Journal of Health Economics 31 (2012) 219–230

Contents lists available at SciVerse ScienceDirect

Journal of Health Economics

journal homepage: www.elsevier.com/locate/econbase

# The medical care costs of obesity: An instrumental variables approach☆

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ARTICLE INFO

*Article history:*
Received 7 October 2010
Received in revised form
29 September 2011
Accepted 11 October 2011
Available online 20 October 2011

*JEL classification:*
I1
I14
I18
D6

*Keywords:*
Obesity
Externalities
Medical care costs
Insurance

ABSTRACT

This paper is the first to use the method of instrumental variables (IV) to estimate the impact of obesity on medical costs in order to address the endogeneity of weight and to reduce the bias from reporting error in weight. Models are estimated using restricted-use data from the Medical Expenditure Panel Survey for 2000–2005. The IV model, which exploits genetic variation in weight as a natural experiment, yields estimates of the impact of obesity on medical costs that are considerably higher than the estimates reported in the previous literature. For example, obesity is associated with $656 higher annual medical care costs, but the IV results indicate that obesity raises annual medical costs by $2741 (in 2005 dollars). These results imply that the previous literature has underestimated the medical costs of obesity, resulting in underestimates of the economic rationale for government intervention to reduce obesity-related externalities.



## 1. Introduction

In the United States, the prevalence of obesity, defined as a body mass index[1] or BMI > 30, has been rising for at least five decades (e.g. Burkhauser et al., 2009; Komlos and Brabec, 2010) and has more than doubled in the past thirty years (Flegal et al., 1998). In 2007–2008, 33.8% of American adults were clinically obese (Flegal et al., 2010). This is troubling because obesity is associated with an increased risk of myocardial infarction, stroke, type 2 diabetes, cancer, hypertension, osteoarthritis, asthma, and depression, among other conditions (Dixon, 2010; Hu, 2008).

Many previous papers have estimated the association of obesity with medical care costs (e.g. Finkelstein et al., 2009; Trasande et al., 2009; Thorpe et al., 2004; Finkelstein et al., 2003; Kortt et al., 1998). Typically, this involves estimating cross-sectional models using large secondary datasets such as the National Medical Expenditure Survey of 1987 (NMES) and the more recent Medical Expenditure Panel Survey (MEPS). These studies have made an important contribution to the literature by demonstrating the significance of medical costs associated with obesity and the diseases linked to obesity. As a result, these papers have been heavily cited and widely influential.[2] For example, these estimates have been used to justify government programs to prevent obesity on the grounds of external costs (e.g. U.S. D.H.H.S., 2010).

However, the previous estimates have important limitations. The most significant is that they measure the correlation of obesity with, not the causal effect of obesity on, medical care costs. The correlation is an overestimate of the causal effect if, for example, some people became obese after suffering an injury or chronic depression, and have higher medical costs because of the injury or

☆ We thank Tania Andreyeva, Virginia Chang, Eric Finkelstein, Alan Monheit, Leo Trasande, Jessica P. Vistnes, and two anonymous referees their helpful comments. We are particularly indebted to Joe Newhouse, who provided many thoughtful and detailed suggestions.

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[1] Body mass index is defined as weight in kilograms divided by height in meters squared.

[2] For example, Finkelstein et al. (2003) has been cited 235 times, as of September 9, 2011, according to the ISI Web of Knowledge.

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doi:10.1016/j.jhealeco.2011.10.003

depression (which is likely to be unobserved by the econometrician). Conversely, the correlation is an underestimate of the causal effect if, for example, those with less access to care, such as disadvantaged minorities and the poor, are more likely to be obese (Fontaine and Bartlett, 2000). Another limitation is that these studies are usually based on self-reported, rather than measured, height and weight, and this reporting error biases the coefficient estimates (Bound et al., 2002).

This paper builds on the previous research by addressing both of these problems – endogeneity of weight and reporting error in weight – by estimating models of instrumental variables. Our instrument for the respondent's weight is the weight of a biological relative, an instrument used in the previous literature to estimate the impact of weight on other outcomes such as wages (e.g. Cawley, 2004; Kline and Tobias, 2008) and mortality (Smith et al., 2009). We estimate the IV model using the 2000–2005 MEPS, the leading source of data on medical care costs and utilization for the U.S. non-institutionalized population. Our results indicate that the effect of obesity on medical care costs is much greater than previously appreciated. The model also passes several falsification tests: it finds a stronger impact of obesity on medical expenditures for diabetes (clearly linked to obesity) than on medical expenditures for other conditions, does not find an impact of obesity on medical care costs for conditions that are unrelated to obesity, and biologically unrelated children (e.g. stepchildren) are not significant predictors of respondent weight.

The limitations of cost of illness studies are widely recognized (Shiell et al., 1987; Roux and Donaldson, 2004). For example, they are not useful for prioritizing the allocation of medical resources because that would amount to a circular argument: some conditions have a large amount of resources devoted to them and thus have a high cost of illness, but that does not imply that even more funding is needed (see, e.g., Shiell et al., 1987). This paper does not estimate the medical care costs of obesity in order to argue that treatment of obesity should be prioritized above treatment of other conditions, but to more accurately measure the marginal effect of obesity on medical care costs.

## 2. Empirical model

### 2.1. Identification: method of instrumental variables

Ideally, to measure the effect of obesity on medical care costs one would conduct a randomized controlled trial in which obesity was assigned by the investigator. Such an experiment would, of course, be unethical, so one must rely on natural experiments. We follow the previous literature (e.g. Cawley, 2004; Kline and Tobias, 2008; Smith et al., 2009) and use the weight of a biological relative as an instrument for the weight of the respondent.

There are two requirements for an instrument. First, it must be powerful. The weight of a biological relative is a powerful predictor of the weight of a respondent because roughly half the variation in weight across people is genetic in origin (Comuzzie and Allison, 1998). As we describe in Section 4, our instrument set easily exceeds the conventional benchmark for power of $F = 10$ in the first stage (Stock et al., 2002). The second requirement is validity – the instrument must be uncorrelated with the error term in the second stage. In the present context, this means that the weight of a biological relative must be uncorrelated with the respondent's residual medical care costs after controlling for predicted respondent weight and other observed characteristics.

Validity would be threatened if both the respondent and the biological relative are affected by a common household environment that is also directly correlated with the respondent's medical expenditures. Although it is impossible to prove the null hypothesis of no effect, and therefore some doubt will always remain, much research in behavioral genetics finds no detectable effect of a shared household environment effect on weight. Adoption studies have consistently found that the correlation in weight between a child and its biological parents is the same for children raised by their biological parents and children raised by adoptive parents (Vogler et al., 1995; Stunkard et al., 1986; Sorensen and Stunkard, 1993). Other studies have found that the weights of unrelated adopted siblings are uncorrelated (Grilo and Pogue-Geile, 1991). Twin studies (which by necessity are based on small samples) find no significant difference between the correlation in the weight of twins reared together and twins reared apart (Price and Gottesman, 1991; Maes et al., 1997), which is consistent with a negligible common household environment effect on weight.

With hundreds of behavioral genetics studies on the subject, there are of course some studies that detect a shared family environment on BMI (e.g. Nelson et al., 2006), but the preponderance of evidence is that any such effects are so small as to be undetectable and ignorable (Hewitt, 1997; Grilo and Pogue-Geile, 1991; Maes et al., 1997). For example, a recent study using the same data as Nelson et al. (2006) concluded: "We also did not find any support for shared environmental effects on BMI at any age." (Haberstick et al., 2010, p. 501).

This may be contrary to conventional wisdom but it is a robust finding; a comprehensive review concluded that "[E]xperiences that are shared among family members appear largely irrelevant in determining individual differences in weight and obesity" (Grilo and Pogue-Geile, 1991), and more recently Wardle et al. (2008) conclude: "Contrary to widespread assumptions about the influence of the family environment, living in the same home in childhood appears to confer little similarity in adult BMI beyond that expected from the degree of genetic resemblance." (Wardle et al., 2008, p. 398)

One must always be cautious with regard to the validity of instruments, but given the consistent finding that similarity in weight between biological relatives can be attributed to genetics, we believe that there is enough suggestive evidence regarding power and validity to proceed with the use of weight of a biological relative as an instrument for respondent weight. As a check of validity, we later conduct a falsification test that uses the weight of a stepchild (when available) instead of a biological child and find that the weight of a stepchild is not a significant predictor of respondent weight, which is consistent with our identifying assumption.

In the previous literature on the medical care costs of obesity, coefficients are likely biased because of measurement error in BMI that is due to using self-reported, rather than measured, weight and height.[3] (Only self-reports or proxy-reports of weight and height are available in the MEPS.) Numerous studies have documented systematic misreporting of height and weight (e.g. Plankey et al., 1997; Villanueva, 2001). For example, Cawley and Burkhauser (2006) examine data from the National Health and Nutrition Examination Survey III, which contains data on both self-reported and measured weight and height. Using self-reported, rather than measured, data to calculate BMI results in considerable underestimation of the prevalence of obesity; e.g. among white females, the prevalence of obesity is 21.6% based on measurements

[3] The direction of the bias due to reporting error in weight is ambiguous, because the reporting error in weight is not classical – errors are not independent of the true value of the variable; in particular, those who are heavier tend to underreport their weight more. See Burkhauser and Cawley (2008) for more on reporting error in weight, and see Bound et al. (2002) for details on the bias resulting from reporting error and the use of IV methods to reduce bias from reporting error.

but 17.4% based on self-reports (Cawley and Burkhauser, 2006). Another source of reporting error is that much of the information contained in the MEPS is reported by a single household member, or proxy, and it is possible that proxies may not provide accurate height and weight information for others in the household. (On the other hand, it is also possible that proxies report other people's weight more accurately than respondents report their own weight.) Both of these sources of reporting error are expected to bias the coefficient estimates in the previous literature. A benefit of the IV method is that it corrects for these multiple sources of measurement error (see, e.g. Bound et al., 2002).

