + \exp(x\alpha_{\text{Pub}} + \delta_f f_{\text{Pub}})}
\]

\[
\text{Pr}(\text{Pvt} = 0, \text{Pub} = 0 | x, f_{\text{Pvt}}, f_{\text{Pub}}) = 1 - \text{Pr}(\text{Pvt} = 1) - \text{Pr}(\text{Pub} = 1),
\]

where \( z \) denotes exogenous covariates (instrumental variables) that enter only the insurance choice model, but not the main outcome model. We denote covariates in this site-choice module by \( z \), and covarlates in the outcome equation by \( x \) to highlight the fact that they contain the instrumental variables in the empirical analysis.

Because the latent factors \( f_{\text{Pvt}} \) and \( f_{\text{Pub}} \) enter both choice of insurance equation (17), and outcome (16 and 16') equations, they capture the unobserved factors that induce self-selection into insurance and are also correlated with unobservable factors related to outcomes. Under these assumptions, the joint distribution of selection and outcome variables, conditional on the common latent factors, is simply the product of the functions described in equations (16), (16'), and (17).

The problem in estimation arises because the common latent factors \( f_{\text{Pvt}} \) and \( f_{\text{Pub}} \) are unknown. We assume that these latent factors are distributed bivariate normal with mean zero, variance one, and arbitrary covariance. Given this assumption, the latent factors can be integrated out of the joint density. For example, the joint density of observing outcome obese = 1 and Pmt = 1 is:

\[
\text{Pr}(\text{Obese} = 1, \text{Pmt} = 1 | x, z) = \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \frac{\exp(x\alpha_{\text{Obese}} + \delta_f f_{\text{Obese}})}{1 + \exp(x\alpha_{\text{Obese}} + \delta_f f_{\text{Obese}}) + \exp(x\alpha_{\text{Pmt}} + \delta_f f_{\text{Pmt}})} \times \phi(f_{\text{Pvt}}, f_{\text{Pub}}) df_{\text{Pvt}} df_{\text{Pub}}
\]

where \( \phi(f_{\text{Pvt}}, f_{\text{Pub}}) \) is the bivariate normal partial density function.

In this form, the unknown parameters of the model may be estimated by maximum likelihood estimators (MLE). The main computational problem is that the double integral in equation (18) does not have, in general, a closed form solution. But this difficulty can be addressed using simulation-based estimation (Gourieroux and Monfort 1996) to numerically integrate the equation (18). Because of the complexity of our model, standard simulation methods are quite slow. Therefore, we adopt an acceleration technique that uses quasi-random draws based on Halton sequences (Bhat 2001; Train 2002). We maximize the simulated likelihood using a quasi-Newton algorithm.

2.5.2 Data and Instruments

Data

The primary data source for our analysis is the National Longitudinal Survey of Youth (NLSY). The NLSY includes a nationally representative sample of 12,656 people aged fourteen to twenty-two years in 1979, who were surveyed annually until 1994, and biennially through 2004. Our study uses NLSY data from 1989 to 2004. We exclude the years prior to 1989, as well as 1991, because the survey did not collect information on health insurance status in those years. We further restrict the sample, excluding pregnant women. After these restrictions, 79,876 person-year observations (40,223 male and 39,653 female) were eligible to be included in the study sample.

Instruments

We use the two sets of instruments for insurance choice. The first set of instruments captures the distribution of firm size in every state and year. These data are obtained from the Statistics of U.S. Businesses (SUSB) available online at http://www.sba.gov/advo/research/data.html. We use these data to construct two instruments at the state-year level: (a) percentage of workers employed in firms with 100 to 499 employees, (b) percentage of workers employed in firms with 500 or more employees. These instruments would be valid under two conditions. First, they should be strong predictors of private insurance coverage. Second, they should affect weight choice only through their effect on insurance choice. In the next section, we show that the instruments are strong predictors of private insurance as large firms are more likely to cover employees. The second assumption cannot be directly tested, however, it seems unlikely that changes in firm size distribution within a state (our models have state fixed effects) would be related to weight choices, except through insurance coverage. However, one important caveat is that it is possible that obese workers might prefer to live in states with larger firms to enjoy the benefits of pooled health insurance at these firms. To the extent that this is true, our IV estimates will overestimate the effects of insurance on body weight and obesity.

