Chapter 3

Making Regression Make Sense

'Let us think the unthinkable, let us do the undoable.

Let us prepare to grapple with the ineffable itself,

and see if we may not eff it after all.'

Douglas Adams, *Dirk Gently's Holistic Detective Agency* (1990)

Angrist recounts:

I ran my first regression in the summer of 1979 between my freshman and sophomore years as a student at Oberlin College. I was working as a research assistant for Allan Meltzer and Scott Richard, faculty members at Carnegie-Mellon University, near my house in Pittsburgh. I was still mostly interested in a career in special education, and had planned to go back to work as an orderly in a state mental hospital, my previous summer job. But Econ 101 had got me thinking, and I could also see that at the same wage rate, a research assistant's hours and working conditions were better than those of a hospital orderly. My research assistant duties included data collection and regression analysis, though I did not understand regression or even statistics at the time.

The paper I was working on that summer (Meltzer and Richard, 1983), is an attempt to link the size of governments in democracies, measured as government expenditure over GDP, to income inequality. Most income distributions have a long right tail, which means that average income tends to be way above the median. When inequality grows, more voters find themselves with below-average incomes. Annoyed by this, those with incomes between the median and the average may join those with incomes below the median in voting for fiscal policies which - following Robin Hood - take from the rich and give to the poor. The size of government consequently increases.

I absorbed the basic theory behind the Meltzer and Richards project, though I didn't find it

all that plausible, since voter turnout is low for the poor. I also remember arguing with Alan Meltzer over whether government expenditure on education should be classified as a public good (something that benefits everyone in society as well as those directly affected) or a private good publicly supplied, and therefore a form of redistribution like welfare. You might say this project marked the beginning of my interest in the social returns to education, a topic I went back to with more enthusiasm and understanding in Acemoglu and Angrist (2000).

Today, I understand the Meltzer and Richard (1983) study as an attempt to use regression to uncover and quantify an interesting causal relation. At the time, however, I was purely a regression mechanic. Sometimes I found the RA work depressing. Days would go by where I didn't talk to anybody but my bosses and the occasional Carnegie-Mellon Ph.D. student, most of whom spoke little English anyway. The best part of the job was lunch with Alan Meltzer, a distinguished scholar and a patient and good-natured supervisor, who was happy to chat while we ate the contents of our brown-bags (this did not take long as Allan ate little and I ate fast). I remember asking Allan whether he found it satisfying to spend his days perusing regression output, which then came on reams of double-wide green-bar paper. Meltzer laughed and said there was nothing he would rather be doing.

Now, we too spend our days (at least, the good ones) happily perusing regression output, in the manner of our teachers and advisors in college and graduate school. This chapter explains why.

3.1   Regression Fundamentals

The end of the previous chapter introduces regression models as a computational device for the estimation of treatment-control differences in an experiment, with and without covariates. Because the regressor of interest in the class size study discussed in Section 2.3 was randomly assigned, the resulting estimates have a causal interpretation. In most cases, however, regression is used with observational data. Without the benefit of random assignment, regression estimates may or may not have a causal interpretation. We return to the central question of what makes a regression causal later in this chapter.

Setting aside the relatively abstract causality problem for the moment, we start with the mechanical properties of regression estimates. These are universal features of the population regression vector and its sample analog that have nothing to do with a researcher's interpretation of his output. This chapter begins by reviewing these properties, which include:

(i) the intimate connection between the population regression function and the conditional expectation function

(ii) how and why regression coefficients change as covariates are added or removed from the model

(iii) the close link between regression and other "control strategies" such as matching

(iv) the sampling distribution of regression estimates

3.1.1   Economic Relationships and the Conditional Expectation Function

Empirical economic research in our field of Labor Economics is typically concerned with the statistical analysis of individual economic circumstances, and especially differences between people that might account for differences in their economic fortunes. Such differences in economic fortune are notoriously hard to explain; they are, in a word, random. As applied econometricians, however, we believe we can summarize and interpret randomness in a useful way. An example of "systematic randomness" mentioned in the introduction is the connection between education and earnings. On average, people with more schooling earn more than people with less schooling. The connection between schooling and average earnings has considerable predictive power, in spite of the enormous variation in individual circumstances that sometimes clouds this fact. Of course, the fact that more educated people earn more than less educated people does not mean that schooling *causes* earnings to increase. The question of whether the earnings-schooling relationship is causal is of enormous importance, and we will come back to it many times. Even without resolving the difficult question of causality, however, it's clear that education predicts earnings in a narrow statistical sense. This predictive power is compellingly summarized by the conditional expectation function (CEF).

The CEF for a dependent variable, Y_i given a $K \times 1$ vector of covariates, X_i (with elements x_{ki}) is the expectation, or population average of Y_i with X_i held fixed. The population average can be thought of as the mean in an infinitely large sample, or the average in a completely enumerated finite population. The CEF is written $E[Y_i|X_i]$ and is a function of X_i. Because X_i is random, the CEF is random, though sometimes we work with a particular value of the CEF, say $E[Y_i|X_i{=}42]$, assuming 42 is a possible value for X_i. In Chapter 2, we briefly considered the CEF $E[Y_i|D_i]$, where D_i is a zero-one variable. This CEF takes on two values, $E[Y_i|D_i = 1]$ and $E[Y_i|D_i = 0]$. Although this special case is important, we are most often interested in CEFs that are functions of many variables, conveniently subsumed in the vector, X_i. For a specific value of X_i, say $X_i = x$, we write $E[Y_i|X_i = x]$. For continuous Y_i with conditional density $f_y(\cdot|X_i = x)$, the CEF is

$$E[Y_i|X_i = x] = \int t f_y(t|X_i = x)\, dt.$$

If Y_i is discrete, $E[Y_i|X_i = x]$ equals the sum $\sum_t t f_y(t|X_i = x)$.

Expectation is a population concept. In practice, data usually come in the form of samples and rarely consist of an entire population. We therefore use samples to make inferences about the population. For example, the sample CEF is used to learn about the population CEF. This is always necessary but we postpone a discussion of the formal inference step taking us from sample to population until Section 3.1.3. Our "population first" approach to econometrics is motivated by the fact that we must define the objects of

interest before we can use data to study them.[1]

