Challenges for causal inference in obesity research

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1 Introduction.

Obesity research is largely an observational science. Some research involves controlled experiments in which a possible contributor to body weight is randomized by the experimenter, but randomizing body weight directly is in principle impossible. Further, the causes of body weight are usually not subject to experimental manipulation: researchers cannot randomize genes, individual characteristics such as income or education or family background, nor contextual influences such as food prices or social norms. For these reasons, social science researchers typically attempt to infer the causes and consequences of body weight using observational data. In this chapter we briefly outline the empirical strategies quantitative social scientists commonly use to make causal inferences in the absence of randomized experiments and then highlight particularly challenging issues in obesity research.

2 A brief overview of causal inference in quantitative social science.

To fix ideas for the discussion that follows, in this section we present a non-technical summary of several approaches to making causal inferences when the researcher only
has observational data available, that is, data in which the researcher cannot control
the values of the treatment of interest.\(^1\) Suppose we are interested in estimating the
causal effect of some variable \(x\) on another \(y\). A univariate model for the outcome can
be written,

\[
y = \beta x + u,
\]

where \(\beta\) denotes the causal effect of a one-unit change in \(x\) on \(y\) and \(u\) represents all causes
of \(y\) other than \(x\).\(^2\) The research goal is to estimate \(\beta\). We refer to \(x\) as the \textit{treatment}
and \(y\) and as the \textit{outcome}. If we were concerned with estimating the causes of overweight
or obesity, then the outcome \(y\) might be the body mass index (BMI), the ratio of hip
to waist circumference, or perhaps a measure of caloric expenditure or caloric intake or
nutrition. The treatment \(x\) could be: some economic variable such as relative price of
healthy vs. unhealthy food, or income, or the opportunity cost of engaging in leisure
time physical activity; the features of ones physical environment, such as proximity to
parks, or the ease with which one can walk or bike to work or a shop; the features of ones
social environment, such as the physical activity and dietary habits of ones friends, or
social norms concerning body weight; or a medical treatment, such as bariatric surgery.
In studies that assess the consequences of overweight/obesity, the treatment is some
measure of overweight/obesity, and the outcome \(y\) might be a health outcome, such as
mortality, functional impairment, medical expenditures, or the presence of diabetes or
some other chronic disease; or \(y\) might be a labour market outcome, such as employment
or earnings; or \(y\) might be any of a number of other physical, mental, or social outcomes
of interest to the researcher.

A linear least squares regression of \(y\) on \(x\) does not generally recover an unbiased
estimate of the causal effect of \(x\) on \(y\), that is, the linear regression estimator of \(\beta\) in

\(^1\)See Pearl (2000) or Heckman (2005) for extensive discussions of causality and causal modeling.
\(^2\)Assume throughout that all variables are measured in deviations from sample means so that the
constant term is zero.
equation (2) is not generally centered on the true causal effect of $x$ on $y$. This problem persists even if the sample size is arbitrarily large. As the sample size increases without bound, the least squares estimator, denoted $\hat{\beta}$, converges to

$$\hat{\beta} \to \frac{\text{Cov}(x, y)}{V(x)} = \beta + \frac{\text{Cov}(x, u)}{V(x)},$$  \hspace{1cm} (2)$$

where Cov denotes covariance and $V$ variance.\(^3\) The regression estimate does not recover the causal effect of $x$ on $y$ unless $\text{Cov}(x, u) = 0$. That is, if other causes of the outcome, $u$, are correlated with the treatment, $x$, then a regression of $y$ on $x$ yields an inconsistent estimate of the causal effect of $x$ on $y$. Other causes of $y$ will be correlated with $x$ if (1) $x$ is measured with error, or (2) $y$ causes $x$ (“reverse causality”), or (3) there are variables which do not appear in the model which affect both $y$ and $x$ (“unobserved heterogeneity”). Any of these problems, referred to in the econometrics literature as endogeneity, imply that correlations between the treatment and outcome do not reveal the causal effect of the treatment on the outcome.