### 2.2. Two-part model of medical expenditures

To estimate the impact of BMI and obesity on medical spending we use a two-part model (2PM) of medical expenditures (Jones, 2000). The first part of the 2PM estimates the probability of positive medical expenditures, while the second part estimates the amount of medical expenditures conditional on having any. We specify the first part as a Logit model and the second part as a Gamma GLM with log link.[4] Following the suggestion of Manning and Mullahy (2001), we used modified Park tests to determine the proper choice of the conditional variance function for the GLM, and Hosmer–Lemeshow tests to confirm that our choice of link function is consistent with the data generating process.[5]

Given our specification of the 2PM, both parts of the model require the use of nonlinear instrumental variables techniques. Because both the Logit model and the Gamma model are among the class of GLMs, one can use the instrumental variable estimator of Carroll et al. (1995) and Hardin et al. (2003) to determine the effect of weight on medical expenditures when the endogenous and mismeasured regressor is either BMI or a discrete indicator for obesity.[6] In both cases, our primary set of instruments for the two-part IV models is the BMI, BMI squared, and BMI cubed of the respondent's oldest biological child.[7]

Prior research suggests that the relationship between body weight and health status is nonlinear. In particular, mortality risk is somewhat U-shaped over BMI, with the underweight (BMI < 18.5) and obese (BMI ≥ 30) facing higher mortality risk than the healthy weight (18.5 ≤ BMI < 25) and overweight (25 ≤ BMI < 30) (Flegal et al., 2005; Seidell et al., 1996). To accommodate nonlinearities in the relationship between medical expenditures and weight status we estimate a second set of two-part IV models in which the endogenous regressors are the respondent's BMI and BMI squared. We also estimated exactly identified IV models that include as endogenous regressors the respondent's BMI, BMI squared, and BMI cubed. Both specifications yield a similar pattern of expenditures over BMI, but we report results from the model that includes BMI and BMI squared but not BMI cubed because the confidence intervals around predicted medical expenditures are much narrower.

The impact of obesity on medical expenditures may vary across the distribution of medical spending. To explore this possibility we estimate the conditional quantile treatment effect (QTE) of obesity at different points in the medical expenditure distribution using Frolich and Melly's (2008) implementation of the IV estimator of Abadie et al. (2002). In this case the instrument must be discrete, so we use the obesity status of the oldest biological child.[8]

All of our IV models control for the following regressors: gender, race/ethnicity (white, black, Hispanic, other race), respondent age (indicator variables for whether age in years is 20–34, 35–44, 45–54, or 55–64), education level (no high school diploma, high school graduate, some college, bachelor's degree or higher), census region (northeast, midwest, south, or west), whether the respondent lives in an MSA, household composition (number of household members age 0–5 years, 6–17, 18–64, and 65 or older), whether the survey information was self-reported as opposed to proxy-reported, whether the individual was employed, fixed effects for year, the gender of the oldest child, and the age of the oldest child

[4] Identifying the appropriate functional form for the second part of 2PM requires analysis of various characteristics of the expenditure distribution. An additional consideration in this case is that we seek to provide estimates for the overall population of non-elderly adults as well as seven sub-populations (men, women, white, non-white, private insurance, Medicaid, uninsured), so our estimator must perform well across sixteen different combinations of sample and empirical specification. The two most widely used estimators for the second part of the 2PM are: (1) OLS of the log of the dependent variable; and (2) the GLM estimator. A significant drawback of the log OLS approach is that re-transformation of the estimates back to the raw scale requires knowledge of the degree and form of heteroscedasticity. In our application this would entail the difficult task of accurately diagnosing and correcting for heteroscedasticity in each sub-sample, making the GLM approach attractive in comparison. However, GLMs can be inefficient if the log-scale disturbances are heavy-tailed (Manning and Mullahy, 2001), so we examined the kurtosis of the log-scale residuals from an OLS model of medical expenditures and found it has an average value of 3.2 in our data. While this is slightly larger than the normal distribution, a properly specified GLM model should be reasonably efficient under this degree of skewness.

[5] The Park tests indicated that the conditional variance is proportional to the square of the conditional mean ($\lambda$ ranges from 1.91–2.06 and is precisely estimated), which is consistent with a gamma-class model. To perform the Hosmer–Lemeshow tests we regressed the prediction errors from each model on deciles of the distribution of predicted expenditures. If the *F*-test of coefficients on the decile indicators is jointly significant it indicates that the model does not fit the data well over the distribution of predicted expenditures. We rejected the null hypothesis that the decile coefficients are jointly equal to zero for only three out of sixteen models, which suggests that the gamma model with log link is broadly appropriate. In addition, Hill and Miller (2009) found that this specification performed relatively well on the 1996–2003 sample of non-elderly MEPS respondents with private insurance.

[6] This approach incorporates a linear first stage, which is most appropriate when the endogenous and mismeasured regressor is continuous. While it is not uncommon to estimate IV models with a linear first stage when the endogenous regressor is discrete, the resulting coefficient estimate may be biased. If the regressor suffers *only* from nonclassical measurement error then the true effect will generally lie between the OLS and IV estimates in the case of a simple univariate regression (Black et al., 2000). When the regressor of interest is both endogenous and mismeasured, Frazis and Loewenstein (2003) demonstrate that the true effect lies within bounds applied to the IV estimate. An alternative approach is to specify the exact distribution of both the binary endogenous regressor and the outcome variable. Even if the distributional assumptions are not correct and the treatment effect estimate is biased, this estimator may still be preferred from a mean square error standpoint. Deb's (2007) treatment effects gamma model provides an estimator of this type that is appropriate for modeling skewed outcomes, such as medical expenditures. The estimation approach makes use of simulated maximum likelihood techniques to predict the impact of the treatment variable (obesity), which is assumed to follow a normal distribution, on an outcome variable (medical expenditures) generated from a gamma distribution (Deb and Trivedi, 2006a,b). To test the sensitivity of our results to an alternative estimator that explicitly accounts for the discrete nature of the endogenous and mismeasured regressor, we re-estimated all of our models using the treatment effects gamma model. While the marginal effects of obesity we derived using this approach were very similar to those derived from the approach of Carroll et al. (1995) on all samples except the uninsured, we prefer the method of Carroll et al. (1995) because it produced more consistent medical expenditure predictions across the full range of the BMI distribution.

[7] We obtained similar results using three other instrument sets: (1) BMI, BMI squared, and BMI cubed of the youngest child; (2) BMI and BMI squared of the youngest child and the BMI of the second youngest child; (3) BMI and BMI squared of the oldest child and the BMI of the second oldest child. All instrument sets have the same theoretical justification, but we prefer the BMI, BMI squared, and BMI cubed of the oldest child because there is a higher response rate to the height and weight questions for older children.

[8] Obesity is defined as a BMI at or above the 95th percentile for children of the same age and gender according to the 2000 CDC growth charts (U.S. D.H.H.S., 2002).

in months.[9] For subgroup analyses the set of regressors is modified to drop irrelevant control variables.

## 3. Data: medical expenditure panel survey (MEPS)

The medical expenditure panel survey (MEPS) is a comprehensive, nationally representative survey of the U.S. civilian non-institutionalized population that has been conducted annually since 1996 and uses an overlapping panel design. Respondents are surveyed about their medical care use and expenditures over the course of two years through five interview rounds. In addition, information from the household is supplemented by expenditure data collected directly from participants' medical service providers and pharmacies through a Medical Provider Component. We use data from the 2000–2005 waves of the MEPS, and convert medical expenditures in each year to 2005 dollars.

We limit the sample to adults between the ages of 20 and 64 with biological children between the ages of 11 years (132 months) and 20 years (240 months), and exclude pregnant women. We use the restricted-use MEPS data, which include relationship mappings for sample members in the same household, to identify biological children, stepchildren, and foster children and thus ensure that only biological children are used as instruments. We do not use information on children younger than 11 years because rates of non-response for their height and weight begin to exceed 14% and worsen for younger children. The weight and height of each individual in the household are typically reported by a single respondent, most often the wife/mother.[10] We excluded eighteen individuals with implausibly high BMIs (greater than 80), as well as two individuals with extremely high reported medical expenditures in excess of $292,000, bringing our final estimation samples to 9852 men and 13,837 women.

We use various measures of medical spending in our empirical models: total medical expenditures, expenditures by all third party payers (typically, public and private insurers), and also expenditures by all payers on specific categories of care: inpatient, outpatient, prescription drugs, and other (which includes dental, vision, home health care services, and medical equipment but excludes spending on over-the-counter medications). Medical expenditures by source of payment are collected directly from households as well as from the household's medical care providers for every medical event. In addition, MEPS respondents are asked whether their medical visits or other events are related to any specific medical conditions. These responses are then professionally coded using the *International Classification of Diseases*, Ninth Revision (ICD-9), and subsequently collapsed to into 259 clinically relevant medical conditions using the Clinical Classification System (CCS) developed by the Agency for Healthcare Research and Quality (AHRQ, 2007).

MEPS data are collected through a stratified multi-stage probability design, which we account for in the calculation of the standard errors for our marginal effects. In particular, we use the method of balanced repeated replications to estimate standard errors in our 2PM and the method of bootstrapping with 500 replications in the IV QTE models. Both methods account for clustering at the PSU-level, stratification, and weighting.