Figure 3.1.1 plots the CEF of log weekly wages given schooling for a sample of middle-aged white men from the 1980 Census. The distribution of earnings is also plotted for a few key values: 4, 8, 12, and 16 years of schooling. The CEF in the figure captures the fact that—the enormous variation individual circumstances notwithstanding—people with more schooling generally earn more, on average. The average earnings gain associated with a year of schooling is typically about 10 percent.
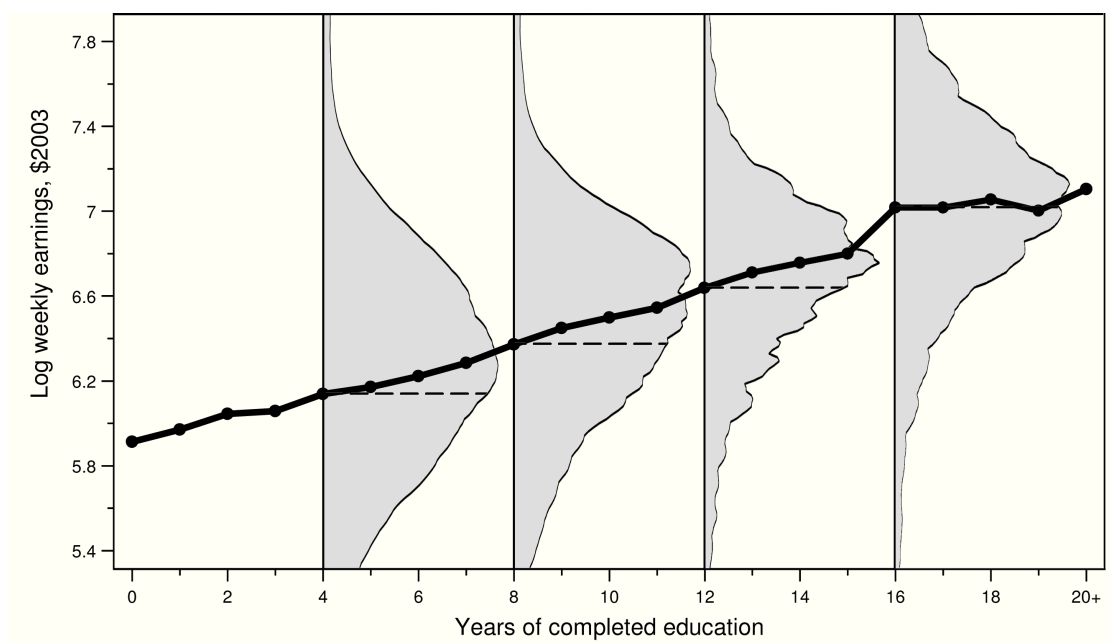

Figure 3.1.1:   Raw data and the CEF of average log weekly wages given schooling. The sample includes white men aged 40-49 in the 1980 IPUMS 5 percent file.

An important complement to the CEF is the law of iterated expectations. This law says that an unconditional expectation can be written as the population average of the CEF. In other words

$$E\left[Y_i\right] = E\{E\left[Y_i|X_i\right]\},\tag{3.1.1}$$

where the outer expectation uses the distribution of X_i. Here is proof of the law of iterated expectations for continuously distributed (X_i, Y_i) with joint density $f_{xy}(u, t)$, where $f_y(t|X_i = x)$ is the conditional

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[1]Examples of pedagogical writing using the "population-first" approach to econometrics include Chamberlain (1984), Goldberger (1991), and Manski (1991).

distribution of Y_i given $X_i = x$ and $g_y(t)$ and $g_x(u)$ are the marginal densities:

$$
\begin{aligned}
E\{E\left[Y_i|X_i\right]\} &= \int E\left[Y_i|X_i = u\right]g_x(u)du \\
&= \int \left[\int tf_y\left(t|X_i = u\right)dt\right]g_x(u)du \\
&= \int \int tf_y\left(t|X_i = u\right)g_x(u)dudt \\
&= \int t \left[\int f_y\left(t|X_i = u\right)g_x(u)du\right]dt = \int t \left[\int f_{xy}\left(u,t\right)du\right]dt \\
&= \int tg_y(t)dt.
\end{aligned}
$$

The integrals in this derivation run over the possible values of X_i and Y_i (indexed by u and t). We've laid out these steps because the CEF and its properties are central to the rest of this chapter.

The power of the law of iterated expectations comes from the way it breaks a random variable into two pieces.

Theorem 3.1.1 *The CEF-Decomposition Property*

$$
Y_i = E\left[Y_i|X_i\right] + \varepsilon_i,
$$

where (i) ε_i is mean-independent of X_i, i.e., $E[\varepsilon_i|X_i] = 0$, and, therefore, (ii) ε_i is uncorrelated with any function of X_i.

Proof. (i) $E[\varepsilon_i|X_i] = E[Y_i - E\left[Y_i|X_i\right] \mid X_i] = E\left[Y_i|X_i\right] - E\left[Y_i|X_i\right] = 0$;(ii) This follows from (i): Let $h(X_i)$ be any function of X_i. By the law of iterated expectations, $E[h(X_i)\varepsilon_i] = E\{h(X_i)E[\varepsilon_i|X_i]\}$ and by mean-independence, $E[\varepsilon_i|X_i] = 0$. ∎

This theorem says that any random variable, Y_i, can be decomposed into a piece that's "explained by X_i", i.e., the CEF, and a piece left over which is orthogonal to (i.e., uncorrelated with) any function of X_i.

The CEF is a good summary of the relationship between Y_i and X_i for a number of reasons. First, we are used to thinking of averages as providing a representative value for a random variable. More formally, the CEF is the best predictor of Y_i given X_i in the sense that it solves a Minimum Mean Squared Error (MMSE) prediction problem. This CEF-prediction property is a consequence of the CEF-decomposition property:

Theorem 3.1.2 *The CEF-Prediction Property.*

Let $m\left(X_i\right)$ be any function of X_i. The CEF solves

$$
E\left[Y_i|X_i\right] = \underset{m(X_i)}{\arg\min} E\left[\left(Y_i - m\left(X_i\right)\right)^2\right],
$$

so it is the MMSE predictor of Y_i given X_i.

Proof. Write

$$
\begin{aligned}
(Y_i - m(X_i))^2 &= ((Y_i - E[Y_i|X_i]) + (E[Y_i|X_i] - m(X_i)))^2 \\
&= (Y_i - E[Y_i|X_i])^2 + 2(E[Y_i|X_i] - m(X_i))(Y_i - E[Y_i|X_i]) \\
&\quad + (E[Y_i|X_i] - m(X_i))^2
\end{aligned}
$$

The first term doesn't matter because it doesn't involve $m(X_i)$. The second term can be written $h(X_i)\varepsilon_i$, where $h(X_i) \equiv 2(E[Y_i|X_i] - m(X_i))$, and therefore has expectation zero by the CEF-decomposition property. The last term is minimized at zero when $m(X_i)$ is the CEF. ∎

A final property of the CEF, closely related to both the CEF decomposition and prediction properties, is the Analysis-of-Variance (ANOVA) Theorem:

Theorem 3.1.3 *The ANOVA Theorem*

$$
V(Y_i) = V(E[Y_i|X_i]) + E[V(Y_i|X_i)]
$$

where $V(\cdot)$ denotes variance and $V(Y_i|X_i)$ is the conditional variance of Y_i given X_i.

Proof. The CEF-decomposition property implies the variance of Y_i is the variance of the CEF plus the variance of the residual, $\varepsilon_i \equiv Y_i - E[Y_i|X_i]$ since ε_i and $E[Y_i|X_i]$ are uncorrelated. The variance of ε_i is

$$
E[\varepsilon_i^2] = E[E[\varepsilon_i^2|X_i]] = E[V[Y_i|X_i]]
$$

where $E[\varepsilon_i^2|X_i] = V[Y_i|X_i]$ because $\varepsilon_i \equiv Y_i - E[Y_i|X_i]$. ∎

The two CEF properties and the ANOVA theorem may have a familiar ring. You might be used to seeing an ANOVA table in your regression output, for example. ANOVA is also important in research on inequality where labor economists decompose changes in the income distribution into parts that can be accounted for by changes in worker characteristics and changes in what's left over after accounting for these factors (See, e.g., Autor, Katz, and Kearney, 2005). What may be unfamiliar is the fact that the CEF properties and ANOVA variance decomposition work in the population as well as in samples, and do not turn on the assumption of a linear CEF. In fact, the validity of linear regression as an empirical tool does not turn on linearity either.