**Standard problems with observational data.** Body weight outcomes are likely to be endogenous to other health and social outcomes. If the researcher is trying to explain body weight, so that $y$ is a measure of weight and $x$ is some possible cause of weight, we rarely have reason to believe that the cause in question is uncorrelated with other causes $u$. For example, if we find that schooling and body weight are negatively correlated, we cannot conclude that more education causes lower body weight because other factors (such as personality characteristics, cognitive ability, or family background) may cause both schooling and body weight. Or suppose that we find that the likelihood that an individual is obese ($y$) is higher the greater is the density of fast food restaurants within five miles of her residence ($x$). It is possible that $x$ exerts a causal impact on $y$. But

\(^3\)Derivation of the estimator and its sampling properties can be found in any econometrics or regression analysis textbook, see for example Wooldridge (2001).
it could also be that both fast food restaurants and obese individuals tend to locate in lower income areas. In other words, both $x$ and $y$ are associated with some other variable which may not be observable to the researcher. Further, it could be that $x$ and $y$ are mutually determined. It could be that density of fast food restaurants causally determines obesity prevalence, but that areas of higher obesity prevalence attract additional fast food restaurants, so that obesity “reverse” causes fast food outlet density.

Similarly, personal characteristics such as income, education, or the number of close friends who are obese are likely to be caused by the same unobservable characteristics which cause overweight or obesity. If we are attempting to determine a consequence of obesity, so that $x$ is a measure of obesity and $y$ is some other outcome, we could only claim that the correlation between $x$ and $y$ means $x$ causes $y$ if we can safely assume that all other causes of $y$ are uncorrelated with body weight. Genetic, developmental, educational, social, economic, and contextual factors which influence body weight $x$ cannot be assumed to be uncorrelated with unobserved causes of body weight $u$. For example, evidence that adolescents who skip breakfast are more likely to be overweight does not imply that skipping breakfast causes overweight, because many personality characteristics unobservable to the statistician (for example, poor eating habits in general) may affect both propensity to skip breakfast and body weight, or overweight may “reverse” cause increased propensity to skip breakfast.\(^4\)

**Randomized controlled trials.** Note that if $x$ can be controlled by the researcher, the researcher can randomize $x$ with respect to all other causes of $y$ and, therefore, make valid causal inferences. In obesity research, medical treatments for obesity can be randomized, but typically personal or social possible causes of body weight, such as income, education, smoking, peers, and food prices, are either costly or impossible to randomize through controlled experimentation.\(^5\) Since controlled experiments are

\(^4\)See Rampersaud et al. (2005) for a summary of the literature on breakfast habits and, among other outcomes, body weight.

\(^5\)See Padwal et al. (2003) for a review of randomized trials on medical interventions for overweight
frequently costly or infeasible, obesity research commonly attempts to use observational data to infer causation.

**Covariate adjustment.** Researchers attempting to estimate causal effects with observational data often use covariate adjustment to mitigate difficulties when controlled randomization is not possible. One can include other variables in the model which make observational data look more like a controlled experiment in the sense that we can control for some common causes of both \( x \) and \( y \). For example, controlling for education in a regression of body weight on income removes one possible common cause of both income and body weight. This approach rarely leads to models which have compelling causal interpretations because there will always be important causes of body weight and other outcomes which cannot be observed by the researcher. Including more control variables in the regression, then, may mitigate but does not generally solve the problem. Typically covariate adjustment is performed through multivariate regression techniques, but closely related methods called matching estimators are becoming popular. Matching estimators, like regression estimators, fail to recover causal effects if there are unobserved causes of both the treatment and the outcome, but matching estimators impose fewer parametric assumptions than conventional regression estimators (Heckman et al., 1998). A special case of covariate adjustment called regression discontinuity designs allow the researcher to make causal inferences even in the presence of reverse causation or confounding by exploiting discontinuities in the causes of the treatment. For example, Schanzenbach (2009) estimates the causal effect of school lunches on children’s body weight by, in effect, statistically comparing the body weight of children just below the income eligibility threshold to those just above—unobserved causes of both income and body weight should be smooth as income passes this point, but school lunch takeup is discontinuous at this point, allowing Schanzenbach to estimate the causal effect of obesity. See Robinson (1999) for an example of a controlled experiment on a behavioral cause of obesity, television viewing.
**Instrumental variables.** Instrumental variable (IV) methods can sometimes produce credible causal estimates in the absence of controlled experiments.\(^6\) IV methods require *instruments*: variables that affect the outcome \(y\) only because they affect the value of the treatment \(x\) and for no other reason. Such instruments, which might not actually exist, cause quasi–experimental variation in \(x\). The IV estimator uses this variation and the attendant variation in \(y\) to estimate a causal effect. For example, if a controlled experiment is conducted in which a randomly assigned treatment affects diet, and diet in turn affects weight, then the part of variation in weight across people attributable to experimental assignment can be used to infer the causal effect of weight on other outcomes. An example of IV methods in obesity research is Cawley (2004). Cawley estimates the effect of body weight on wages. To do so, he assumes that mother’s body weight causes daughter’s wage only because mother’s body weight causes daughter’s body weight, which in turn affects daughter’s wage.