**Table 1**
Descriptive statistics for sample of men with biological children ($N = 9852$).

| | Mean | S.D. | Min | Max |
|---|---|---|---|---|
| Medical expenditures > 0 | 0.79 | 0.41 | 0 | 1 |
| Total medical expenditures | 1999 | 5406 | 0 | 212,681 |
| Third party med. expenditures > 0 | 0.72 | 0.45 | 0 | 1 |
| Third party medical expenditures | 1577 | 4970 | 0 | 197,501 |
| Body mass index | 28.17 | 4.88 | 14.98 | 59.30 |
| Body mass index squared | 817 | 308 | 224 | 3516 |
| Obesity (BMI ≥ 30) | 0.28 | 0.45 | 0 | 1 |
| White | 0.72 | 0.45 | 0 | 1 |
| Hispanic | 0.14 | 0.35 | 0 | 1 |
| Black | 0.09 | 0.28 | 0 | 1 |
| Other race | 0.05 | 0.23 | 0 | 1 |
| Age is 20–34 | 0.06 | 0.24 | 0 | 1 |
| Age is 35–44 | 0.43 | 0.49 | 0 | 1 |
| Age is 45–54 | 0.43 | 0.49 | 0 | 1 |
| Age is 55–64 | 0.09 | 0.28 | 0 | 1 |
| No. high school diploma | 0.16 | 0.37 | 0 | 1 |
| High school graduate | 0.34 | 0.47 | 0 | 1 |
| Some college | 0.21 | 0.41 | 0 | 1 |
| Bachelor's degree or higher | 0.29 | 0.45 | 0 | 1 |
| Northeastern census region | 0.19 | 0.39 | 0 | 1 |
| Midwestern census region | 0.24 | 0.43 | 0 | 1 |
| Southern census region | 0.34 | 0.47 | 0 | 1 |
| Western census region | 0.23 | 0.42 | 0 | 1 |
| Residence in MSA | 0.81 | 0.40 | 0 | 1 |
| No. HH members 0–5 | 0.18 | 0.48 | 0 | 5 |
| No. HH members 6–17 | 1.74 | 1.02 | 0 | 9 |
| No. HH members 18–64 | 2.30 | 0.73 | 1 | 9 |
| No. HH members 65+ | 0.02 | 0.17 | 0 | 3 |
| Survey info. is self-reported | 0.25 | 0.43 | 0 | 1 |
| Not employed | 0.09 | 0.28 | 0 | 1 |
| Oldest child is female | 0.47 | 0.50 | 0 | 1 |
| Oldest child age in months | 191.44 | 31.20 | 132.00 | 240.00 |
| Oldest child BMI | 22.49 | 5.10 | 7.60 | 109.19 |
| Oldest child BMI squared | 532 | 330 | 58 | 11,923 |
| Oldest child BMI cubed | 13,504 | 26,148 | 439 | 1,301,949 |
| Oldest child is obese | 0.12 | 0.33 | 0 | 1 |
| Year is 2000 | 0.16 | 0.36 | 0 | 1 |
| Year is 2001 | 0.17 | 0.37 | 0 | 1 |
| Year is 2002 | 0.17 | 0.38 | 0 | 1 |
| Year is 2003 | 0.17 | 0.37 | 0 | 1 |
| Year is 2004 | 0.16 | 0.37 | 0 | 1 |
| Year is 2005 | 0.17 | 0.37 | 0 | 1 |

*Notes*: Data: MEPS 2000–2005. All means are weighted and monetary values expressed in 2005 USD.

## 4. Results

### 4.1. Summary statistics

Descriptive statistics for the main set of variables used in our empirical analysis are contained in Table 1 for men and Table 2 for women. (The samples are limited to adults with biological children, as they are the only MEPS respondents for whom we can estimate the IV model.) Among men, 79% incur some medical expenditures in the survey year, and the unconditional average medical expenditures in that year was $1999 (which includes zeros for those with no expenditures) in 2005 dollars. Among women, 88% incurred some medical expenditures, and the unconditional average medical expenditures in that year was $2617. These expenditures are lower than those for the comparable population of all men and women (i.e. those both with and without biological children) (AHRQ, 2010). For men (women), approximately 72% (80%) of expenditures are covered by third party payers.

In our sample, the average BMI (calculated using self-reported or proxy-reported weight and height) is 28.17 for men and 27.37 for

[9] As a robustness check, we also estimated models that include controls for health insurance status (private or public insurance), employer size (indicators of whether the firm contained less than 25, 25–100, 101–500, or over 500 employees), whether the individual belonged to a union, whether the individual was married, and net income per adult equivalent (total household income minus health insurance premiums divided by the square root of household size). Including these additional regressors has little effect on the estimated impact of BMI and obesity on medical expenditures.

[10] The exception to this is when all adult members of the household are present during the interview, in which case each adult self-reports their height and weight.

**Table 2**
Descriptive statistics for sample of women with biological children ($N$ = 13,837).

| | Mean | S.D. | Min | Max |
|---|---|---|---|---|
| Medical expenditures > 0 | 0.88 | 0.32 | 0 | 1 |
| Total medical expenditures | 2617 | 6565 | 0 | 220,924 |
| Third party med. expenditures > 0 | 0.80 | 0.40 | 0 | 1 |
| Third party medical expenditures | 2050 | 6199 | 0 | 218,323 |
| Body mass index | 27.37 | 6.42 | 14.50 | 78.3 |
| Body mass index squared | 790 | 410 | 210 | 6131 |
| Obesity (BMI ≥ 30) | 0.28 | 0.45 | 0 | 1 |
| White | 0.66 | 0.47 | 0 | 1 |
| Hispanic | 0.15 | 0.35 | 0 | 1 |
| Black | 0.14 | 0.34 | 0 | 1 |
| Other race | 0.06 | 0.23 | 0 | 1 |
| Age is 20–34 | 0.11 | 0.31 | 0 | 1 |
| Age is 35–44 | 0.52 | 0.50 | 0 | 1 |
| Age is 45–54 | 0.34 | 0.47 | 0 | 1 |
| Age is 55–64 | 0.03 | 0.18 | 0 | 1 |
| No. high school diploma | 0.16 | 0.37 | 0 | 1 |
| High school graduate | 0.34 | 0.47 | 0 | 1 |
| Some college | 0.25 | 0.43 | 0 | 1 |
| Bachelor's degree or higher | 0.24 | 0.43 | 0 | 1 |
| Northeastern census region | 0.19 | 0.39 | 0 | 1 |
| Midwestern census region | 0.24 | 0.43 | 0 | 1 |
| Southern census region | 0.35 | 0.48 | 0 | 1 |
| Western census region | 0.22 | 0.42 | 0 | 1 |
| Residence in MSA | 0.81 | 0.39 | 0 | 1 |
| No. HH members 0–5 | 0.16 | 0.44 | 0 | 5 |
| No. HH members 6–17 | 1.72 | 1.00 | 0 | 9 |
| No. HH members 18–64 | 2.12 | 0.80 | 1 | 9 |
| No. HH members 65+ | 0.03 | 0.20 | 0 | 3 |
| Survey info. is self-reported | 0.83 | 0.37 | 0 | 1 |
| Employed | 0.25 | 0.43 | 0 | 1 |
| Oldest child is female | 0.49 | 0.50 | 0 | 1 |
| Oldest child age in months | 191.38 | 31.09 | 132.00 | 240.00 |
| Oldest child BMI | 22.66 | 5.22 | 6.70 | 109.19 |
| Oldest child BMI squared | 541 | 321 | 45 | 11,923 |
| Oldest child BMI cubed | 13,820 | 23,206 | 2803 | 1,301,949 |
| Oldest child is obese | 0.13 | 0.34 | 0 | 1 |
| Year is 2000 | 0.15 | 0.36 | 0 | 1 |
| Year is 2001 | 0.16 | 0.37 | 0 | 1 |
| Year is 2002 | 0.18 | 0.38 | 0 | 1 |
| Year is 2003 | 0.17 | 0.38 | 0 | 1 |
| Year is 2004 | 0.17 | 0.38 | 0 | 1 |
| Year is 2005 | 0.17 | 0.37 | 0 | 1 |

*Notes*: Data: MEPS 2000–2005. All means are weighted and monetary values expressed in 2005 USD.

women. For both the men and women in our sample, the prevalence of obesity is 28%; this is significantly lower than the prevalence of obesity based on measured weight and height in the NHANES 2007–08, which is 32.2% among men and 35.5% among women aged 20 and older (Flegal et al., 2010). The difference in the prevalence of obesity in the MEPS and the NHANES is certainly due in part to the self-reporting of weight in the MEPS, but may also be due to differences in the ages of the samples and the fact that our MEPS sample is restricted to adults with biological children in the household, who may be less likely to be obese than other adults. Tables 1 and 2 indicate that 83% of women and 25% of men self-report their weight; for the remainder, weight is proxy reported by the primary respondent for the household.

### 4.2. Power and validity of instruments

We now present empirical evidence regarding the power and validity of the instruments. Because we must test the power of our instrument set in both parts of the 2PM across eight populations for two measures of weight status (BMI and obesity), we calculated 32 first stage $F$-statistics. The values of the $F$ statistics range from 31 to 281, with an average of 144. In each case, the power of the instruments easily exceeds the conventional minimum standard of power of $F$ = 10 (Stock et al., 2002). In addition, Hansen's (1982) test for over-identification is consistent with the validity of our instruments. In order to compute the Hansen $J$-statistic we estimate linear IV models using GMM for both the first and second parts of the 2PM when the endogenous regressor is BMI and when it is an indicator for obesity. Because we use three instruments (BMI, BMI squared, and BMI cubed of the adult's oldest biological child) the $J$-statistic follows a chi-square distribution with 2 degrees of freedom.[11] We fail to reject the null hypothesis that the instruments are valid in each of the 32 tests.

### 4.3. Weight, obesity, and medical expenditures

Table 3 lists regression results for the entire sample (row 1) and various subpopulations: men, women, white, nonwhite, those with private insurance, those with Medicaid, and the uninsured. Each cell of the table lists the marginal effect (reflecting both parts of the two-part model) and the standard errors of the marginal effect. The first two columns of Table 3 contain the results of the non-IV two-part Gamma GLM models in which the regressor of interest is BMI (column 1) or obesity (column 2). Column 1 indicates that weighing an additional unit of BMI is associated with $49 higher annual expenditures for the pooled sample, $59 higher annual medical expenditures for men and $47 higher annual medical expenditures for women. Column 2 indicates that obesity (relative to having a BMI less than 30) is associated with $656 higher medical expenditures for the pooled sample, $564 higher medical expenditures for men and $749 higher medical expenditures for women.

The middle two columns of Table 3 provide results from our IV 2PM. The point estimates of the marginal effects on BMI and the indicator for obesity are considerably higher for IV than non-IV. The standard errors are also much larger; as a result, the IV marginal effects on BMI and the indicator variable for obesity are not statistically significant for men or Medicaid recipients, despite having point estimates that are much larger than the non-IV estimates that are statistically significant.

Column 3 of Table 3 indicates that weighing an additional unit of BMI raises medical expenditures by $149 in the pooled sample, $80 for men (which is not statistically significant), and $173 for women. Column 4 indicates that obesity (relative to being non-obese) raises medical expenditures by $2741 for the pooled sample, $1152 for men (which is not statistically significant), and $3613 for women. A comparison of the results of the IV and non-IV models indicates that the IV estimate of the effect of obesity on medical expenditures is more than four times higher than the non-IV estimate, for both the pooled sample and for women. In other words, relying on correlations (as in the previous literature) results in considerable underestimation of the impact of obesity on medical costs.

