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

John Cawley[a,b,c,*], Chad Meyerhoefer[d]

[a] Department of Policy Analysis and Management, Cornell University, United States
[b] Department of Economics, Cornell University, United States
[c] NBER, United States
[d] Department of Economics, Lehigh University, United States

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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

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* Corresponding author at: Department of Policy Analysis and Management, Cornell University, Ithaca NY 14853, United States. Tel.: +1 607 255 0952; fax: +1 607 255 4071.
E-mail addresses: JHC38@cornell.edu (J. Cawley), CHM308@lehigh.edu (C. Meyerhoefer).

[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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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.