3.1.2   Linear Regression and the CEF

So what's the regression you want to run?

In our world, this question or one like it is heard almost every day. Regression estimates provide a valuable baseline for almost all empirical research because regression is tightly linked to the CEF, and the CEF

provides a natural summary of empirical relationships. The link between regression functions – i.e., the best-fitting line generated by minimizing expected squared errors – and the CEF can be explained in at least 3 ways. To lay out these explanations precisely, it helps to be precise about the regression function we have in mind. This chapter is concerned with the vector of *population* regression coefficients, defined as the solution to a population least squares problem. At this point, we are not worried about causality. Rather, we let the K×1 regression coefficient vector β be defined by solving

$$\beta = \arg\min_b E\left[\left(Y_i - X_i'b\right)^2\right]. \tag{3.1.2}$$

Using the first-order condition,

$$E\left[X_i\left(Y_i - X_i'b\right)\right] = 0.$$

the solution for b can be written $\beta = E\left[X_i X_i'\right]^{-1} E\left[X_i Y_i\right]$. Note that by construction, $E\left[X_i\left(Y_i - X_i'\beta\right)\right] = 0$. In other words, the population residual, which we *define* as $Y_i - X_i'\beta = e_i$, is uncorrelated with the regressors, X_i. It bears emphasizing that this error term does not have a life of its own. It owes its existence and meaning to β.

In the simple bivariate case where the regression vector includes only the single regressor, x_i, and a constant, the slope coefficient is $\beta_1 = \frac{Cov(Y_i, x_i)}{V(x_i)}$, and the intercept is $\alpha = E[Y_i] - \beta_1 E[X_i]$. In the multivariate case, i.e., with more than one non-constant regressor, the slope coefficient for the k-th regressor is given below:

REGRESSION ANATOMY

$$\beta_k = \frac{Cov\left(Y_i, \tilde{x}_{ki}\right)}{V\left(\tilde{x}_{ki}\right)}, \tag{3.1.3}$$

where $\tilde{x}_{ki}$ is the residual from a regression of x_{ki} on all the other covariates.

In other words, $E\left[X_i X_i'\right]^{-1} E\left[X_i Y_i\right]$ is the K×1 vector with k-th element $\frac{Cov(Y_i, \tilde{x}_{ki})}{V(\tilde{x}_{ki})}$. This important formula is said to describe the "anatomy of a multivariate regression coefficient" because it reveals much more than the matrix formula $\beta = E\left[X_i X_i'\right]^{-1} E\left[X_i Y_i\right]$. It shows us that each coefficient in a multivariate regression is the bivariate slope coefficient for the corresponding regressor, after "partialling out" all the other variables in the model.

To verify the regression-anatomy formula, substitute

$$Y_i = \beta_0 + \beta_1 x_{1i} + ... + \beta_k x_{ki} + ... + \beta_K x_{Ki} + e_i$$

in the numerator of (3.1.3). Since $\tilde{x}_{ki}$ is a linear combination of the regressors, it is uncorrelated with e_i. Also, since $\tilde{x}_{ki}$ is a residual from a regression on all the other covariates in the model, it must be uncorrelated these covariates. Finally, for the same reason, the covariance of $\tilde{x}_{ki}$ with x_{ki} is just the variance of $\tilde{x}_{ki}$. We

therefore have that $Cov\left(Y_i, \tilde{x}_{ki}\right) = \beta_k V\left(\tilde{x}_{ki}\right)$.[2]

The regression-anatomy formula is probably familiar to you from a regression or statistics course, perhaps with one twist: the regression coefficients defined in this section are not estimators, but rather they are non-stochastic features of the joint distribution of dependent and independent variables. The joint distribution is what you would observe if you had a complete enumeration of the population of interest (or knew the stochastic process generating the data). You probably don't have such information. Still, it's kosher—even desirable—to think about what a set of population parameters might mean, without initially worrying about how to estimate them.

Below we discuss three reasons why the vector of population regression coefficients might be of interest. These reasons can be summarized by saying that you are interested in regression parameters if you are interested in the CEF.

Theorem 3.1.4 *The Linear CEF Theorem (Regression-justification I)*

Suppose the CEF is linear. Then the population regression function is it.

Proof. Suppose $E\left[Y_i|X_i\right] = X_i'\beta^*$ for a K×1 vector of coefficients, β^*. Recall that $E\left[X_i\left(Y_i - E\left[Y_i|X_i\right]\right)\right] = 0$ by the CEF-decomposition property. Substitute using $E\left[Y_i|X_i\right] = X_i'\beta^*$ to find that $\beta^* = E\left[X_iX_i'\right]^{-1}E\left[X_iY_i\right] = \beta$. ■

The linear CEF theorem raises the question of under what circumstances a CEF is linear. The classic scenario is joint Normality, i.e., the vector $(Y_i, x_i')'$ has a multivariate Normal distribution. This is the scenario considered by Galton (1886), father of regression, who was interested in the intergenerational link between Normally distributed traits such as height and intelligence. The Normal case is clearly of limited empirical relevance since regressors and dependent variables are often discrete, while Normal distributions are continuous. Another linearity scenario arises when regression models are saturated. As reviewed in Section 3.1.4, the saturated regression model has a separate parameter for every possible combination of values that the set of regressors can take on. For example a saturated regression model with two dummy covariates includes both covariates (with coefficients known as the main effects) and their product (known as an interaction term). Such models are inherently linear, a point we also discuss in Section 3.1.4.

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[2]The regression-anatomy formula is usually attributed to Frisch and Waugh (1933). You can also do regression anatomy this way:

$$\beta_k = \frac{Cov\left(\tilde{Y}_{ki}, \tilde{x}_{ki}\right)}{V\left(\tilde{x}_{ki}\right)},$$

where $\tilde{Y}_{ki}$ is the residual from a regression of Y_i on every covariate except x_{ki}. This works because the fitted values removed from $\tilde{Y}_{ki}$ are uncorrelated with $\tilde{x}_{ki}$. Often it's useful to plot $\tilde{Y}_{ki}$ against $\tilde{x}_{ki}$; the slope of the least-squares fit in this scatterplot is your estimate of the multivariate β_k, even though the plot is two-dimensional. Note, however, that it's not enough to partial the other covariates out of Y_i only. That is,

$$\frac{Cov\left(\tilde{Y}_{ki}, x_{ki}\right)}{V\left(x_{ki}\right)} = \left[\frac{Cov\left(\tilde{Y}_{ki}, \tilde{x}_{ki}\right)}{V\left(\tilde{x}_{ki}\right)}\right]\left[\frac{V\left(\tilde{x}_{ki}\right)}{V\left(x_{ki}\right)}\right] \neq \beta_k,$$

unless x_{ki} is uncorrelated with the other covariates.