The second instrument captures generosity of Medicaid coverage. There has been a significant expansion of Medicaid eligibility during this period, and there is significant variation across states in the pace at which these expansions have occurred. Prior research documents a strong association between Medicaid expansions and public insurance coverage. We use data
from several years of the Current Population Survey (CPS) to construct this instrument. First, we regress a binary variable for Medicaid coverage on detailed information on demographics, family income, income, and state \times time fixed effects. The state \times time fixed effects measure the generosity of Medicaid coverage in each state and year after controlling for other important determinants of Medicaid coverage. We posit that these state \times time fixed effects essentially capture differences in Medicaid eligibility rules or enforcement of these rules. We use these fixed effects to create a predicted probability of Medicaid coverage for a standardized population and use these predicted probabilities as an instrument for public insurance coverage. Again, our instrument is valid if variation in our measure of Medicaid eligibility within a state is not correlated with unobserved determinants of obesity within a state. For example, our IV estimates would be biased upwards if deteriorating economic conditions increased obesity rates and also prompted states to expand Medicaid eligibility.

Finally, we also explored using state marginal income tax rates as an instrument for insurance coverage. State marginal income taxes are an attractive candidate for an instrumental variable because employer-sponsored health insurance premiums are exempt from state and federal payroll taxes. Therefore, the subsidy for employer-sponsored insurance is greater in states with higher marginal income taxes. If the demand for insurance slopes downward, then states with higher marginal income tax rates should have a higher proportion of people with employer-provided insurance. Unfortunately, this did not hold true in our sample. We found no significant relationship between state marginal income tax rates and insurance coverage.

**Other Explanatory Variables**

We include several other explanatory variables including race, age, gender, income, Armed Forces Qualifying Test (AFQT) scores and year fixed effects. All these variables are plausibly exogenous and important predictors of weight and insurance choices. In addition, in our preferred specifications we include state fixed effects to control for time invariant differences across states. This is important as our instruments are measured at the state level.

2.5.3 Results

Table 2.3 presents the results from the second stage of the two-stage least squares (2SLS) regressions. Appendix table 2A.2 presents the first stage results. The aim of these regressions is to estimate the causal effect of public and private insurance on BMI and obesity. The first model presents results from the regression model without state fixed effects. The results show that both private and public insurance have a statistically significant effect on BMI. The point estimates for both public and private insurance are positive, but are estimated imprecisely. This is despite the strong predictive power of the instruments in the first stage [F-stat = 139].

The next model includes state fixed effects to capture time invariant
differences across states. The point estimates from this model are implausibly large and very imprecisely estimated. The results indicate that public and private insurance coverage reduce BMI by 3.8 and 7.7 points respectively. However, despite these large point estimates, these estimates are statistically insignificant. These are classical symptoms of the weak instruments problem in a two stage least squares estimate (Staiger and Stock 1997). In this specification the instruments are weak predictors of insurance coverage (F-stat 2.9).

The last models present results from models with obese status as the outcome variable. The results are consistent with the BMI model. Public and private insurance coverage have no statistically significant effect on obesity, and the point estimates from the specification with state fixed effects are implausibly large.

Table 2.4 and appendix table 2A.3 present the results from the MLE models. These models are our preferred specification. The MLE models have several advantages in this context. First, they respect the categorical nature of the endogenous variable. Second, well-specified MLE models are more efficient than 2SLS models. Third, MLE models are less affected by weak instruments in terms of both bias and confidence intervals of the regression coefficient (Staiger and Stock 1997; Lakdawalla, Sood, and Goldman 2006).

The results from the first model show that both public and private insurance have a statistically significantly effect on BMI. The results indicate that private insurance increases BMI by 1.3 points, and public insurance increases BMI by 2.1 points. Both these effects are quite large and are precisely estimated. For example, the effects of private insurance on BMI are similar to moving from the highest AFQT quartile to the lowest AFQT quartiles or moving from less than eight years of education to more than twice years of education. The second model includes state fixed effects. The results are virtually unchanged. The last two models use obesity as an outcome model. The results from these regressions are consistent with the BMI models—both public and private insurance increase obesity.

2.6 Conclusion
Our results indicate that extending insurance coverage to the uninsured will increase body weight. We find that both public and private insurance increase body weight, with somewhat larger effects for public insurance coverage. The effect sizes we measure of the effect of both public and private insurance coverage on body weight are large and precisely estimated. By contrast, we find no evidence that increasing the generosity of insurance coverage for the already insured leads to increases in body weight.