Results for specific subgroups indicate that the impact of obesity on medical costs is higher for the uninsured ($3153) than for those with private insurance ($2568), but the difference is not statistically significant (see Table 3, column 4). The difference in point estimates could be due to the uninsured receiving expensive acute care treatment for obesity-related conditions (e.g. asthma) that the insured avoid through preventive care and prescription drugs, or

[11] Letting $r$ denote the total number of included and excluded instruments in the model, and $q$ denote the number of included instruments (the right-hand-side exogenous variables), the optimal GMM estimator for the parameter vector sets equal to zero $q$ linear combinations of the $r$ sample orthogonality conditions. As a result, there are $r$–q sample orthogonality conditions that are close to zero, but are not set equal to zero. Hansen (1982) shows that the test statistic based on the convergence of these orthogonality conditions to zero in probability has an asymptotic chi-square distribution with $r$–$q$ degrees of freedom.

**Table 3**
Marginal effects of BMI and obesity on annual medical expenditures (standard errors in parenthesis).

| Population | Non-IV (total expenditures) | | IV (total expenditures) | | IV (third party expenditures) | |
|---|---|---|---|---|---|---|
| | BMI | Obesity | BMI | Obesity | BMI | Obesity |
| Total ($N$ = 23,689) | 49 (9) | 656 (113) | 149 (35) | 2741 (745) | 129 (30) | 2418 (649) |
| Men ($N$ = 9852) | 59 (11) | 564 (128) | 80$^{ns}$ (52) | 1152$^{ns}$ (831) | 69$^{ns}$ (46) | 967$^{ns}$ (736) |
| Women ($N$ = 13,837) | 47 (11) | 749 (150) | 173 (34) | 3613 (837) | 151 (30) | 3220 (728) |
| White ($N$ = 12,575) | 63 (13) | 817 (178) | 150 (47) | 2739 (997) | 135 (39) | 2502 (849) |
| Non-white ($N$ = 11,114) | 31 (10) | 400 (134) | 151 (42) | 2731 (798) | 124 (39) | 2330 (776) |
| Private insurance ($N$ = 16,475) | 55 (12) | 666 (138) | 140 (39) | 2568 (813) | 127 (34) | 2346 (715) |
| Medicaid ($N$ = 2835) | 50 (21) | 857 (441) | 186$^{ns}$ (119) | 3674$^{ns}$ (2708) | 178$^{ns}$ (122) | 3521$^{ns}$ (2761) |
| Uninsured ($N$ = 4379) | 21 (8) | 267 (123) | 166 (90) | 3153 (1449) | – | – |

*Notes*: Data: MEPS 2000–2005. Standard errors are adjusted for the complex design of the MEPS and values are reported in 2005 USD. ns denotes that the estimate is not statistically significant at $\alpha$ = .10.

it may result from the uninsured being charged higher prices for medical care (Anderson, 2007).

Despite the fact that the correlation between obesity and medical expenditures is twice as large for whites ($817) as non-whites ($400), the causal effect of obesity is nearly the same for the two groups ($2739 for whites versus $2731 for non-whites). One possible explanation for this pattern is that minorities who lack access to care have lower medical expenditures and a greater risk of developing obesity; this reverse causality would be reflected in the correlation but not the IV estimate of the causal effect of obesity on medical expenditures.

The final two columns of Table 3 list results for a model of third-party medical expenditures, which represent a possible pathway for obesity-related externalities. Specifically, some of the medical costs of obesity may be borne by other enrollees in private insurance pools or by taxpayers in the form of higher expenditures by the Medicaid program. The results in the final column imply that obesity may be associated with substantial externalities: obesity raises annual third-party medical expenditures by $2418 in the pooled sample, which is 88% of the effect of obesity on total medical costs. (We are not able to calculate the exact amount of the externality because we lack data on the respondents' contributions to these third-party payers in the form of insurance premia and taxes.) The impact of obesity on third-party medical expenditures is significantly higher for women ($3220) than men ($967, which is not statistically significant). Despite the large point estimate of the impact of obesity on third-party medical expenditures for the Medicaid population ($3521), it is not statistically significant.

We also explore the impact of BMI and obesity on specific categories of medical expenditures. Table 4 lists the association of BMI and obesity with, and the effect of BMI and obesity on, spending on inpatient care, outpatient services, prescription drugs, and other care (vision, dental, home health, and medical equipment). The marginal effects from the IV regressions indicate that an additional unit of BMI raises medical spending by $54 for inpatient care, $49 for outpatient services, $48 for prescription drugs, and has a negligible impact on spending on other medical care. The IV estimates indicate that obesity raises medical spending by $1116 for inpatient care, $860 for outpatient services, $919 for prescription drugs, with again no significant change in other medical spending. These causal effects are a multiple of the associations implied by non-IV models (except for the "other" category). Reassuringly, the sum of effects across all categories of expenditures in Table 4 is very similar to the effect for total medical expenditures in the first row of Table 3.

Table 5 contains the predicted per capita medical expenditures of the non-obese (column 1) and obese (column 4), with the marginal effect of obesity (the difference between the medical expenditures of the non-obese and obese) estimated from the IV 2PM. For most sub-populations, the increase in medical expenditures resulting from obesity is larger than the total predicted


**Fig. 1.** Predicted relationship between BMI and annual medical expenditures for all adults with biological children. *Notes*: Data: MEPS 2000–2005. Expenditures are in 2005 USD. Dashed lines represent the 90% confidence interval, which has been adjusted for the complex design of the MEPS. Medical expenditures are denoted by the solid line, while the distribution of individuals in the population is indicated by the dotted line. BMI is calculated using self-reports or proxy reports of weight and height.

expenditures for the non-obese; that is, in most subgroups obesity causes at least a doubling of medical expenditures (the exception is for men, for whom obesity raises medical expenditures by 75%). For the pooled sample, obesity raises predicted medical expenditures by roughly 150%, from $1763 to $4458. For women, obesity raises predicted medical expenditures by roughly 180%, from $1928 to $5363. The largest relative increase in expenditure occurs among the uninsured, for whom obesity raises medical expenditures by 540%, from $512 to $3271.

While the marginal effects reported in Tables 3 and 4 are informative, they predict the impact of changes in body weight near the mean (i.e. mean BMI or mean weight of all those who are obese), which can be misleading if the relationship between weight and medical expenditures is nonlinear. We illustrate the nonlinear relationship between predicted medical expenditures over the range of BMI (calculated using self-reports or proxy reports of weight and height, and thus contain reporting error) for all adults (Fig. 1), men (Fig. 2), and women (Fig. 3), allowing for nonlinearity through the inclusion of BMI squared (in addition to BMI) in our IV 2PM.[12] Medical expenditures are denoted by the solid line and are measured on

[12] We also estimated models that included BMI, BMI squared, and BMI cubed; these models yield similar patterns of medical care costs over BMI, but the standard errors were considerably higher, so we prefer the model that includes BMI and BMI squared but omits BMI cubed.

**Table 4**
Marginal effects of BMI and obesity on total annual medical expenditures by type of medical care (standard errors in parenthesis). Men and women pooled; $N = 23{,}689$.

| Medical care | Non-IV (total expenditures) | | IV (total expenditures) | |
|---|---|---|---|---|
| | BMI | Obesity | BMI | Obesity |
| Inpatient | 11 (4) | 118 (67) | 54 (21) | 1116 (520) |
| Outpatient | 19 (4) | 264 (58) | 49 (19) | 860 (396) |
| Prescription drugs | 22 (2) | 263 (23) | 48 (8) | 919 (177) |
| Other | $0.1^{ns}$ (1.3) | $14^{ns}$ (14) | $-0.9^{ns}$ (3.2) | $-15^{ns}$ (51) |

*Notes*: Data: MEPS 2000–2005. Standard errors are adjusted for the complex design of the MEPS and values are reported in 2005 USD. Other includes: vision, dental, home health, and medical equipment but not over-the-counter medications.

**Table 5**
Medical expenditures on obesity as a percentage of expenditures by the non-obese (standard errors in parenthesis).

| Population | Predicted expenditures for non-obese | Marginal effect of obesity | Average ratio of col. 2 to col. 1 | Predicted expenditures for obese |
|---|---|---|---|---|
| Total ($N = 23{,}689$) | 1763 (104) | 2741 (745) | 1.6 (0.5) | 4458 (618) |
| Men ($N = 9852$) | 1657 (170) | $1152^{ns}$ (831) | $0.7^{ns}$ (0.6) | 2907 (668) |
| Women ($N = 13{,}837$) | 1928 (97) | 3613 (837) | 1.9 (0.5) | 5363 (712) |
| White ($N = 12{,}575$) | 2026 (132) | 2739 (997) | 1.4 (0.6) | 4786 (863) |
| Non-white ($N = 11{,}114$) | 1144 (124) | 2731 (798) | 2.4 (0.9) | 3900 (636) |
| Private insurance ($N = 16{,}475$) | 1920 (114) | 2568 (813) | 1.3 (0.5) | 4393 (675) |
| Medicaid ($N = 2835$) | 2494 (628) | $3674^{ns}$ (2708) | $1.5^{ns}$ (1.5) | 6455 (2085) |
| Uninsured ($N = 4379$) | 512 (70) | 3153 (1449) | 6.2 (3.4) | 3271 (1153) |

*Notes*: Data: MEPS 2000–2005. Standard errors are adjusted for the complex design of the MEPS and values are reported in 2005 USD. ns denotes that the estimate is not statistically significant at $\alpha = .10$. Marginal effect of obesity is estimated using the IV 2PM, and is reported in the fifth column of Table 3.

the left axis, while the distribution of individuals in the population is indicated by the dotted line and is measured on the right axis.

Figs. 1–3 each reveal interesting nonlinearities. For men and women pooled (Fig. 1), expenditures have a J-shape over BMI; i.e. expenditures fall with BMI through the underweight and healthy weight categories, are relatively constant with BMI in the overweight category, then rise sharply with BMI through the obese category. The BMI value associated with minimum expenditures is roughly 25, the threshold for overweight.