The following two reasons for focusing on regression are relevant when the linear CEF theorem does not apply.

Theorem 3.1.5 *The Best Linear Predictor Theorem (Regression-justification II)*

   The function $X_i'\beta$ *is the best* linear *predictor of* Y_i *given* X_i *in a MMSE sense.*

Proof. $\beta = E[X_i X_i']^{-1} E[X_i Y_i]$ solves the population least squares problem, (3.1.2). $\blacksquare$

   In other words, just as the CEF, $E[Y_i|X_i]$, is the best (i.e., MMSE) predictor of Y_i given X_i in the class of *all* functions of X_i, the population regression function is the best we can do in the class of *linear* functions.

Theorem 3.1.6 *The Regression-CEF Theorem (Regression-justification III)*

   The function $X_i'\beta$ *provides the MMSE linear approximation to* $E[Y_i|X_i]$*, that is,*

$$\beta = \arg\min_b E\{(E[Y_i|X_i] - X_i'b)^2\}. \tag{3.1.4}$$

Proof. Write

$$
\begin{aligned}
\left(Y_i - X_i'b\right)^2 &= \{(Y_i - E[Y_i|X_i]) + (E[Y_i|X_i] - X_i'b)\}^2 \\
&= (Y_i - E[Y_i|X_i])^2 + (E[Y_i|X_i] - X_i'b)^2 \\
&\quad + 2(Y_i - E[Y_i|X_i])(E[Y_i|X_i] - X_i'b).
\end{aligned}
$$

The first term doesn't involve b and the last term has expectation zero by the CEF-decomposition property (ii). The CEF-approximation problem, (3.1.4), therefore has the same solution as the population least squares problem, (3.1.2). $\blacksquare$

   These two theorems show us two more ways to view regression. Regression provides the best linear predictor for the dependent variable in the same way that the CEF is the best unrestricted predictor of the dependent variable. On the other hand, if we prefer to think about approximating $E[Y_i|X_i]$, as opposed to predicting Y_i, the Regression-CEF theorem tells us that even if the CEF is nonlinear, regression provides the best linear approximation to it.

   The regression-CEF theorem is our favorite way to motivate regression. The statement that regression approximates the CEF lines up with our view of empirical work as an effort to describe the essential features of statistical relationships, without necessarily trying to pin them down exactly. The linear CEF theorem is for special cases only. The best linear predictor theorem is satisfyingly general, but it encourages an overly clinical view of empirical research. We're not really interested in predicting *individual* Y_i; it's the *distribution* of Y_i that we care about.

   Figure 3.1.2 illustrates the CEF approximation property for the same schooling CEF plotted in Figure 3.1.1. The regression line fits the somewhat bumpy and nonlinear CEF as if we were estimating a model

for $E[Y_i|X_i]$ instead of a model for Y_i. In fact, that is exactly what's going on.   An implication of the regression-CEF theorem is that regression coefficients can be obtained by using $E[Y_i|X_i]$ as a dependent variable instead of Y_i itself. To see this, suppose that X_i is a discrete random variable with probability mass function, $g_x(u)$ when $X_i = u$.   Then

$$E\{(E[Y_i|X_i] - X_i'b)^2\} = \sum_u (E[Y_i|X_i = u] - u'b)^2 g_x(u).$$

This means that β can be constructed from the weighted least squares regression of $E[Y_i|X_i = u]$ on u, where u runs over the values taken on by X_i.   The weights are given by the distribution of X_i, i.e., $g_x(u)$ when $X_i = u$. Another way to see this is to iterate expectations in the formula for β:

$$\beta = E[X_iX_i']^{-1}E[X_iY_i] = E[X_iX_i']^{-1}E[X_iE(Y_i|X_i)]. \tag{3.1.5}$$

The CEF or grouped-data version of the regression formula is of practical use when working on a project that precludes the analysis of micro data.   For example, Angrist (1998), studies the effect of voluntary military service on earnings later in life.   One of the estimation strategies used in this project regresses civilian earnings on a dummy for veteran status, along with personal characteristics and the variables used by the military to screen soldiers. The earnings data come from the US Social Security system, but Social Security earnings records cannot be released to the public. Instead of individual earnings, Angrist worked with average earnings conditional on race, sex, test scores, education, and veteran status.

An illustration of the grouped-data approach to regression appears below.   We estimated the schooling coefficient in a wage equation using 21 conditional means, the sample CEF of earnings given schooling.   As the Stata output reported here shows, a grouped-data regression, weighted by the number of individuals at each schooling level in the sample, produces coefficients *identical* to what would be obtained using the underlying microdata sample with hundreds of thousands of observations. Note, however, that the standard errors from the grouped regression do not correctly reflect the asymptotic sampling variance of the slope estimate in repeated *micro-data* samples; for that you need an estimate of the variance of $Y_i - X_i'\beta$. This variance depends on the microdata, in particular, the second-moments of $W_i \equiv \begin{bmatrix} Y_i; & X_i' \end{bmatrix}'$, a point we elaborate on in the next section.

3.1.3   Asymptotic OLS Inference

In practice, we don't usually know what the CEF or the population regression vector is. We therefore draw statistical inferences about these quantities using samples.   Statistical inference is what much of traditional econometrics is about. Although this material is covered in any Econometrics text, we don't want to skip the inference step completely. A review of basic asymptotic theory allows us to highlight the important fact that the process of statistical inference is entirely distinct from the question of how a particular set of regression

Sample is limited to white men, age 40-49. Data is from Census IPUMS 1980, 5% sample.
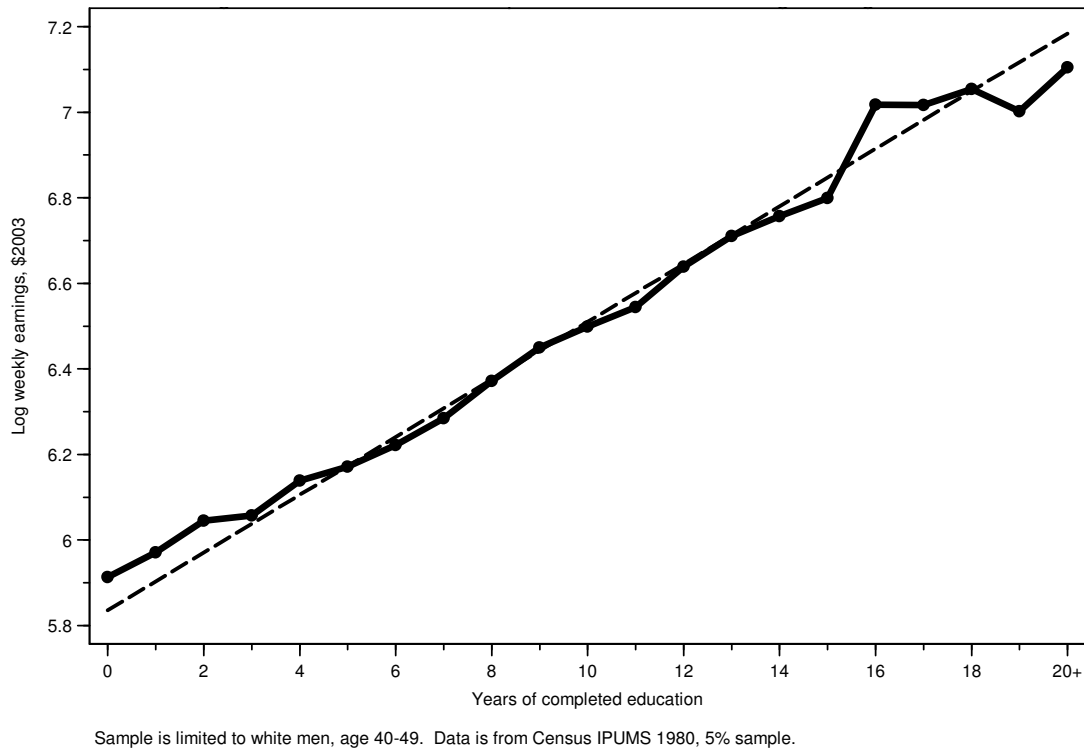
Figure 3.1.2: Regression threads the CEF of average weekly wages given schooling