If \(z\) is an instrumental variable such as mother’s body weight, then, as the sample grows in size, \(\hat{\beta}_{IV}\), the linear instrumental variable estimator of \(\beta\), converges to

\[
\hat{\beta}_{IV} \to \frac{\text{Cov}(z, y)}{\text{Cov}(z, x)} = \beta + \frac{\text{Cov}(z, u)}{\text{Cov}(z, x)}.
\]

The IV estimator recovers a causal effect of \(x\) on \(y\) if, and only if, \(z\) causes \(y\) only via \(z\)’s effect on \(x\). In other words, all other causes of \(y\) besides \(x\) are uncorrelated with \(z\): \(\text{Cov}(z, u) = 0\). If that assumption fails and \(z\) and \(u\) covary, then the estimator may provide highly misleading estimates. As discussed further below in the context of obesity research, recent literature has highlighted serious difficulties with IV estimators when the instrumental variables either explain little of the variation in the treatment or are

\(^6\)See Auld (2006) or Grootendorst (2007) for lengthier and more technical discussions of IV methods in health research.
even slightly correlated with unobserved causes of the outcome, that is, \( z \) and \( u \) are even slightly correlated. Another recent strand of literature points out that the implicit assumption in equation (2) that all individuals experience the same effect on \( y \) of a given change in \( x \) (that is, that \( \beta \) does not vary across people) is not innocuous in this context, as IV estimates recover a weighted average of causal effects for people affected by changes in the instruments. If the causal effect varies across people, then the researcher will recover different causal effects using different instruments even when textbook conditions are satisfied because different instruments affect the treatment status of different people.\(^7\) The most important difficulty with IV methods, however, is that it is often difficult or impossible to find good instruments, a problem we return to later in this chapter.

**Methods exploiting panel data.** Panel data—data which tracks individuals over time and give us multiple observations on a person’s body weight and other outcomes—provides another approach to causal inference. If, in equation (2), we suppose that all other causes of \( y \), \( u \), is the sum of a component which does not vary over time and a component that does, then it is possible to remove the time-invariant component via a suitable transformation of the equation. Removal of the time-invariant portion of \( u \) eliminates a potential source of correlation between \( u \) and \( x \). One transformation that removes the time-invariant, individual-specific unobserved component is the first difference. For example, if \( y \) is wages and \( x \) is body weight, the correlation between \( x \) and \( y \) reflects in part aspects of personality which affect both weight and outcomes in the labor market. With longitudinal data, we can ask whether changes in a subjects body weight over time are correlated with changes in her wages instead of just how levels of body weight are correlated with levels of wages. If personality is fixed over the sampling window for the population of interest, then personality does not affect these changes and, provided that changes in weight are not correlated with any unobservable

\(^7\)See Imbens and Angrist (1994) or Heckman et al. (2006) for discussions of instrumental variables estimation when causal effects differ across units.
time-varying determinants of wages, we can estimate causal effects. Various regression methods embody this intuition, including fixed effects regression, differenced models including difference–in–difference estimation, and long difference models.\textsuperscript{8} Less frequently, panels may include a dimension other than time which gives multiple observations on the same cross-sectional unit; one can then employ variants of these methods. Examples include data on twins in which differences between twins’ outcomes are used analogously to differences across time to remove common genetic causes and data in which multiple members of the same family are observed such that family–specific factors can be statistically removed.\textsuperscript{9}

Summarizing this section, social scientists studying obesity generally cannot conduct controlled experiments to make causal inferences. Methods which use observational data to make such inferences usually rely on some combination of: multivariate regression methods which hold observable confounders fixed, instrumental variable methods which infer causality from correlations between the outcome and variables which should only affect the outcome because they affect regressors of interest, or longitudinal data methods which difference out common causes of the treatment and the outcome. In the remainder of this chapter we discuss the use of these methods in obesity research, highlighting difficulties which make their use particularly challenging in the context of body weight.