There are several reasons why the extensive margin of insurance matters and the intensive margin is less effective in influencing body weight choices.
First, changes in the intensive margin of insurance likely have a smaller effect on changes in expected out-of-pocket medical expenditures due to obesity. This means that changes in the intensive margin of insurance produce weaker financial incentives to change body weight. Second, changes in the extensive margin of insurance might be more salient to consumers; consequently, such changes might affect behavior more than changes in insurance benefits. Finally, risk-averse consumers might respond to changes in the likelihood of large losses. Changes in the intensive margin of insurance do affect the probability of catastrophic out-of-pocket expenditures, thus they might not influence body weight choices by much. One interpretation of our findings is that large changes in financial incentives (such as those encountered along the extensive margin) affect body weight outcomes, while smaller changes in financial incentives (such as those encountered along the intensive margin) do not.

While our results indicate that insurance increases obesity other authors have come to a different conclusion using a similar approach (Rashad and Markowitz 2010). We demonstrate that the difference is likely due to the method of estimation. When we estimate the model using two-stage least squares, which does not account for the discrete nature of the endogenous indicator of health insurance, our estimates are similar in the sense that we find little evidence that body weight is elastic with respect to insurance coverage. Adopting an alternative maximum likelihood method of estimation, which handles explicitly the discrete endogenous variable and is more robust to weak instruments problem, we reach a different conclusion. Body weight is responsive to health insurance coverage in these models. The estimate is both relatively large in magnitude and precise, and does not vary across the different model specifications.

Our estimates suggest that, by insulating people from the costs of obesity-related medical care expenditures, insurance coverage expansions create moral hazard in behaviors related to body weight. These effects are larger in public insurance programs where premiums are not risk-adjusted, and smaller in private insurance markets where the obese might pay for incremental medical care costs in the form of lower wages (Bhattacharya and Bundorf 2009). By contrast, our estimates also suggest that making insurance more generous has no effect on body weight. Taken together, these findings indicate that providing incentives for healthy behaviors such as risk-rating insurance premiums in private may be effective in reducing body weight in the population (though Bhattacharya and Bundorf 2009 find that employer provided health insurance is already implicitly risk-rated for obesity). Policies that impose costs on increases in body weight among those with public coverage may also reduce body weight, though in that case equity concerns are certain to be important in the policy discussion. The policy challenge will be to design mechanisms that impose costs along the extensive margin, which given our results are likely to be effective, but not along the intensive margin, which are not.

Appendix

A Characterization of the Social Optimum

In this section, we derive necessary conditions characterizing the socially optimal level of weight loss for a society of $f = 1, \ldots, F$ individuals. Each has the following expected utility, taken from equation (1):

$$ EU_f = \sum_{i=1}^{N} \pi_i (W_{yf} - \omega_j) U(C_{j}) - \Phi(\omega) $$

We define total social welfare, $\Omega$, as the sum of expected utilities over all individuals in the society:

$$ \Omega = \sum_{f=1}^{F} EU_f $$

In equation (A.2), $\gamma_f$ represents the Pareto weight that individual $f$ has in the social welfare function. In the social budget constraint, total income equals total expenditures on consumption plus total medical expenditures over all individuals. Both income and the distribution of medical expenditures depend upon body weight decisions:

$$ \sum_{f=1}^{F} [I(W_{yf} - \omega) - \sum_{i=1}^{N} \pi_i (W_{yf} - \omega_j)(M_{j} + C_{j})] = 0 $$

Equation (A.3) builds in our assumption that expectations about the distribution of medical expenditures in the population correspond to the observed distribution of expenditures.