estimates should be interpreted. Whatever a regression coefficient may mean, it has a sampling distribution that is easy to describe and use for statistical inference.[3]

We are interested in the distribution of the sample analog of

$$\beta = E[\mathrm{X}_i \mathrm{X}_i']^{-1} E[\mathrm{X}_i \mathrm{Y}_i]$$

in repeated samples. Suppose the vector $W_i \equiv \begin{bmatrix} \mathrm{Y}_i; & \mathrm{X}_i' \end{bmatrix}'$ is independently and identically distributed in a sample of size N. A natural estimator of the first population moment, $E[W_i]$, is the sum, $\frac{1}{N}\sum_{i=1}^{N} W_i$. By the law of large numbers, this sample moment gets arbitrarily close to the corresponding population moment as the sample size grows. We might similarly consider higher-order moments of the elements of W_i, e.g., the matrix of second moments, $E[W_i W_i']$, with sample analog $\frac{1}{N}\sum_{i=1}^{N} W_i W_i'$. Following this principle, the method of moments estimator of β replaces each expectation by a sum. This logic leads to the Ordinary Least Squares (OLS) estimator

$$\hat{\beta} = \left[\sum_i \mathrm{X}_i \mathrm{X}_i' \right]^{-1} \sum_i \mathrm{X}_i \mathrm{Y}_i.$$

Although we derived $\hat{\beta}$ as a method of moments estimator, it is called the OLS estimator of β because it solves the sample analog of the least-squares problem described at the beginning of Section 3.1.2.[4]

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[3]The discussion of asymptotic OLS inference in this section is largely a condensation of material in Chamberlain (1984). Important pitfalls and problems with this asymptotic theory are covered in the last chapter.

[4]Econometricians like to use matrices because the notation is so compact. Sometimes (not very often) we do too. Suppose

A - Individual-level data

```
. regress earnings school, robust

      Source |       SS        df        MS          Number of obs =   409435
-------------+------------------------------         F(  1,409433) =49118.25
       Model | 22631.4793        1  22631.4793       Prob > F      =   0.0000
    Residual | 188648.31    409433  .460755019        R-squared     =   0.1071
-------------+------------------------------         Adj R-squared =   0.1071
       Total | 211279.789   409434   .51602893        Root MSE      =  .67879

-------------+----------------------------------------------------------------
             |            Robust                     Old Fashioned
    earnings |    Coef.  Std. Err.      t             Std. Err.        t
-------------+----------------------------------------------------------------
      school |  .0674387  .0003447   195.63           .0003043     221.63
      const. | 5.835761  .0045507  1282.39           .0040043    1457.38
-----------------------------------------------------------------------------
```

B - Means by years of schooling

```
. regress average_earnings school [aweight=count], robust
(sum of wgt is    4.0944e+05)

      Source |       SS        df        MS          Number of obs =       21
-------------+------------------------------         F(  1,    19) =   540.31
       Model | 1.16077332        1  1.16077332       Prob > F      =   0.0000
    Residual | .040818796       19  .002148358        R-squared     =   0.9660
-------------+------------------------------         Adj R-squared =   0.9642
       Total | 1.20159212       20  .060079606        Root MSE      =  .04635

-------------+----------------------------------------------------------------
     average |            Robust                     Old Fashioned
    _earnings |    Coef.  Std. Err.      t             Std. Err.        t
-------------+----------------------------------------------------------------
      school |  .0674387  .0040352    16.71           .0029013      23.24
      const. | 5.835761  .0399452   146.09           .0381792     152.85
-----------------------------------------------------------------------------
```

Figure 3.1.3: Micro-data and grouped-data estimates of returns to schooling. Source: 1980 Census - IPUMS, 5 percent sample. Sample is limited to white men, age 40-49. Derived from Stata regression output. Old-fashioned standard errors are the default reported. Robust standard errors are heteroscedasticity-consistent. Panel A uses individual-level data. Panel B uses earnings averaged by years of schooling.

The asymptotic sampling distribution of $\hat{\beta}$ depends solely on the definition of the estimand (i.e., the nature of the thing we're trying to estimate, β) and the assumption that the data constitute a random sample. Before deriving this distribution, it helps to record the general asymptotic distribution theory that covers our needs. This basic theory can be stated mostly in words. For the purposes of these statements, we assume the reader is familiar with the core terms and concepts of statistical theory (e.g., moments, mathematical expectation, probability limits, and asymptotic distributions). For definitions of these terms and a formal mathematical statement of the theoretical propositions given below, see, e.g., Knight (2000).

THE LAW OF LARGE NUMBERS Sample moments converge in probability to the corresponding population moments. In other words, the probability that the sample mean is close to the population mean can be made as high as you like by taking a large enough sample.

THE CENTRAL LIMIT THEOREM Sample moments are asymptotically Normally distributed (after subtracting the corresponding population moment and multiplying by the square root of the sample size). The covariance matrix is given by the variance of the underlying random variable. In other words, in large enough samples, appropriately normalized sample moments are approximately Normally distributed.

SLUTSKY'S THEOREM

(a) Consider the sum of two random variables, one of which converges in distribution and the other converges in probability to a constant: the asymptotic distribution of this sum is unaffected by replacing the one that converges to a constant by this constant. Formally, let a_N be a statistic with a limiting distribution and let b_N be a statistic with probability limit b. Then $a_N + b_N$ and $a_N + b$ have the same limiting distribution.

(b) Consider the product of two random variables, one of which converges in distribution and the other converges in probability to a constant: the asymptotic distribution of this product is unaffected by replacing the one that converges to a constant by this constant. This allows us to replaces some sample moments by population moments (i.e., by their probability limits) when deriving distributions. Formally, let a_N be a statistic with a limiting distribution and let b_N be a statistic with probability limit b. Then $a_N b_N$ and $a_N b$ have the same asymptotic distribution.

THE CONTINUOUS MAPPING THEOREM Probability limits pass through continuous functions. For example, the probability limit of any continuous function of a sample moment is the function evaluated at the corresponding population moment. Formally, the probability limit of $h(b_N)$ is $h(b)$ where *plim* $b_N = b$ and $h(\cdot)$ is continuous at b.