This J-shaped pattern for the pooled sample masks considerable differences between men and women. In Fig. 2, for men, expenditures are U-shaped over BMI. Expenditures drop sharply with BMI through the healthy weight category, fall modestly with BMI in the overweight category, then rise slowly at first then sharply with BMI in the obese category. The BMI value associated with minimum expenditures for men is roughly 30, but expenditures are quite similar for men with BMI in the range of 26–35.

Fig. 3 shows a quite different pattern for women. For a wide range of BMI, roughly 15–25, expected expenditures are relatively constant. As BMI rises beyond 25, however, expected expenditures rise through the overweight and obese categories, increasing rapidly at the high end of the obese range.

In each Figure, the dotted line indicates the distribution of BMI. This, in conjunction with the solid line that shows expected expenditures by BMI unit, confirms that it is a very small percentage of the sample at high levels of BMI that incur disproportionate shares of obesity-related medical costs. Thus, the obese are a heterogeneous group; the large average effects of obesity shown in Table 3 ($1152 for men and $3613 for women) are explained by relatively

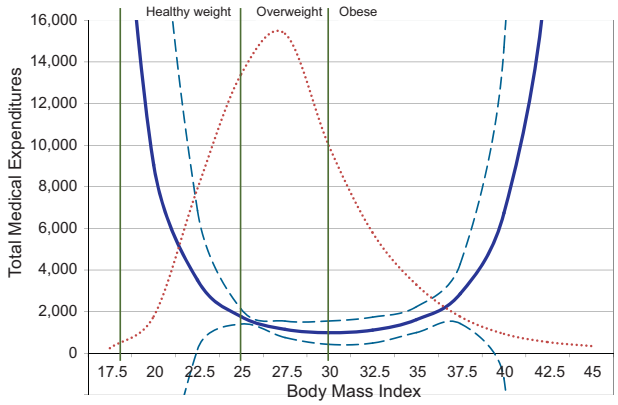


**Fig. 2.** Predicted relationship between BMI and annual medical expenditures for men with biological children. *Notes*: Data: MEPS 2000–2005. Expenditures are in 2005 USD. Dashed lines represent the 90% confidence interval, which has been adjusted for the complex design of the MEPS. Medical expenditures are denoted by the solid line, while the distribution of individuals in the population is indicated by the dotted line. BMI is calculated using self-reports or proxy reports of weight and height.

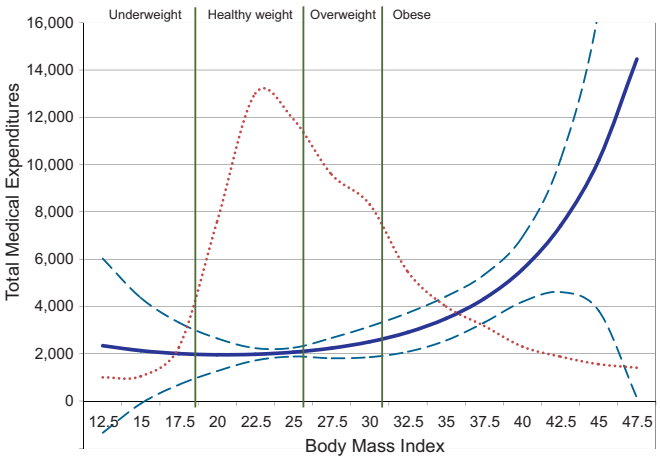


**Fig. 3.** Predicted relationship between BMI and annual medical expenditures for women with biological children. *Notes*: Data: MEPS 2000–2005. Expenditures are in 2005 USD. Dashed lines represent the 90% confidence interval, which has been adjusted for the complex design of the MEPS. Medical expenditures are denoted by the solid line, while the distribution of individuals in the population is indicated by the dotted line. BMI is calculated using self-reports or proxy reports of weight and height.

**Table 6**
Marginal (treatment) effect of obesity and share of selected expenditure categories at different points in the distribution of total annual medical expenditures (standard errors in parenthesis).

| | M.E. Pctl. | Marginal effect | Pctl. range | Inpatient | Outpatient | Prescription drugs | Other | Third party payments | Out-of-pocket |
|---|---|---|---|---|---|---|---|---|---|
| Men | 20th | 108 (27) | 0–20th | >0.00 | 0.40 | 0.21 | 0.39 | 0.55 | 0.45 |
| | 40th | 281 (86) | 20th–40th | >0.00 | 0.40 | 0.24 | 0.36 | 0.59 | 0.41 |
| | 60th | 532 (151) | 40th–60th | >0.00 | 0.39 | 0.30 | 0.31 | 0.62 | 0.38 |
| | 80th | 1321 (537) | 60th–80th | >0.00 | 0.38 | 0.35 | 0.26 | 0.67 | 0.33 |
| | 90th | 2832 (1444) | 80th–90th | 0.03 | 0.47 | 0.33 | 0.16 | 0.72 | 0.28 |
| | | | 90th–100th | 0.26 | 0.45 | 0.20 | 0.09 | 0.84 | 0.16 |
| Women | 20th | 180 (96) | 0–20th | >0.00 | 0.45 | 0.23 | 0.33 | 0.52 | 0.48 |
| | 40th | 460 (204) | 20th–40th | >0.00 | 0.43 | 0.26 | 0.31 | 0.58 | 0.42 |
| | 60th | 1258$^{ns}$ (3954) | 40th–60th | >0.00 | 0.43 | 0.30 | 0.27 | 0.62 | 0.38 |
| | 80th | 3429$^{ns}$ (4326) | 60th–80th | 0.01 | 0.45 | 0.33 | 0.21 | 0.67 | 0.33 |
| | 90th | 8307 (4903) | 80th–90th | 0.08 | 0.50 | 0.26 | 0.17 | 0.74 | 0.26 |
| | | | 90th–100th | 0.28 | 0.46 | 0.18 | 0.08 | 0.83 | 0.17 |

*Notes*: Data: MEPS 2000–2005. Standard errors are adjusted for the complex design of the MEPS and values are reported in 2005 USD. ns denotes that the estimate is not statistically significant at $\alpha = .10$. Other includes vision, dental, home health, and medical equipment, but not over-the-counter medications.

few individuals with very high BMI that incur very high medical expenditures.

We next investigate whether the average treatment effect of obesity varies across the distribution of total expenditures. First, however, we examine the types of expenditures incurred at various points in the distribution. Not surprisingly, inpatient costs represent a larger fraction of the costs for those with high expenditures than those with low expenditures. For example, Table 6 shows that hospital costs account for 1% or less of medical expenditures for men and women at the 80th percentile of expenditures and below, whereas it accounts for over a quarter of expenditures for men and women whose medical expenditures are in the top decile. Percent of expenditures on other utilization (dental, vision, home health, and medical equipment) follows the opposite pattern; it constitutes a third or more of spending at the 20th percentile but less than a tenth of spending in the top decile. Expenditures on prescription drugs represent less than a quarter of expenditures for those at the 20th percentile, rise to constitute a third of spending for those at the 80th percentile, before falling to represent 20% or less of spending of those in the top decile.

Table 6 also contains IV quantile treatment effect estimates for men and women at five different points in the distribution: the 20th, 40th, 60th, 80th, and 90th percentiles. Obesity has a small impact on expenditures for relatively healthy individuals with low baseline expenditures; it raises medical expenditures by just $108 for men and $180 for women at the 20th percentile of expenditures. However, obesity raises medical expenditures significantly at the upper end of the distribution. For example, at the 90th percentile of annual medical expenditures, the IV estimate of the impact of obesity on medical expenditures is $2832 for men and $8307 for women. At each of the five percentiles we examine, the point estimates for women are higher than those for men, although the estimates for women at the 60th and 80th percentiles of expenditures are not statistically significant.

### 4.4. Estimates of the aggregate direct medical costs of obesity for the U.S.

Our IV estimates allow us to provide estimates of the direct cost of obesity that are arguably more accurate than those in the previous literature. Table 7 contains estimates of annual medical costs (in 2005 USD) due to obesity based on our IV 2PM model that correspond to the population of adults with biological children, which averaged 33.4 million people during 2000–2005. Our estimates indicate that the annual direct medical cost of obesity averaged $26.0 billion over the six year period, of which $23.2 billion (89%) was borne by third party payers. In the most recent data, from 2005, the cost of obesity in this population was $28.8 billion, of which $25.7 billion (89%) was borne by third party payers.

**Table 7**
Annual medical cost of obesity for non-elderly adults with biological children (billions of 2005 USD; 90% confidence interval in parenthesis).

| Year | Total expenditures | Third party expenditures | Population (millions) |
|---|---|---|---|
| 2000 | 19.7 (10.8, 28.5) | 17.7 (9.8, 25.5) | 30.8 |
| 2001 | 23.6 (13.0, 34.2) | 20.8 (11.6, 29.9) | 33.2 |
| 2002 | 26.8 (15.0, 38.8) | 24.2 (13.7, 34.8) | 35.0 |
| 2003 | 26.7 (14.4, 39.0) | 23.8 (13.0, 34.6) | 34.2 |
| 2004 | 30.4 (17.5, 43.4) | 27.3 (15.9, 38.7) | 33.7 |
| 2005 | 28.8 (16.1, 41.6) | 25.7 (14.6, 36.9) | 33.7 |
| 2000–2005 average | 26.0 (14.6, 37.4) | 23.2 (13.3, 33.2) | 33.4 |

*Notes*: Data: MEPS 2000–2005. Confidence intervals are adjusted for the complex design of the MEPS.

Under the (admittedly, very strong) assumption that the effect of obesity in our subpopulation generalizes to the full non-institutionalized population of adults aged 18 and older, we estimate that total medical costs for the full non-institutionalized population of adults aged 18 and older was $190.2 billion in 2005 USD.[13] To put this in context, total U.S. health expenditures in 2005 for the non-institutionalized population of adults sampled in the MEPS were $923.2 billion (in 2005 dollars), so our estimates imply that 20.6% of U.S. national health expenditures is spent treating obesity-related illness. However, we acknowledge that the effect of obesity in our subpopulation (adults with children between 11 and 20 years) may not generalize to the entire population, so the primary focus of this paper is the marginal effect of obesity for our subpopulation rather than the national estimate.