The social problem is to pick consumption and body weight for all individuals in every state of the world—$(C_{j}, \omega_{j})$, $\forall f, j$—to maximize $\Omega$ subject to the social budget constraint. To this end, we construct the following Lagrangian function, where $\lambda$ is the multiplier associated with the social budget constraint (A.3):

$$ L = \sum_{f=1}^{F} \sum_{i=1}^{N} \pi_i (W_{yf} - \omega_j) U(C_{j}) - \gamma_f(\omega) $$

$$ -\lambda \sum_{f=1}^{F} [I(W_{yf} - \omega) - \sum_{i=1}^{N} \pi_i (W_{yf} - \omega_j)(M_{j} + C_{j})] $$

There are two sets of first order conditions:
(A.5) \[
\frac{\partial L}{\partial C_j} = \gamma U(C_j) + \lambda = 0 \ \forall i, j, \text{ and}
\]

(A.6) \[
\frac{\partial L}{\partial \omega q} = -\sum_{i=1}^{N} \pi_i (W_q - \omega_q) \gamma U(C_q) - \gamma \Phi(\omega_q)
\]

\[
+ \lambda \left( f(W_q - \omega_q) + \sum_{i=1}^{N} \pi_i (W_q - \omega_q) \lambda M_i + C_q \right) = 0 \ \forall j.
\]

An immediate implication of equation (A.5) is that at the social optimum, each individual’s utility in the society must set his (or her) consumption level to the same value, say \(C^*_j\), across all the \(N\) different health states:

(A.7) \[C^*_j = C^*_q \ \forall i, j.
\]

Applying equation (A.7) to equation (A.6) yields the following:

(A.8) \[-(\gamma U(C_q) + \lambda C_q) \sum_{i=1}^{N} \pi_i (W_q - \omega_q) - \gamma \Phi(\omega_q)
\]

\[
+ \lambda \left( f(W_q - \omega_q) + \sum_{i=1}^{N} \pi_i (W_q - \omega_q) \lambda M_i + C_q \right) = 0 \ \forall j.
\]

By definition, \(\sum_{i=1}^{N} \pi_i (W_q - \omega_q) = 1\), so we have \(\sum_{i=1}^{N} \pi_i (W_q - \omega_q) = 0\). Furthermore, differentiating equation (7), which defines the risk-adjusted premium, \(P(W_q - \omega_q)\), yields the fact that:

(A.9) \[P(W_q - \omega_q) = -\sum_{i=1}^{N} \pi_i (W_q - \omega_q) M_i \ \forall j.
\]

These equations and equation (A.5) permit a further simplification of equation (A.6):

(A.10) \[-\Phi(\omega_q) - U(C_q) f(W_q - \omega_q) - P(W_q - \omega_q) = 0 \ \forall j.
\]

Hence, the social optimum requires each individual to equate the marginal (utility) costs of weight loss with the marginal (utility) benefits from the weight loss—an increase in income and a reduction in expected medical costs.

One feasible allocation that meets equation (A.10) would set consumption equal to income, less the risk-adjusted premium given weight:

(A.11) \[C^*_j = f(W_q - \omega_q) - P(W_q - \omega_q) \ \forall j.
\]

It is easy to show that this allocation would be optimal for some distribution of initial body weight, \(\{W_q\}\), and some set of Pareto weights, \(\{\gamma_q\}\). In this allocation, there are no transfers between individuals with different initial body weights. Other optimal and feasible allocations are possible, but these would involve fixed transfers between individuals that do not

<table>
<thead>
<tr>
<th>Table 2A.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>R&amp;D needs of belong and health status can be used as an index of health status.</td>
</tr>
<tr>
<td>Individual</td>
</tr>
<tr>
<td>25%</td>
</tr>
<tr>
<td>All</td>
</tr>
<tr>
<td>Race—White</td>
</tr>
<tr>
<td>Race—Black</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Education (years of schooling completed)</td>
</tr>
<tr>
<td>Full participation above 75%</td>
</tr>
<tr>
<td>Full participation above 75%</td>
</tr>
<tr>
<td>Self-reported health status—bedridden</td>
</tr>
<tr>
<td>Self-reported health status—ill</td>
</tr>
<tr>
<td>Self-reported health status—severely</td>
</tr>
<tr>
<td>Self-reported health status—disabled</td>
</tr>
<tr>
<td>Self-reported health status—good</td>
</tr>
<tr>
<td>Self-reported health status—excellent</td>
</tr>
<tr>
<td>Note: Data are means of data on health status for self-reported health status.</td>
</tr>
</tbody>
</table>

This table shows the relationship between health status and R&D needs, controlling for race, sex, education, and self-reported health status.
Table 2A.2  First stage results for 2SLS models