X is the matrix whose rows are given by X_i' and y is the vector with elements y_i, for $i = 1, ..., N$. The sample moment $\frac{1}{N} \sum X_i X_i'$ is $X'X/N$ and the sample moment $\frac{1}{N} \sum X_i y_i$ is $X'y/N$. Then we can write $\hat{\beta} = (X'X)^{-1} X'y$, a familiar matrix formula.

THE DELTA METHOD Consider a vector-valued random variable that is asymptotically Normally distributed. Most scalar functions of this random variable are also asymptotically Normally distributed, with covariance matrix given by a quadratic form with the covariance matrix of the random variable on the inside and the gradient of the function evaluated at the probability limit of the random variable on the outside. Formally, the asymptotic distribution of $h(b_N)$ is Normal with covariance matrix $\nabla h(b)'\Omega\nabla h(b)$ where $plim\ b_N = b$, $h(\cdot)$ is continuously differentiable at b with gradient $\nabla h(b)$, and b_N has asymptotic covariance matrix Ω.[5]

We can use these results to derive the asymptotic distribution of $\hat{\beta}$ in two ways. A conceptually straightforward but somewhat inelegant approach is to use the delta method: $\hat{\beta}$ is a function of sample moments, and is therefore asymptotically Normally distributed. It remains only to find the covariance matrix of the asymptotic distribution from the gradient of this function. (Note that consistency of $\hat{\beta}$ comes immediately from the continuous mapping theorem). An easier and more instructive derivation uses the Slutsky and central limit theorems. Note first that we can write

$$\mathrm{Y}_i = \mathrm{X}_i'\beta + [\mathrm{Y}_i - \mathrm{X}_i'\beta] \equiv \mathrm{X}_i'\beta + e_i, \tag{3.1.6}$$

where the residual e_i is *defined* as the difference between the dependent variable and the population regression function, as before. This is as good a place as any to point out that these residuals are uncorrelated with the regressors *by definition of* β. In other words, $E[\mathrm{X}_i e_i] = 0$ is a consequence of $\beta = E[\mathrm{X}_i\mathrm{X}_i']^{-1}E[\mathrm{X}_i\mathrm{Y}_i]$ and $e_i = \mathrm{Y}_i - \mathrm{X}_i'\beta$, and not an assumption about an underlying economic relation. We return to this important point in the discussion of causal regression models in Section 3.2.[6]

Substituting the identity 3.1.6 for Y_i in the formula for $\hat{\beta}$, we have

$$\hat{\beta} = \beta + \left[\sum \mathrm{X}_i\mathrm{X}_i'\right]^{-1}\sum \mathrm{X}_i e_i.$$

The asymptotic distribution of $\hat{\beta}$ is the asymptotic distribution of $\sqrt{N}(\hat{\beta}-\beta) = N\left[\sum \mathrm{X}_i\mathrm{X}_i'\right]^{-1}\frac{1}{\sqrt{N}}\sum\mathrm{X}_i e_i$. By the Slutsky theorem, this has the same asymptotic distribution as $E[\mathrm{X}_i\mathrm{X}_i']^{-1}\frac{1}{\sqrt{N}}\sum\mathrm{X}_i e_i$. Since $E[\mathrm{X}_i e_i] = 0$, $\frac{1}{\sqrt{N}}\sum\mathrm{X}_i e_i$ is a root-N-normalized and centered sample moment. By the central limit theorem, this is asymptotically Normally distributed with mean zero and covariance matrix $E[\mathrm{X}_i\mathrm{X}_i'e_i^2]$, since this fourth moment is the covariance matrix of $\mathrm{X}_i e_i$. Therefore, $\hat{\beta}$ has an asymptotic Normal distribution, with probability limit β, and covariance matrix

$$E[\mathrm{X}_i\mathrm{X}_i']^{-1}E[\mathrm{X}_i\mathrm{X}_i'e_i^2]E[\mathrm{X}_i\mathrm{X}_i']^{-1}. \tag{3.1.7}$$

The standard errors used to construct *t*-statistics are the square roots of the diagonal elements of this

---

[5] For a derivation of the the delta method formula using the Slutsky and continuous mapping theorems, see, e.g., Knight, 2000, pp. 120-121.

[6] Residuals defined in this way are not necessarily *mean-independent* of X_i; for mean-independence, we need a linear CEF.

matrix. In practice these standard errors are estimated by substituting sums for expectations, and using the estimated residuals, $\hat{e}_i = \text{Y}_i - \text{X}_i'\hat{\beta}$ to form the empirical fourth moment, $\sum[\text{X}_i\text{X}_i\hat{e}_i^2]/N$.

Asymptotic standard errors computed in this way are known as heteroskedasticity-consistent standard errors, White (1980a) standard errors, or Eicker-White standard errors in recognition of Eicker's (1967) derivation. They are also known as "robust" standard errors (e.g., in Stata). These standard errors are said to be robust because, in large enough samples, they provide accurate hypothesis tests and confidence intervals given minimal assumptions about the data and model. In particular, our derivation of the limiting distribution makes no assumptions other than those needed to ensure that basic statistical results like the central limit theorem go through. These are not, however, the standard errors that you get by default from packaged software. Default standard errors are derived under a homoskedasticity assumption, specifically, that $E[e_i^2|\text{X}_i] = \sigma^2$, a constant. Given this assumption, we have

$$E[\text{X}_i\text{X}_i'e_i^2] = E(\text{X}_i\text{X}_i'E[e_i^2|\text{X}_i]) = \sigma^2 E[\text{X}_i\text{X}_i'],$$

by iterating expectations. The asymptotic covariance matrix of $\hat{\beta}$ then simplifies to

$$
\begin{aligned}
E[\text{X}_i\text{X}_i']^{-1}E[\text{X}_i\text{X}_i'e_i^2]E[\text{X}_i\text{X}_i']^{-1} &= E[\text{X}_i\text{X}_i']^{-1}\sigma^2 E[\text{X}_i\text{X}_i']E[\text{X}_i\text{X}_i]^{-1} \\
&= E[\text{X}_i\text{X}_i']^{-1}\sigma^2.
\end{aligned}
\tag{3.1.8}
$$

The diagonal elements of (3.1.8) are what SAS or Stata report unless you request otherwise.