3 Challenges to causal inference in obesity research.

3.1 Measurement of body weight and other variables.

One or more of the variables of interest, either the outcome \( y \) or an explanatory variable \( x \), may be difficult to measure. Such difficulties often arise in empirical obesity research.

\textsuperscript{8}See Wooldridge (2001) for a review of panel methods.

\textsuperscript{9}See Maes et al. (2005) for an example of an effort using twins data to determine the portion of body weight variability attributable to genetics. See Anderson et al. (2003) for an obesity research example of the use of family fixed effects.
Indeed, one need look no further than the concept of obesity itself. It is well known that
the standard measurement instrument, BMI, is a noisy measure of underlying obesity,
for at least two reasons. First, BMI, height adjusted weight, fails to capture important
dimensions of obesity. For instance, it does not distinguish fat mass from lean mass.
Nor does it distinguish the location of fat mass, yet upper-body obesity is more strongly
associated with metabolic disorders and cardiovascular disease than is lower-body obe-
sity.10 There is some evidence that the hip-to-waist ratio is a better measure of obesity
than BMI (Yusuf et al., 2005), yet hip-to-waist measurements are generally unavailable
in the survey data. Second, the inputs into the BMI formula, height and weight, are
frequently misreported by survey respondents. This misreporting is not random. In-
stead, in some groups, reported BMI tends to understate actual BMI and the degree of
under-reporting increases with actual BMI (Cawley, 2000).

Obesity is not the only variable that is hard to measure accurately in this line of
research. Other examples include the “built environment”: how does one accurately
measure the degree to which the layout of streets, sidewalks and buildings promotes or
hinders physical activity? Or consider food prices. The price that one pays for the same
item varies considerably depending on where it is purchased (a local convenience store,
say, or a warehouse type retail outlet such as Costco). It is also hard to model prices
for the entire array of foods that are available (Powell and Chaloupka, 2009).

Measurement error adversely affects the properties of one’s estimator, although the
manner in which the estimator is affected depends on specifics. Measurement error in an
explanatory variable renders conventional estimators of causal effects inconsistent. If x
is measured with error in equation (2) and the error is random, the estimate of the causal
effect of x on y will be biased towards zero, and we will tend to systematically find that

10 See Burkhauser and Cawley (2008) for a discussion of BMI and other measures of body weight
outcomes in social science research. See Ben-Noun et al. (2001); Willett et al. (1999) for evidence on
fat distribution and health.
the effect is smaller than it truly is.\textsuperscript{11} Random measurement error in an *outcome* variable deflates the precision of one’s estimator, which, unlike inconsistency, can be remedied by increasing one’s sample size. If the measurement error in the outcome varies with the treatment $x$ or other covariates in the model, then the estimator is generally inconsistent. Similarly, if a binary outcome (such as obese or not) is mismeasured, then the standard estimators (logit or probit) are also inconsistent (Kennedy, 2008). Finally, panel methods relying on differencing or fixed effects exacerbate problems arising from measurement error. These methods use changes in body weight rather than levels of body weight, and changes in observed body weight may largely reflect noisy measurement. The statistical properties of panel estimators are often, therefore, poor when measurement error is present.\textit{(Griliches and Hausman, 1986).}

How can one deal with the problems caused by measurement error? If the error occurs in a continuous outcome variable, such as BMI, one can follow Cawley (2000) and estimate the relationship between self-reported and actual BMI using data from a survey which contains information on both variables (such as the US National Health and Nutrition Examination Survey) and, using this estimated relationship, replace self-reported with predicted actual BMI. If the measurement error occurs in an explanatory variable, then one can use an instrumental variables estimator, provided that one has access to either multiple independent measures of the problematic explanatory variable or other variables that are correlated with the problematic explanatory variable but not with unobserved causes of the dependent variable.

\textsuperscript{11}By “random” in this context we mean: the measurement error in $x$ is statistically independent of the error term $u$. 
3.2 Difficulty explaining body weight with observable characteristics and contexts.