### 4.5. Generalizability

Because of the instrument we use in our IV models, we are forced to limit our sample to adults with a biological child between the ages of 11 and 20 years. As a result, our estimates of obesity-attributable medical expenditures may not generalize to all adults. To explore differences between the two populations, we estimated

[13] Using the MEPS sampling weights we determined that non-institutionalized population of adults 18 years of age and older was 222.6 million in 2005, whereas the subpopulation of adults with biological children between the ages of 11 and 20 years was 33.7 million. Therefore, we multiplied our subpopulation estimate by 222.6/33.7 to inflate it to the full population.

the ordinary (non-IV) two-part GLM version of our model for the full sample of 20–64 year old adults (i.e. unconditional on number of children), as well as for the sample of 20–64 year old adults with biological children between the ages of 11 and 20 years (our IV sample). Comparing non-IV estimates for the two groups may yield information about the generalizability of results based on our IV sample to the general population. On average, the medical care costs associated with obesity were approximately 16% larger for the full sample of adults. One possible explanation is that the obese with biological children are in relatively better health than the obese who are childless. If the direction of bias is the same in the causal effect as in the association (which we caution we cannot verify), the above comparison suggests that the IV results for adults with children underestimates the effect of obesity on medical care costs for the general population of adults.

### 4.6. Falsification tests

In order to test the validity of our results we perform three falsification tests. The logic behind the first test is that there are some types of medical expenditures that obesity should not affect. It is challenging to find a category of medical costs that is unrelated to obesity because obesity has been linked by physicians to so many ailments. Trayhurn and Beattie (2001) discuss how fat cells secrete hormones and proteins that damage health, such as resistin that causes diabetes and leptin which can damage the cardiovascular system; many of the potentially adverse consequences of such secretions are not well understood. Hu (2008) documents the pathways by which obesity raises the risk of cancer and a wide variety of other conditions. However, we are aware of no established medical relationship between obesity and epilepsy, brain damage, and central nervous system disorders. Thus, our falsification test consists of using our IV method to examine whether obesity appears to affect expenditures on those conditions.[14] If our IV method indicates that obesity affects expenditures on such conditions, then that would suggest that the IV method lacks specificity and that the earlier results may be spurious.

The results of our IV model indicate that the causal effect of an additional unit of BMI on expenditures for epilepsy, brain damage, and central nervous system disorders is a statistically insignificant $4, and the effect of obesity on such expenditures is a statistically insignificant $72. Thus the IV model does not fail the falsification test; the model does not suggest that obesity raises expenditures for conditions that should be unaffected by it. Not failing a falsification test is of course different from proof that the model is valid, but it does increase confidence that the earlier findings are not spurious.

The second falsification test is whether our model predicts that obesity has a larger impact on diabetes-related medical expenditures than other medical expenditures. The logic is similar to that in the first falsification test: the model should find greater effects of obesity for conditions that one would, for clinical reasons, expect to see greater effects. The IV model indicates that obesity raises the probability of incurring diabetes-related expenditures by 33.4 percentage points, which is much greater than the impact of obesity on the probability of incurring non-diabetes-related expenditures (9.0 percentage points). The IV model reassuringly finds bigger effects where one should expect to find them.

Our third falsification test is to examine whether the weight of biologically unrelated stepchildren appear to be powerful instruments for respondent weight. The logic is that stepchildren and stepparents, lacking any biological relation, cannot be similar in weight because of genetics, so if the weights of the two are strongly correlated it must be for reasons other than genetics, which suggests that the instrument may be invalid. There are 407 non-elderly men and women in our original sample with stepchildren between the ages of 11 and 20. When we conduct *F*-tests of the power of our instruments in this case, the test statistics range between 2.1 and 4.1 and we cannot reject the null hypotheses that they are uncorrelated. In contrast, the average *F*-statistic is 144 for the weight of a biological child and the null hypothesis is strongly rejected in all cases.

Falsification tests are never definitive but can be helpful in demonstrating the sensitivity and specificity of the model. The results of these three falsification tests are consistent with the identifying assumptions of IV (i.e. the validity of the instrument) and are consistent with the model being sensitive and specific.

## 5. Discussion

This paper provides the first estimates of the impact of obesity on medical costs that adjust for endogeneity and measurement error in weight. The impact of obesity on annual medical costs (in 2005 dollars) is estimated to be $2741 for men and women pooled, $3613 for women, and $1152 for men (which is not statistically significant). These averages are driven by relatively few individuals with very high BMI and very high medical expenditures. The estimated effects are much greater than those from models that do not use IV to adjust for potential endogeneity and measurement error.

If one can generalize from the subsample we used, then the IV model implies that the effect of obesity on medical costs is significantly higher than estimated in the previous literature. This paper's estimate of the national medical care costs of obesity-related illness in adults is $209.7 billion, which is more than twice the estimate of $85.7 billion by Finkelstein et al. (2009)[15]; both estimates are derived from the MEPS data and are expressed in year 2008 dollars. The results of this paper suggest that 20.6% of U.S. national health expenditures are spent treating obesity-related illness, which is considerably higher than the previous estimate of 9.1% (Finkelstein et al., 2009).

Two important differences should be kept in mind when comparing our paper to the earlier literature (e.g. Finkelstein et al., 2009). First, this paper compares the obese to the non-obese (which includes the healthy weight and the overweight), whereas Finkelstein et al. (2009) compares the obese to the healthy weight. (The models used in this paper do not include an indicator variable for overweight because we lack additional instruments for

[14] Expenditures on epilepsy, brain damage, and central nervous system disorders are identified as those with Clinical Classification System CCS codes of 83, 85, 93–95, 210, 211.

[15] Specifically, the IV estimate of the impact of obesity on medical costs for men and women combined is $2741 in 2005 dollars (Table 3); adjusting from 2005 dollars to 2008 dollars requires multiplying by 1.10242 (according to the Bureau of Labor Statistics' inflation calculator), which results in the final estimate of $3022 in 2008 dollars. This is roughly twice the estimate of $1429 (also in 2008 dollars) by Finkelstein et al. (2009). Finkelstein et al. (2009) also provide estimates of the medical care costs of obesity that are based on data from the 2006 National Health Expenditure Accounts which, unlike MEPS, includes the institutionalized population and a wider array of health-related expenditures such as over-the-counter medicines. They multiply the fraction of total spending attributable to obesity from the MEPS by the total spending in NHEA, on the assumption that the percentage of medical costs due to obesity is the same for institutionalized and non-institutionalized populations. Because the NHEA covers a wider array of expenditures and the sicker institutionalized population, this results in a higher estimate of the aggregate medical costs of obesity: $147 billion in 2008 dollars. We focus in this paper on the MEPS data and thus compare our results to the MEPS results of Finkelstein et al. (2009).

additional categories of weight classification.[16]) It is reasonable to combine the healthy weight and overweight in this context because the two groups have similar health care costs (see Figs. 1–3) and mortality risks (see e.g. Mehta and Chang, 2009; Flegal et al., 2005). A second important difference to keep in mind is that the estimates in this paper are based on adults with at least one biological child between the ages of 11 and 20 years (because of our IV strategy), whereas the previous literature typically uses all adults. As a result, the individuals in our sample may be healthier on average.

One might ask whether our estimates of the causal effect of obesity on medical costs are implausibly high. Physicians have documented biological pathways through which obesity raises the risk of type 2 diabetes, cardiovascular disease and cancer (see, e.g., chapters 8–10 in Hu (2008)). Part of the challenge in identifying an appropriate falsification test was finding conditions not worsened by obesity. Thus, estimates of the impact of obesity on medical care costs would be suspect if they were not substantial.

There are at least two reasons that our estimates are higher than the associations reported in the previous literature. First, reporting error in weight and height may cause attenuation bias in previous estimates, and this reporting error is addressed by our IV method. We suspect that this is the primary explanation for the large discrepancy between the correlations and causal effects. Second, previous estimates may have suffered omitted variables bias. For example, certain subgroups with disproportionately high prevalence of obesity have reduced access to care (e.g. disadvantaged minorities and those of low socioeconomic status; see Fontaine and Bartlett, 2000), and the inability to control for access to care may have biased downward previous estimates of the association of obesity with medical care costs.

Our finding that the causal impact of obesity on medical care costs is greater than previously appreciated has important implications. For example, many estimates of the cost-effectiveness of anti-obesity interventions are based on published estimates of the association of weight with medical care costs, which underestimate the causal effect. For example, cost-effectiveness estimates for the school-based interventions Coordinated Approach to Child Health or CATCH (Brown et al., 2007) and Planet Health (Wang et al., 2003) are based on the estimates of the association of weight with medical costs reported in Gorsky et al. (1996) and Oster et al. (1999). As a result, the cost-effectiveness of CATCH and Planet Health have likely been underestimated.

Likewise, the results of this paper may be useful for estimates of the cost effectiveness of government programs to reduce and prevent obesity. Trasande (2010) recently estimated the cost-effectiveness of government spending to reduce childhood obesity using the estimates of adult medical expenditures attributable to obesity from Finkelstein et al. (2009). To the extent that our IV estimates are more accurate estimates of the impact of obesity on medical care costs, Trasande (2010) understates the cost-effectiveness of government spending to reduce childhood obesity. However, an important caveat is that interventions may affect subpopulations for whom the impact of obesity on medical care differs from that found for our subpopulation of adults with children aged 11–20 years.

The results of this paper also indicate that insurance companies spend more treating obesity-related illness than was previously thought, and thus there may be a better business case for insurance companies to cover effective treatments for obesity (such as prescription drugs for weight loss, bariatric surgery, and nutrition counseling) than was previously appreciated (Finkelstein and Trogdon, 2008).

Our findings also imply that the external costs of obesity that operate through insurance are likely to be greater than previously appreciated. This is relevant for government policy because "External costs provide one of the strongest economic justifications for government interventions. . ." (Zohrabian and Philipson, 2010, p. 2468). Thus the economic case for government intervention to reduce obesity-related externalities has been underestimated. There are several ways that such externalities could be reduced. Some have advocated taxes on energy dense food (e.g. Brownell and Frieden, 2009), although some research suggests that there may be little impact on weight from taxes on soda pop (Fletcher et al., 2010) or food away from home (Schroeter et al., 2008). Implementing cost-effective school-based interventions to prevent youth obesity could also reduce future obesity-related externalities (Cawley, 2010).