Our view of regression as an approximation to the CEF makes heteroskedasticity seem natural. If the CEF is nonlinear and you use a linear model to approximate it, then the quality of fit between the regression line and the CEF will vary with X_i. Hence, the residuals will be larger, on average, at values of X_i where the fit is poorer. Even if you are prepared to assumed that the conditional variance of Y_i given X_i is constant, the fact that the CEF is nonlinear means that $E[(\text{Y}_i - \text{X}_i'\beta)^2|\text{X}_i]$ will vary with X_i. To see this, note that, as a rule,

$$
\begin{aligned}
E[(\text{Y}_i - \text{X}_i'\beta)^2|\text{X}_i] &= \tag{3.1.9} \\
E\{[(\text{Y}_i - E[\text{Y}_i|\text{X}_i]) &+ (E[\text{Y}_i|\text{X}_i] - \text{X}_i'\beta)]^2|\text{X}_i\} \\
&= V[\text{Y}_i|\text{X}_i] + (E[\text{Y}_i|\text{X}_i] - \text{X}_i'\beta)^2.
\end{aligned}
$$

Therefore, even if $V[\text{Y}_i|\text{X}_i]$ is constant, the residual variance increases with the square of the gap between the regression line and the CEF, a fact noted in White (1980b).[7]

In the same spirit, it's also worth noting that while a linear CEF makes homoskedasticity possible, this is

---

[7]The cross-product term resulting from an expansion of the quadratic in the middle of 3.1.9 is zero because $\text{Y}_i - E[\text{Y}_i|\text{X}_i]$ is mean-independent of X_i.

not a sufficient condition for homoskedasticity. Our favorite example in this context is the linear probability model (LPM). A linear probability model is any regression where the dependent variable is zero-one, i.e., a dummy variable such as an indicator for labor force participation.   Suppose the regression model is saturated, so the CEF is linear.   Because the CEF is linear, the residual variance is also the conditional variance, $V[Y_i|X_i]$. But the dependent variable is a Bernoulli trial and the variance of a Bernoulli trial is $P[Y_i|X_i](1 - P[Y_i|X_i])$.   We conclude that LPM residuals are necessarily heteroskedastic unless the only regressor is a constant.

These points of principle notwithstanding, as an empirical matter, heteroskedasticity may matter little. In the micro-data schooling regression depicted in Figure 3.1.3, the robust standard error is .0003447, while the old-fashioned standard error is .0003043, only slightly smaller. The standard errors from the grouped-data regression, which are necessarily heteroskedastic if group sizes differ, change somewhat more; compare the .004 robust standard to the .0029 conventional standard error. Based on our experience, these differences are typical. If heteroskedasticity matters too much, say, more than a 30% increase or any marked decrease in standard errors, you should worry about possible programming errors or other problems (for example, robust standard errors below conventional may be a sign of finite-sample bias in the robust calculation; see Chapter 8, below.)

3.1.4   Saturated Models, Main Effects, and Other Regression Talk

We often discuss regression models using terms like *saturated* and *main effects*.   These terms originate in an experimentalist tradition that uses regression to model discrete treatment-type variables.   This language is now used more widely in many fields, however, including applied econometrics.   For readers unfamiliar with these terms, this section provides a brief review.

Saturated regression models are regression models with discrete explanatory variables, where the model includes a separate parameter for all possible values taken on by the explanatory variables. For example, when working with a single explanatory variable indicating whether a worker is a college graduate, the model is saturated by including a single dummy for college graduates and a constant. We can also saturate when the regressor takes on many values.   Suppose, for example, that $S_i = 0, 1, 2, ..., \tau$.   A saturated regression model for S_i is

$$Y_i = \beta_0 + \beta_1 d_{1i} + \beta_2 d_{2i} + ... + \beta_\tau d_{\tau i} + \varepsilon_i,$$

where $d_{ji} = 1[S_i = j]$ is a dummy variable indicating schooling level-j, and β_j is said to be the jth-level schooling *effect*.   Note that

$$\beta_j = E[Y_i|S_i = j] - E[Y_i|S_i = 0],$$

while $\beta_0 = E[Y_i|S_i = 0]$. In practice, you can pick any value of S_i for the reference group; a regression model is saturated as long as it has one parameter for every possible j in $E[Y_i|S_i = j]$. Saturated models fit the

CEF perfectly because the CEF is linear in the dummy regressors used to saturate. This is an important special case of the regression-CEF theorem.

If there are two explanatory variables, say one dummy indicating college graduates and one dummy indicating sex, the model is saturated by including these two dummies, their product, and a constant. The coefficients on the dummies are known as main effects, while the product is called an *interaction term*. This is not the only saturated parameterization; any set of indicators (dummies) that can be used to identify each value taken on by the covariates produces a saturated model. For example, an alternative saturated model includes dummies for male college graduates, male dropouts, female college graduates, and female dropouts, but no intercept.

Here's some notation to make this more concrete. Let x_{1i} indicate college graduates and x_{2i} indicate women. The CEF given x_{1i} and x_{2i} takes on four values:

$$E\left[Y_i|x_{1i}=0, x_{2i}=0\right],$$
$$E\left[Y_i|x_{1i}=1, x_{2i}=0\right],$$
$$E\left[Y_i|x_{1i}=0, x_{2i}=1\right],$$
$$E\left[Y_i|x_{1i}=1, x_{2i}=1\right].$$

We can label these using the following scheme:

$$
\begin{aligned}
E\left[Y_i|x_{1i}=0, x_{2i}=0\right] &= \alpha \\
E\left[Y_i|x_{1i}=1, x_{2i}=0\right] &= \alpha + \beta \\
E\left[Y_i|x_{1i}=0, x_{2i}=1\right] &= \alpha + \gamma \\
E\left[Y_i|x_{1i}=1, x_{2i}=1\right] &= \alpha + \beta + \gamma + \delta.
\end{aligned}
$$

Since there are four Greek letters and the CEF takes on four values, this parameterization does not restrict the CEF. It can be written in terms of Greek letters as

$$E[Y_i|x_{1i}, x_{2i}] = \alpha + \beta x_{1i} + \gamma x_{2i} + \delta(x_{1i}x_{2i}),$$

a parameterization with two main effects and one interaction term.[8] The saturated regression equation becomes

$$Y_i = \alpha + \beta x_{1i} + \gamma x_{2i} + \delta(x_{1i}x_{2i}) + \varepsilon_i.$$

Finally, we can combine the multi-valued schooling variable with sex to produce a saturated model that

---

[8] With a third dummy variable in the model, say x_{3i}, a saturated model includes 3 main effects, 3 second-order interaction terms $\{x_{1i}x_{2i}, x_{2i}x_{3i}, x_{1i}x_{2i}\}$ and one third-order term, $x_{1i}x_{2i}x_{3i}$.

has τ main effects for schooling, one main effect for sex, and τ sex-schooling interactions:

$$Y_i = \beta_0 + \sum_{j=1}^{\tau} \beta_j d_{ji} + \gamma x_{2i} + \sum_{j=1}^{\tau} \delta_j (d_{ji} x_{2i}) + \varepsilon_i. \tag{3.1.10}$$

The interaction terms, δ_j, tell us how each of the schooling effects differ by sex. The CEF in this case takes on $2(\tau + 1)$ values while the regression has this many parameters.

Note that there is a natural hierarchy of modeling strategies with saturated models at the top. It's natural to start with a saturated model because this fits the CEF. On the other hand, saturated models generate a lot of interaction terms, many of which may be uninteresting or imprecise. You might therefore sensibly choose to omit some or all of these. Equation (3.1.10) without interaction terms approximates the CEF with a purely additive model for schooling and sex. This is a good approximation if the returns to college are similar for men and women. And, in any case, schooling coefficients in the additive specification give a (weighted) average return across both sexes, as discussed in Section 3.3.1, below. On the other hand, it would be strange to estimate a model which included interaction terms but omitted the corresponding main effects. In the case of schooling, this would be something like

$$Y_i = \beta_0 + \gamma x_{2i} + \sum_{j=1}^{\tau} \delta_j (d_{ji} x_{2i}) + \varepsilon_i. \tag{3.1.11}$$

This model allows schooling to shift wages only for women, something very far from the truth. Consequently, the results of estimating (3.1.11) are likely to be hard to interpret.