In multiple regression analyses common in the obesity research literature, it is standard practice to report goodness of fit statistics for the model. These analyses show that body weight is not highly correlated with observable demographic, economic, and contextual outcomes and thus model fits are usually poor.\textsuperscript{12} Lack of a good fit does not mean the model is not useful, but it does pose problems for causal inference. When the analytic goal is to estimate determinants of body weight, these low correlations directly imply the endeavor has not been highly successful, as by definition most of the variation in body weight has been left unexplained. When the goal of the analysis is to assess the consequences of body weight, the paucity of strong predictors of body weight undercuts efforts to use instrumental variables and similar techniques, as discussed in detail in the following subsection.

To illustrate the problem, in Table 1 we display the coefficient of determination, $R^2$, or the proportion of variation explained, from selected recent studies using large survey datasets. In each study the dependent variable is BMI; the covariates differ across studies but all include detailed demographic information. The proportion of variation explained by observed characteristics is commonly less than 10\% and ranges down to 1\%. The proportion of variation explained is modestly high only in studies in which measures of parental (such as Chou et al. (2008)) or peer (Fletcher et al. (2009)) body weight outcomes are included as covariates, suggesting genetic or common household characteristics and sorting on friendship networks have good explanatory power.

\textsuperscript{12}In some studies, individual or regional fixed effects provide high goodness of fit, but this explanatory power merely means there is variation across people and places not attributable to observable characteristics. Goodness of fit can also be reasonable to good when detailed measures of food consumption and physical activity are included, but these proximate causes of body weight are themselves difficult to explain.
Table 1: Coefficients of determination from selected BMI regressions.

<table>
<thead>
<tr>
<th>Study</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chou et al. (2004)</td>
<td>0.08</td>
</tr>
<tr>
<td>Rashad et al. (2006)</td>
<td>0.10 – 0.13</td>
</tr>
<tr>
<td>Chou et al. (2008)</td>
<td>0.15 – 0.24</td>
</tr>
<tr>
<td>Lakdawalla and Philipson (2009)</td>
<td>0.09 – 0.12</td>
</tr>
<tr>
<td>Bhattacharya et al. (2009)</td>
<td>0.01</td>
</tr>
<tr>
<td>Davis and Carpenter (2009)</td>
<td>0.10</td>
</tr>
<tr>
<td>Fletcher et al. (2009)</td>
<td>0.03</td>
</tr>
<tr>
<td>Zhao and Kaestner (2009)</td>
<td>0.13</td>
</tr>
<tr>
<td>Cawley et al. (2005)</td>
<td>0.03</td>
</tr>
<tr>
<td>Auld and Powell (2009)</td>
<td>0.05 – 0.07</td>
</tr>
</tbody>
</table>

To the best of our knowledge, the problem of poor fit in body weight models has been little-discussed in the primary literature, yet it is pervasive, reflects our difficulty in explaining the distribution of body weight, and leads to difficulties determining the consequences of body weight when using observational data.

### 3.3 Finding good instruments.

Instrumental variables estimators based on the intuition provided by equation (3) require the researcher to specify a causal model in which some variables affect $y$ only because they affect $x$. Such models are sometimes able to produce compelling estimates of causation from observational data. However, the major challenge in specifying such models is finding variables which have a strong effect on the problematic explanatory variable $x$ and at the same time only affect the outcome $y$ because they affect $x$.

If the instrumental variable has a low correlation with $x$, the instrument is said to be *weak* and many difficult inference problems arise. In an influential paper, Bound et al. (1995) showed that fake instruments produced by a random number generated yielded estimates of the causal effect of schooling on wages similar to estimates produced by using quarter of birth as instruments, which are weak predictors of schooling.
If the instrumental variable is correlated with $x$ but also correlated with other causes of $y$, the instrument is said to be invalid. Put another way, the instrument is subject to the same problem as the problematic explanatory variable $x$—it is correlated with causes of $y$ unobserved by the researcher. Equation (3) shows that the resulting estimator will not recover the true causal effect of interest no matter how large the sample, because $\text{Cov}(z, u) \neq 0$. This problem arises even when the instrument is not weak, but recent literature has emphasized that when an instrumental variables estimate is based on both weak and invalid instruments the resulting estimates may be very misleading. The point estimate may be centered on a value even farther from the true value than the linear regression estimator; the sampling distribution of the estimator may be much more dispersed than the linear regression estimator, and the standard errors conventionally calculated for the instrumental variables estimate may not reflect the true sampling distribution (Stock et al., 2002).