<table>
<thead>
<tr>
<th></th>
<th>Public-no state FE</th>
<th>Public-state FE</th>
<th>Private-no state FE</th>
<th>Private-state FE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coefficient</td>
<td>Std. error</td>
<td>Coefficient</td>
<td>Std. error</td>
<td>Coefficient</td>
</tr>
<tr>
<td>% in firms 0 to 499</td>
<td>0.4866</td>
<td>0.0981</td>
<td>0.2126</td>
<td>0.4822</td>
</tr>
<tr>
<td>% in firms 500+</td>
<td>-0.0248</td>
<td>0.0315</td>
<td>0.1217</td>
<td>0.1322</td>
</tr>
<tr>
<td>Pr. Medicaid enrollment</td>
<td>0.3074</td>
<td>0.0125</td>
<td>0.0959</td>
<td>0.0323</td>
</tr>
<tr>
<td>Year—1990</td>
<td>0.0030</td>
<td>0.0038</td>
<td>0.0034</td>
<td>0.0035</td>
</tr>
<tr>
<td>Year—1992</td>
<td>-0.0177</td>
<td>0.0044</td>
<td>-0.0037</td>
<td>0.0046</td>
</tr>
<tr>
<td>Year—1993</td>
<td>0.0240</td>
<td>0.0045</td>
<td>0.0345</td>
<td>0.0048</td>
</tr>
<tr>
<td>Year—1994</td>
<td>0.0190</td>
<td>0.0048</td>
<td>0.0343</td>
<td>0.0054</td>
</tr>
<tr>
<td>Year—1996</td>
<td>-0.0027</td>
<td>0.0055</td>
<td>0.0168</td>
<td>0.0063</td>
</tr>
<tr>
<td>Year—1998</td>
<td>-0.0045</td>
<td>0.0057</td>
<td>0.0081</td>
<td>0.0066</td>
</tr>
<tr>
<td>Year—2000</td>
<td>-0.0137</td>
<td>0.0061</td>
<td>-0.0019</td>
<td>0.0073</td>
</tr>
<tr>
<td>Year—2002</td>
<td>-0.0144</td>
<td>0.0067</td>
<td>-0.0004</td>
<td>0.0079</td>
</tr>
<tr>
<td>Year—2004</td>
<td>-0.0124</td>
<td>0.0075</td>
<td>0.0003</td>
<td>0.0051</td>
</tr>
<tr>
<td>Age</td>
<td>0.0024</td>
<td>0.0028</td>
<td>0.0026</td>
<td>0.0028</td>
</tr>
<tr>
<td>Age squared</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td>Nonwhite</td>
<td>0.0034</td>
<td>0.0021</td>
<td>0.0452</td>
<td>0.0022</td>
</tr>
<tr>
<td>Male</td>
<td>-0.0081</td>
<td>0.0018</td>
<td>-0.0519</td>
<td>0.0018</td>
</tr>
<tr>
<td>Education 6 to 8 yrs.</td>
<td>0.0444</td>
<td>0.0058</td>
<td>0.0486</td>
<td>0.0058</td>
</tr>
<tr>
<td>Education 8 to 12 yrs.</td>
<td>0.0134</td>
<td>0.0021</td>
<td>0.0330</td>
<td>0.0021</td>
</tr>
<tr>
<td>Income (1000s)</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td>AFQT quintile 1</td>
<td>0.0999</td>
<td>0.0044</td>
<td>0.0105</td>
<td>0.0032</td>
</tr>
<tr>
<td>AFQT quintile 2</td>
<td>0.0166</td>
<td>0.0028</td>
<td>0.0166</td>
<td>0.0028</td>
</tr>
<tr>
<td>AFQT quintile 3</td>
<td>0.0039</td>
<td>0.0026</td>
<td>0.0040</td>
<td>0.0026</td>
</tr>
<tr>
<td>Constant</td>
<td>-0.2119</td>
<td>0.0050</td>
<td>-0.3087</td>
<td>0.0054</td>
</tr>
</tbody>
</table>