Some have noted that one way to internalize some of the external costs of obesity is to charge the obese higher premiums for health insurance (e.g. Finkelstein and Zuckerman, 2008). Recent health care reform legislation made it easier for employers to internalize to workers the external health care costs of obesity. Specifically, section 2705 of the 2010 Patient Protection and Affordable Care Act (PPACA) allows employers to provide premium discounts, rebates, or rewards of up to 30% of employee-only insurance premiums (up to 50% with approval from the Secretary of Health and Human Services) if they participate in qualifying wellness programs, such as those to promote healthy weight.

Bhattacharya and Sood (2007, 2010) point out that the obesity externalities that operate through pooling of risk in insurance result in deadweight loss only to the extent that they distort decisionmaking. (The pooling of obesity-related medical costs always results in a transfer from the non-obese to the obese, but this may or may not entail deadweight loss to society.[17]) There are two major ways that the obesity externality that operates through insurance distorts consumer decisionmaking. First, consumers may buy less insurance because the pooling of obesity-related medical care costs raises insurance premia. The literature on the premium elasticity of demand for health insurance finds that consumers are modestly sensitive to the price of insurance (e.g. Gruber and Washington, 2005; Marquis and Long, 1995), which is consistent with some deadweight loss through this pathway. The second way in which the obesity externality distorts consumer decisionmaking is through moral hazard; risk pooling ensures that the obese do not pay the full medical care costs of their obesity so they may invest less in weight loss or preventing weight gain. Using methods of instrumental variables that exploit state Medicaid expansions, Kelly and Markowitz (2010) and Bhattacharya et al. (2011) find evidence that having health insurance (i.e. the extensive margin) raises BMI[18]. Bhattacharya et al. (2011) find weaker evidence that

[16] To make a more apples-to-apples comparison, we used an IV model of medical expenditures as a function of BMI and BMI squared to compare the average difference in medical costs between those in the obese range and those in the healthy weight range. In general, the estimates are imprecise with considerable variation from year to year. For the year that estimates were most precise (2001), the implied aggregate costs of obesity (relative to being healthy weight) were 23.7% less than our estimate of the aggregate cost of obesity (relative to being non-obese). Based on this calculation, only slightly less than a quarter of the difference between the aggregate cost estimates of obesity found by this paper and Finkelstein et al. (2003) is driven by our comparison of the obese to the non-obese as opposed to the healthy weight.

[17] Because obesity is partly genetic, some may consider a certain amount of risk pooling through health insurance to be desirable.

[18] To clarify, in this paper we use exogenous variation in weight to estimate the impact of obesity on medical costs. In contrast, Kelly and Markowitz (2010) and Bhattacharya et al. (2011) exploit exogenous variation in insurance coverage to measure the effect of insurance coverage on weight.

the generosity of insurance (i.e. the intensive margin) raises BMI. Bhattacharya and Sood (2007) calculate that the welfare loss in the U.S. associated with this second channel totals $150 per capita (in 1998 dollars). In summary, the obesity related externality that operates through insurance seems to be not just a transfer from the non-obese to the obese, but to also impose deadweight loss on society by distorting decisions about purchases of insurance and about optimal investment in weight loss and prevention of weight gain.

The results of this paper cannot alone indicate whether increased cost sharing for obesity-related treatments would increase or decrease social welfare. On the one hand, a high deductible represents an incentive for the insured to keep his weight at a level that avoids obesity-related medical expenditures, and may reduce moral hazard of the type identified by Kelly and Markowitz (2010), Bhattacharya et al. (2011), and Bhattacharya and Sood (2007). On the other hand, if treatment programs are cost saving or highly cost effective, society may not wish to discourage their use, and there is a case for insurers to fully cover such services.

Future directions for research include finding additional natural experiments that affect weight but do not directly affect medical care expenditures. These would shed light on the generalizability of the results presented here. It would also be useful to test explanations for differences between subsamples in the impact of obesity on medical expenditures. For example, our point estimate of the impact of obesity on medical expenditures is higher for Medicaid recipients ($3674) and the uninsured ($3153) than those with private insurance coverage ($2568); it would be useful to know whether this is due to differences in the health impact of obesity across the three groups or due to differences in the ways that the groups use medical care (e.g. are the uninsured and those covered by Medicaid more likely to visit the emergency department whereas those with private coverage are likely to use an outpatient visit, in response to the same condition?).[19]

This paper has several limitations. The first limitation that should be discussed in any paper that uses IV concerns the validity of the instrument. Our identifying assumption is that the weight of a biological relative is strongly correlated with the respondent's weight, but uncorrelated with residual medical care costs. A large literature in behavioral genetics confirms that there is a strong genetic component to weight, and that any similarity in weight due to shared environment is so small as to be undetectable. However, other threats to the validity of the instrument are that the genes that affect weight may also affect other things that could directly affect residual medical care costs (pleitropy), and the genes that affect weight may lie next to genes that directly affect residual medical care costs (proximity matters because genes are inherited in blocks). We are unable to test whether these are problems in the current context, but, like the previous literature (e.g. Smith et al., 2009; Kline and Tobias 2008; Cawley, 2004), we acknowledge them as possible limitations of the instrument.

We investigated the possibility that having obese children might lead to further contact with the health care system, which in turn could lead to greater utilization by the parents themselves. However, when we regress the probability of incurring any medical expenditures on respondent BMI, respondent BMI squared, and an indicator variable for whether the child is obese, having an obese child is associated with less than a one percentage point increase in the probability of incurring medical care costs. (And even that may be due in part to child weight (through genetics) being a proxy for true respondent weight (which is reported with error).) Although our instrument passes the standard statistical tests for power and over-identification, as always there is no way to prove the validity of an instrument; one can only fail to reject the null hypothesis that the instrument is valid.

Another limitation is that we have data on BMI and medical care costs for only a single interval of time for each respondent. Ideally we would have longitudinal data that would allow us to examine the life cycle impacts of obesity, including early mortality (the costs of which are presumably mainly internal to the household), and to estimate the importance of duration of obesity (which is unknown in the MEPS) for medical costs.[20]

Like the previous literature, this paper uses BMI, which has been criticized as a measure of fatness because it does not distinguish fat from muscle (Burkhauser and Cawley, 2008). Ideally, we would use more accurate measures of fatness such as percent body fat, but only the components of BMI (weight and height) are available in the MEPS. Another limitation is that weight and height are self-reported or proxy-reported instead of measured. Although the IV method helps eliminate the influence of reporting error, measured values would be preferable. Because we can implement our IV method only for adults with biological children, caution should be used when generalizing our results to the entire population of U.S. adults.

Despite these limitations, this paper makes an important contribution by providing the first estimates of the causal impact of obesity on medical care costs. The estimates of the causal effect are significantly higher than the estimates of the association that are published in the previous literature, with corresponding implications for insurance and government policy.

## References


Abadie, A., Angrist, J., Imbens, G., 2002. Instrumental variables estimates of the effect of subsidized training on the quantiles of trainee earnings. Econometrica 70, 91–117.

Anderson, G., 2007. From 'Soak the Rich' to 'Soak the Poor': recent trends in hospital pricing. Health Affairs 26 (3), 780–789.

Agency for Healthcare Research and Quality (AHRQ), 2007. Clinical Classification Software (CCS) for ICD-9-CM Fact Sheet, http://www.hcup-s.ahrq.gov/toolssoftware/ccs/ccsfactsheet.jsp (accessed 22.07.10).

Agency for Healthcare Research and Quality (AHRQ), 2010. MEPSnet/HC Query Tool, http://www.meps.ahrq.gov/mepsweb/data_stats/MEPSnetHC.jsp (accessed 22.07.10).

Bhattacharya, J., Bundorf, K., Pace, N., Sood, N., 2011. Does health insurance make you fat? In: Grossman, M., Mocan, N.H. (Eds.), Economic Aspects of Obesity. University of Chicago Press, Chicago IL (Chapter 2).

Bhattacharya, J., Sood, N., 2007. Health insurance and the obesity externality. Advances in Health Economics and Health Services Research 17, 279–318.

Bhattacharya, J., Sood, N., 2010. Who Pays for Obesity? Unpublished manuscript, Stanford University.

Black, D.A., Berger, M.C., Scott, F.A., 2000. Bounding parameter estimates with nonclassical measurement error. Journal of the American Statistical Association 95 (451), 739–748.

Bound, J., Brown, C., Mathiowetz, N., 2002. Measurement error in survey data. In: Heckman, J., Leamer, Ed. (Eds.), Handbook of Econometrics, vol. 5. Springer-Verlag, New York, pp. 3705–3843.

Brown III, H.S., Pérez, A., Li, Y.-P., Hoelscher, D.M., Kelder, S.H., Rivera, R., 2007. The cost-effectiveness of a school-based overweight program. Journal of the International Society of Behavioral Nutrition and Physical Activity 4, 47.


[19] It is possible that such moral hazard from insurance documented by Kelly and Markowitz (2010) and Bhattacharya et al. (2011) could lead to different local average treatment effects for our IV in the insured and uninsured groups, partly explaining the larger impact of obesity on medical care costs for the uninsured ($3153) than those with private insurance ($2568). For example, having private insurance coverage could lead to greater use of preventive care that reduces the medical expenditures associated with becoming obese.

[20] Although the MEPS conducts multiple interviews, they are only months apart and as a result are not sufficiently longitudinal to observe the timing of the onset of obesity or determine how duration of obesity affects the impact of obesity on medical care costs. As a result, our estimates derived from the MEPS represent the marginal effect of obesity for the average duration of obesity.