These problems with instrumental variables strategies are severe in the context of obesity research. First consider attempting to estimate the causal effect of body weight outcomes on other outcomes, so that $x$ is some body weight outcome and $y$ is a result of body weight of interest. The researcher needs to find at least one variable $z$ which affects body weight but has no effect on $y$ except through its effect on $x$. Further, the effect of the candidate instrument $z$ on $x$ must be substantial, not just statistically different from zero, to avoid a weak instrument problem. But as detailed in the preceding subsection of this chapter, obesity researchers have trouble finding variables that are strongly correlated with body weight, much less variables which can be credibly thought to affect body weight but not directly the outcome of interest. Many seemingly promising instruments fail once scrutinized closely: for example, a researcher attempting to estimate the effect of child obesity on academic performance may consider parental education as instruments, reasoning that parental education is correlated with child obesity but does
not directly affect child academic performance. However, parental education does not affect child academic performance only because of an effect on child body weight—the child’s academic performance may be affected by genetic, parental inputs, and contextual factors which are all correlated with parental education even after holding child body weight fixed and which cannot all be observed by the researcher. So parental education is probably an invalid instrument. In light of these difficulties, attempts to use instrumental variables to estimate the causal effect of body weight on other outcomes are rare.\(^\text{13}\)

These problems also arise when the outcome of interest, \(y\), is a body weight outcome and researchers attempt to find instruments for suspected causes of body weight. To illustrate by example, social and medical scientists have both paid considerable attention to the association between income and body weight.\(^\text{14}\) It is well-known that low income is associated with higher obesity rates, that is, that a regression of the form of equation (2) of obesity on household income produces a negative estimate of \(\beta\). However, it is plausible that unobserved causes of body weight \(u\) are correlated with income and that body weight may “reverse” cause household income through its effects on labor market outcomes and marital outcomes. To solve these difficulties, researchers need to specify variables which are correlated with income but only affect body weight because they affect income, which rules out any number of personal or household characteristics, since these characteristics (for example, education) can be expected to affect both body weight and income. Schmeiser (2008) attempts to get around these issues by exploiting an experiment induced by changes in tax rates: he uses changes in the Earned Income Tax Credit as an instrumental variable for income of households affected by these changes, and finds that even though the correlation between income and BMI is negative, income appears to have a positive causal effect on BMI for women. Another example of an

\(^{13}\)See Cawley (2004) for an example of such an attempt.

\(^{14}\)See McLaren in this volume.
instrumental variable estimate of a purported cause of body weight is Dunn (2008). Dunn is interested in estimating the effect of fast food outlet density on body weight, noting that the correlation between these outcomes does not recover a causal effect because fast food restaurants are not randomly assigned across regions. Dunn uses the number of interstate exits as an instrument for fast food outlets and finds that a ten percent increase in fast food outlets causes BMI to rise by 0.33 units.

3.4 Dynamics.

One’s current body weight reflects the sum total of weight changes in previous years. Body weight and composition, in other words, is a “stock” that reflects the “flow” of caloric intake and expenditure in each year going back to birth. This complicates empirical research into the causes of obesity in several ways.

First, a model of individual obesity would need to consider, in addition to contemporaneous causal factors, those factors that operated in previous periods. But data on historical causal factors specific to each individual might not always be available. Even longitudinal data may be insufficient given that these data sets typically do not follow individuals for a long time.

Second, even if historical data on the causal determinants of obesity were available, it may be difficult to incorporate these into a parsimonious empirical model. Suppose, for instance, that one’s weight loss/gain in a given year depends on just 1 contemporaneous variable: the relative price of “healthy vs. “unhealthy food. If an analyst only had access to data on the current weight of a cross section of individuals, then she would need to model the effect of price in each of the previous years of life. In its most general form, the model would contain as many covariates as the number of years of age. Some restrictions would be required to make the empirical model tractable. If one were willing to assume that the impact of price on weight change was the same
in each period, then one could simplify considerably; the model would have only one covariate, the sum total of previous prices. Similarly, if the only price variation was cross-sectional (i.e., individuals face different prices but these prices do not vary over time), then individual weight must reflect its long-run steady state value. Or if the analyst had data on individuals’ weight changes, and she was again willing to assume that the effect of price on weight change was time-invariant, then the model would again have just one covariate: current price.