Number of observations: 70,168

F-statistic: 139.14 2.91 120.51 4.52

Table 2A.3  First stage results for SML models

<table>
<thead>
<tr>
<th></th>
<th>Public-no state FE</th>
<th>Public-state FE</th>
<th>Private-no state FE</th>
<th>Private-state FE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coefficient</td>
<td>Std. error</td>
<td>Coefficient</td>
<td>Std. error</td>
<td>Coefficient</td>
</tr>
<tr>
<td>% in firms 0 to 499</td>
<td>38.856</td>
<td>2.4072</td>
<td>3.3308</td>
<td>7.6902</td>
</tr>
<tr>
<td>% in firms 500+</td>
<td>4.796</td>
<td>0.7933</td>
<td>1.6022</td>
<td>2.7916</td>
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<tr>
<td>Pr. Medicaid enrollment</td>
<td>4.482</td>
<td>0.3433</td>
<td>2.6147</td>
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<tr>
<td>Year—1990</td>
<td>0.3162</td>
<td>0.1116</td>
<td>0.3262</td>
<td>0.1127</td>
</tr>
<tr>
<td>Year—1992</td>
<td>-0.0120</td>
<td>0.1262</td>
<td>0.1355</td>
<td>0.1351</td>
</tr>
<tr>
<td>Year—1993</td>
<td>0.4193</td>
<td>0.1100</td>
<td>0.6246</td>
<td>0.1271</td>
</tr>
<tr>
<td>Year—1994</td>
<td>0.3385</td>
<td>0.1344</td>
<td>0.6093</td>
<td>0.1416</td>
</tr>
<tr>
<td>Year—1996</td>
<td>-0.0088</td>
<td>0.1413</td>
<td>0.3621</td>
<td>0.1672</td>
</tr>
<tr>
<td>Year—1998</td>
<td>0.0833</td>
<td>0.1901</td>
<td>0.3477</td>
<td>0.1760</td>
</tr>
<tr>
<td>Year—2000</td>
<td>-0.0818</td>
<td>0.1633</td>
<td>0.2923</td>
<td>0.2035</td>
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<tr>
<td>Year—2002</td>
<td>-0.2355</td>
<td>0.1747</td>
<td>0.1370</td>
<td>0.2090</td>
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<tr>
<td>Year—2004</td>
<td>-0.0777</td>
<td>0.1092</td>
<td>0.2090</td>
<td>0.2387</td>
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<td>0.0206</td>
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<tr>
<td>Age squared</td>
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<td>0.0030</td>
<td>0.0153</td>
<td>0.0045</td>
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<td>0.0948</td>
<td>0.0418</td>
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<tr>
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<td>0.0518</td>
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<tr>
<td>Education 6 to 8 yrs.</td>
<td>-0.0444</td>
<td>0.1125</td>
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<tr>
<td>Education 8 to 12 yrs.</td>
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<td>0.0614</td>
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<td>0.0630</td>
</tr>
<tr>
<td>Income (1000s)</td>
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<td>-0.0001</td>
<td>0.0000</td>
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<td>AFQT quintile 1</td>
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<td>0.1183</td>
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<tr>
<td>AFQT quintile 2</td>
<td>0.0320</td>
<td>0.1115</td>
<td>0.8727</td>
<td>0.1136</td>
</tr>
<tr>
<td>AFQT quintile 3</td>
<td>0.0493</td>
<td>0.1125</td>
<td>0.0319</td>
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<tr>
<td>Constant</td>
<td>-11.4186</td>
<td>1.4551</td>
<td>-4.5813</td>
<td>2.5977</td>
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</tbody>
</table>

Number of observations: 70,168
depend upon final body weight (though they might depend upon initial body weight). Optimal transfers would clearly vary with (τ), though all optimal allocations would need to obey condition (A.10).

References


Food Prices and the Dynamics of Body Weight

Dana Goldman, Darius Lakdawalla, and Yuhui Zheng

3.1 Introduction

A great many policy approaches to the obesity epidemic have been proposed. A popular choice among these has been the imposition of a "fat tax" on selected foods that are deemed to promote obesity, as a result of high caloric density, low nutritional value, or high fat content (Jacobson and Brownell 2006; Nestle and Jacobson 2000). In the year 2000, for example, there were nineteen states and cities in the United States that imposed taxes on less nutritious foods like soft drinks, sweets, or snack foods (Jacobson and Brownell 2006). In the past, policymakers viewed these primarily as "sin taxes" designed to raise revenue rather than influence health. Most localities use revenues for general purposes. Others earmark them for specific purposes, like violence prevention (Washington), Medicaid (Arkansas), or medical schools (West Virginia). Such taxes were strongly opposed by the soft drink and food industries. Perhaps as a result, twelve localities have reduced or repealed such taxes in recent years.

Understanding the public economics of fat taxes requires an understand-