Brownell, K.D., Frieden, T.R., 2009. Ounces of prevention – the public policy case for taxes on sugared beverages. New England Journal of Medicine 360 (18), 1805–1808.
Burkhauser, R.V., Cawley, J., 2008. Beyond BMI: the value of more accurate measures of fatness and obesity in social science research. Journal of Health Economics 27 (2), 519–529.
Burkhauser, R.V., Cawley, J., Schmeiser, M.D., 2009. The timing of the rise in U.S. obesity varies with measure of fatness. Economics and Human Biology 7 (3), 307–318.
Carroll, R.J., Ruppert, D., Stefanski, L.A., 1995. Measurement Error in Nonlinear Models. Chapman and Hall, New York, NY.
Cawley, J., 2004. The impact of obesity on wages. Journal of Human Resources 39 (2), 451–474.
Cawley, J., 2010. The economics of childhood obesity. Health Affairs 29 (3), 364–371.
Cawley, J., Burkhauser, R.V., 2006. Beyond BMI: the value of more accurate measures of fatness and obesity in social science research. National Bureau of Economic Research Working Paper #12291.
Comuzzie, A.G., Allison, D.B., 1998. The search for human obesity genes. Science 280 (5368), 1374–1377.
Deb, P., 2007. STATA ado file and documentation for TREATREG2, version 1.1.1.
Deb, P., Trivedi, P., 2006a. Maximum simulated likelihood estimation of a negative binomial regression model with multinomial endogenous treatment. The STATA Journal 6 (2), 246–255.
Deb, P., Trivedi, P., 2006b. Specification and simulated likelihood estimation of a non-normal treatment-outcome model with selection: application to health care utilization. Econometrics Journal 9, 30–331.
Dixon, J.B., 2010. The effect of obesity on health outcomes. Molecular and Cellular Endocrinology 316, 104–108.
Finkelstein, E.A., Trogdon, J.G., 2008. Public health interventions for addressing childhood overweight: analysis of the business case. American Journal of Public Health 98 (3), 8–12.
Finkelstein, E.A., Trogdon, J.G., Cohen, J.W., Dietz, W., 2009. Annual medical spending attributable to obesity: payer- and service-specific estimates. Health Affairs, Web Exclusive. July 27, 2009.
Finkelstein, E.A., Fiebelkorn, I.C., Wang, G., 2003. National medical spending attributable to overweight and obesity: how much and who's paying? Health Affairs, W3–W219.
Finkelstein, E.A., Zuckerman, L.L., 2008. The Fattening of America. John Wiley & Sons, Inc., NJ.
Flegal, K.M., Carroll, M.D., Kuczmarski, R.J., Johnson, C.L., 1998. Overweight and obesity in the United States: prevalence and trends, 1960–1994. International Journal of Obesity and Related Metabolic Disorders 22 (1), 39–47.
Flegal, K.M., Carroll, M.D., Ogden, C., Curtin, L.R., 2010. Prevalence and trends in obesity among US adults, 1999–2008. Journal of the American Medical Association 303 (3), 235–241.
Flegal, K., Graubard, B., Williamson, D., Gail, M., 2005. Excess deaths associates with underweight overweight, and obesity. Journal of the American Medical Association 293 (15), 1861–1867.
Fletcher, J.M., Frisvold, D., Tefft, N., 2010. Can soft drink taxes reduce population weight? Contemporary Economic Policy 28 (1), 23–35.
Fontaine, K.R., Bartlett, S.J., 2000. Access and use of medical care among obese persons. Obesity Research 8, 403–406.
Frazis, H., Loewenstein, M., 2003. Estimating linear regressions with mismeasured, possibly, endogenous binary explanatory variables. Journal of Econometrics 117, 151–178.
Frolich, M., Melly, D., 2008. Estimation of Quantile Treatment Effects with STATA. Working Paper. University of Mannheim, 22nd July, 2008.
Gorsky, R.D., Pamuk, E., Williamson, D.F., Shaffer, P.A., Koplan, J.P., 1996. The 25-year health care costs of women who remain overweight after 40 years of age. American Journal of Preventive Medicine 12 (5), 388–394.
Grilo, C.M., Pogue-Geile, M.F., 1991. The nature of environmental influences on weight and obesity: a behavioral genetic analysis. Psychological Bulletin 110 (3), 520–537.
Gruber, J., Washington, E., 2005. Subsidies to employee health insurance premiums and the health insurance market. Journal of Health Economics 24 (2), 253–276.
Haberstick, B.C., Lessem, J.M., McQueen, M.B., Boardman, J.D., Hopfer, C.J., Smolen, A., Hewitt, J.K., 2010. Stable genes and changing environments: body mass index across adolescence and young adulthood. Behavior Genetics 40 (4), 495–504.
Hansen, L., 1982. Large sample properties of generalized method of moments estimators. Econometrica 50, 1029–1054.
Hardin, J., Schmiediche, H., Carroll, R., 2003. Instrumental variables bootstrapping, and generalized linear models. The STATA Journal 3 (4), 351–360.
Hewitt, J.K., 1997. The genetics of obesity: what have genetic studies told us about the environment? Behavior Genetics 27 (4), 353–358.
Hill, S., Miller, G., 2009. Health expenditure estimation and function form: applications of the generalized gamma and extended estimating equations models. Health Economics, doi:10.1002/hec.1498, Published Online: May 11 2009.
Hu, F.B., 2008. Obesity Epidemiology. Oxford University Press, New York.
Jones, A., 2000. Health econometrics. In: Culyer, A.J., Newhouse, J.P. (Eds.), Handbook of Health Economics, vol. 1A, pp. 265–336 (Chapter 6).
Kelly, I.R., Markowitz, S., 2009/2010. Incentives in Obesity and Health Insurance. Inquiry 46 (4), 418–432.
Kline, B., Tobias, J.L., 2008. The wages of BMI: Bayesian analysis of a skewed treatment–response model with nonparametric endogeneity. Journal of Applied Econometrics 23, 767–793.
Komlos, J., Brabec, M., 2010. The trend of mean BMI values of US adults, birth cohorts 1882–1986 indicates that the obesity epidemic began earlier than hitherto thought. American Journal of Human Biology 22 (5), 631–638.
Kortt, M.A., Langley, P.C., Cox, E.R., 1998. A review of cost-of-illness studies on obesity. Clinical Therapeutics 20 (4), 772–779.
Maes, H.H.M., Neale, M.C., Eaves, L.J., 1997. Genetic and environmental factors in relative body weight and human adiposity. Behavior Genetics 27 (4), 325–351.
Manning, W.G., Mullahy, J., 2001. Estimating log models: to transform or not to transform? Journal of Health Economics 20 (4), 461–494.
Marquis, M.S., Long, S.H., 1995. Worker demand for health insurance in the nongroup market. Journal of Health Economics 14 (1), 47–63.
Mehta, N.K., Chang, V.W., 2009. Mortality attributable to obesity among middle-aged adults in the United States. Demography 46 (4), 851–872.
Nelson, M.C., Gordon-Larsen, P., North, K.E., Adair, L.S., 2006. Body mass index gain, fast food and physical activity: effects of shared environment over time. Obesity 14, 701–709.
Oster, G., Thompson, D., Edelsberg, J., Bird, A.P., Colditz, G.A., 1999. Lifetime health and economic benefits of weight loss among obese persons. American Journal of Public Health 89 (10), 1536–1542.
Plankey, M., Stevens, J., Rust, P., 1997. Prediction equations do not eliminate systemic error in self-reported body mass index. Obesity Research 5 (4), 308–314.
Price, R.A., Gottesman, I.I., 1991. Body fat in identical twins reared apart: roles for genes and environment. Behavior Genetics 21 (1), 1–7.
Roux, L., Donaldson, C., 2004. Economics and obesity: costing the problem or evaluating solutions? Obesity Research 12 (2), 173–179.
Schroeter, C., Lusk, J., Tyner, W., 2008. Determining the impact of food price and income changes on body weight. Journal of Health Economics 27 (1), 45–68.
Seidell, J., Verschuren, W., van Leer, E., Kromhout, D., 1996. Underweight, overweight, and mortality. Archives of Internal Medicine 156 (9), 958–963.
Shiell, A., Gerard, K., Donaldson, C., 1987. Cost of illness studies: an aid to decision-making? Health Policy 8, 317–323.
Smith, G.D., Jonathan, A.C.S., Abigail, F., Per, T., Debbie, A.L., Finn, R., 2009. The association between BMI and mortality using offspring BMI as an indicator of own BMI: large intergenerational mortality study. British Medical Journal 339, b5043.
Sorensen, T.I.A., Stunkard, A.J., 1993. Does obesity run in families because of genes? an adoption study using silhouettes as a measure of obesity. Acta Psychiatrica Scandanavia 370 Suppl., 67–72.
Stock, J.H., Wright, J.H., Yogo, M., 2002. A survey of weak instruments and weak identification in generalized method of moments. Journal of Business and Economic Statistics 20 (4), 518–529.
Stunkard, A.J., Sorensen, T.I.A., Hanis, C., Teasdale, T.W., Chakraborty, R., Schull, W.J., Schulsinger, F., 1986. An adoption study of human obesity. New England Journal of Medicine 314 (4), 193–198.
Thorpe, K.E., Florence, C.S., Howard, D.H., Peter, J., 2004. The impact of obesity on rising medical spending. Health Affairs 23 (6), W4480–W4486.
Trasande, L., Liu, Y., Fryer, G., Weitzman, M., 2009. Effects of childhood obesity on hospital care and costs, 1999–2005. Health Affairs 28 (4), w751–w760.
Trasande, L., 2010. How much should we invest in preventing childhood obesity? Health Affairs 29 (3), 372–378.
Trayhurn, P., Beattie, J.H., 2001. Physiological role of adipose tissue: white adipose tissue as an endocrine and secretory organ. Proceedings of the Nutrition Society 60, 329–339.
U.S. Department of Health and Human Services, 2002. 2000 CDC growth charts for the United States: methods and development. In: Vital and National Health Statistics, Series 11, No. 246, May.
U.S. Department of Health and Human Services, 2010, January. The Surgeon General's Vision for a Healthy and Fit Nation. U.S. Department of Health and Human Services, Office of the Surgeon General, Rockville, MD.
Villanueva, E.V., 2001. The validity of self-reported weight in US adults: a population-based cross-sectional study. BMC Public Health 1 (11).
Vogler, G.P., Sorensen, T.I.A., Stunkard, A.J., Srinivasan, M.R., Rao, D.C., 1995. Influences of genes and shared family environment on adult body mass index assessed in an adoption study by a comprehensive path model. International Journal of Obesity 19, 40–45.
Wang, L.Y., Yang, Q., Lowry, R., Wechsler, H., 2003. Economic analysis of a school-based obesity prevention program. Obesity Research 11, 1313–1324.
Wardle, J., Carnell, S., Haworth, C.M.A., Plomin, R., 2008. Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. American Journal of Clinical Nutrition 87, 398–404.
Zohrabian, A., Philipson, T.J., 2010. External costs of risky health behaviors associated with leading actual causes of death in the U.S.: a review of the evidence and implications for future research. International Journal of Environmental Research Public Health 7, 2460–2472.