In reality, of course, weight change is affected by numerous factors. Economics emphasizes that net caloric intake depends on the relative prices of the entire array of food and beverages available to the consumer, as well as the (implicit) price of physical activity and income. The consumer reacts to dozens of relative prices, and this makes it difficult to write down a reasonably parsimonious model. And, again, this exercise requires accurate measurements on a wide range of prices. The exclusion from the model of prices on some commodities leads to inconsistent estimates of the effects on weight change of prices that are included in the model, if these prices are correlated with the excluded prices. Consistent estimation also requires that prices be determined independently of the factors that determine obesity. And economic variables are only part of the story. Social interactions, for instance, are likely important as well.

An additional challenge in this line of research is that very small changes in behaviour can produce large changes in weight over time. Consider, for instance, a 25-year-old male with a BMI of 24 (in the “normal range) who, owing to a permanent change in relative prices, elects to consume an additional 155-calorie can of soda each week. Assuming no compensatory changes in net caloric intake, this modest increase in soda consumption will render him overweight by age 28 and obese by age 43. So while the magnitude of price change on weight gain is small in the short run, the long run effect can be large, only because the small impact accumulates over many periods. The difficulty for the
analyst is that one's data may not be sufficiently informative to precisely estimate the effects of price on weight change, if these effects are small in magnitude. There are a number of reasons for this. One is that one needs more data to distinguish a small effect than a large effect. Another reason is that the precision with which one can estimate the impact of a particular explanatory variable on the outcome (say soda prices on BMI) depends on how well the model fits the data. But, as we have already noted, models of weight outcomes typically don't fit the data well. Estimator precision also requires variation in the explanatory variable. But price variation may not be particularly large during the time period of study. A final reason is that weight gain/loss is often measured with less precision than weight itself. Technically, the variance of the change in weight is twice the variance of weight plus the covariance of the two weight measurements. These dynamic issues interact with the measurement error problem discussed above since they imply that measurement errors may not be white noise but rather serially correlated within individuals, and such correlation leads to even more bias and inconsistency than white noise error (Griliches and Hausman, 1986).

The discussion so far has focused on dynamic aspects of causal models of obesity. The same issues, however, can also apply to models of the consequences of obesity. Models of the health outcomes of obesity are a good case in point. The adverse sequelae of obesity—type 2 diabetes and heart disease, for instance—typically occur only after a long lag. This means that the analyst modeling the morbidity impact of obesity would need to record not only current obesity (and other risk factors), but obesity and other risks factors in previous years as well.

Given these difficulties, some analysts have attempted to identify causal effects using indirect approaches. As an example, Alston et al. (2008) questioned whether farm subsidies in the US have caused higher obesity rates by making fattening foods relatively cheap and plentiful. They note for subsidies to have affected obesity, it must be the case
that (i) farm subsidies have reduced the price of inputs used to making fattening foods, (ii) these lower input prices have reduced the prices consumers pay for such foods, and (iii) in response to lower prices, consumers have consumed more fattening food. They focus on each link in the causal chain and find that “the magnitude of the impact in each case is zero or small. They conclude that, contrary to some media reports, US farm subsidies have had at best a very modest effect on obesity rates.

4 Conclusions.

Causal inference in quantitative social science is challenging because controlled experiments are almost never available, and body weight is an outcome which cannot be experimentally manipulated. Body weight is an outcome of complex genetic, personality, demographic, and contextual influences, so that correlations between weight and most observable personal, family, or regional characteristics do not typically recover causation even after regression adjustment.

This chapter sketched common empirical strategies used to make estimate causal relations when treatments are not randomized with respect to outcomes and highlighted particularly difficult issues with their application in obesity research. In particular, (1) body weight is difficult to measure, in both conceptual and practical senses, (2) body weight is difficult to explain, making the search for identification through instrumental variables even more difficult than usual and undermining efforts to characterize the causes of body weight itself, (3) body weight is a stock which changes slowly over time, such that small influences on body weight over time may eventually cause large changes in weight but will be very difficult to detect statistically. These issues often arise in combination, making causal inference in this area particularly difficult.
References