Finally, it's important to recognize that a saturated model fits the CEF perfectly regardless of the distribution of Y_i. For example, this is true for linear probability models and other limited dependent variable models (e.g., non-negative Y_i), a point we return to at the end of this chapter.

3.2   Regression and Causality

Section 3.1.2 shows how regression gives the best (MMSE) linear approximation to the CEF. This understanding, however, does not help us with the deeper question of when regression has a causal interpretation. When can we think of a regression coefficient as approximating the causal effect that might be revealed in an experiment?

3.2.1   The Conditional Independence Assumption

A regression is causal when the CEF it approximates is causal. This doesn't answer the question, of course. It just passes the buck up one level, since, as we've seen, a regression inherits it's legitimacy from a CEF. Causality means different things to different people, but researchers working in many disciplines have found it useful to think of causal relationships in terms of the potential outcomes notation used in Chapter 2 to

describe what would happen to a given individual in a hypothetical comparison of alternative hospitalization scenarios. Differences in these potential outcomes were said to be the causal effect of hospitalization. The CEF is causal when it describes differences in average potential outcomes for a fixed reference population.

It's easiest to expand on the somewhat murky notion of a causal CEF in the context of a particular question, so let's stick with the schooling example. The causal connection between schooling and earnings can be defined as the functional relationship that describes what a given individual would earn if he or she obtained different levels of education. In particular, we might think of schooling decisions as being made in a series of episodes where the decision-maker might realistically go one way or another, even if certain choices are more likely than others. For example, in the middle of junior year, restless and unhappy, Angrist glumly considered his options: dropping out of high school and hopefully getting a job, staying in school but taking easy classes that lead to a quick and dirty high school diploma, or plowing on in an academic track that leads to college. Although the consequences of such choices are usually unknown in advance, the idea of alternative paths leading to alternative outcomes for a given individual seems uncontroversial. Philosophers have argued over whether this personal notion of potential outcomes is precise enough to be scientifically useful, but individual decision-makers seem to have no trouble thinking about their lives and choices in this manner (as in Robert Frost's celebrated *The Road Not Taken*: the traveller-narrator sees himself looking back on a moment of choice. He believes that the decision to follow the road less traveled "has made all the difference," though he also recognizes that counterfactual outcomes are unknowable).

In empirical work, the causal relationship between schooling and earnings tells us what people would earn—on average—if we could either change their schooling in a perfectly-controlled environment, or change their schooling randomly so that those with different levels of schooling would be otherwise comparable. As we discussed in Chapter 2, experiments ensure that the causal variable of interest is independent of potential outcomes so that the groups being compared are truly comparable. Here, we would like to generalize this notion to causal variables that take on more than two values, and to more complicated situations where we must hold a variety of "control variables" fixed for causal inferences to be valid. This leads to the *conditional independence assumption* (CIA), a core assumption that provides the (sometimes implicit) justification for the causal interpretation of regression. This assumption is sometimes called selection-on-observables because the covariates to be held fixed are assumed to be known and observed (e.g., in Goldberger, 1972; Barnow, Cain, and Goldberger, 1981). The big question, therefore, is what these control variables are, or should be. We'll say more about that shortly. For now, we just do the econometric thing and call the covariates "X_i". As far as the schooling problem goes, it seems natural to imagine that X_i is a vector that includes measures of ability and family background.

For starters, think of schooling as a binary decision, like whether Angrist goes to college. Denote this by a dummy variable, c_i. The causal relationship between college attendance and a future outcome like earnings can be described using the same potential-outcomes notation we used to describe experiments in

Chapter 2. To address this question, we imagine two potential earnings variables:

$$\textit{potential outcome} = \begin{cases} Y_{1i} & \text{if } C_i = 1 \\ Y_{0i} & \text{if } C_i = 0 \end{cases}.$$

In this case, Y_{0i} is i's earnings without college, while Y_{1i} is i's earnings if he goes. We would like to know the difference between Y_{1i} and Y_{0i}, which is the causal effect of college attendance on individual i. This is what we would measure if we could go back in time and nudge i onto the road not taken. The observed outcome, Y_i, can be written in terms of potential outcomes as

$$Y_i = Y_{0i} + (Y_{1i} - Y_{0i})C_i.$$

We get to see one of Y_{1i} or Y_{0i}, but never both. We therefore hope to measure the average of $Y_{1i} - Y_{0i}$, or the average for some group, such as those who went to college. This is $E[Y_{1i} - Y_{0i}|C_i = 1]$.

In general, comparisons of those who do and don't go to college are likely to be a poor measure of the causal effect of college attendance. Following the logic in Chapter 2, we have

$$\underbrace{E\left[Y_i|C_i = 1\right] - E[Y_i|C_i = 0]}_{\text{Observed difference in earnings}} = \underbrace{E[Y_{1i} - Y_{0i}|C_i = 1]}_{\text{average treatment effect on the treated}} \qquad (3.2.1)$$
$$+ \underbrace{E\left[Y_{0i}|C_i = 1\right] - E\left[Y_{0i}|C_i = 0\right]}_{\text{selection bias}}.$$

It seems likely that those who go to college would have earned more anyway. If so, selection bias is positive, and the naive comparison, $E\left[Y_i|C_i = 1\right] - E[Y_i|C_i = 0]$, exaggerates the benefits of college attendance.

The CIA asserts that conditional on observed characteristics, X_i, selection bias disappears. In this example, the CIA says,

$$\{Y_{0i}, Y_{1i}\} \amalg C_i|X_i. \qquad (3.2.2)$$

Given the CIA, conditional-on-X_i comparisons of average earnings across schooling levels have a causal interpretation. In other words,

$$E\left[Y_i|X_i, C_i = 1\right] - E\left[Y_i|X_i, C_i = 0\right] = E[Y_{1i} - Y_{0i}|X_i].$$

Now, we'd like to expand the conditional independence assumption to causal relations that involve variables that can take on more than two values, like years of schooling, s_i. The causal relationship between schooling and earnings is likely to be different for each person. We therefore use the individual-specific notation,

$$Y_{si} \equiv f_i(s)$$

to denote the potential earnings that person i would receive after obtaining s years of education. If s takes on only two values, 12 and 16, then we are back to the college/no college example:

$$Y_{0i} = f_i(12); Y_{1i} = f_i(16).$$

More generally, the function $f_i(s)$ tells us what i would earn for *any* value of schooling, s. In other words, $f_i(s)$ answers causal "what if" questions. In the context of theoretical models of the relationship between human capital and earnings, the form of $f_i(s)$ may be determined by aspects of individual behavior and/or market forces.

The CIA in this more general setup becomes

$$Y_{si} \amalg S_i | X_i \qquad \text{(CIA)}$$

In many randomized experiments, the CIA crops up because S_i *is* randomly assigned conditional on X_i (In the Tennessee STAR experiment, for example, small classes were randomly assigned within schools). In an observational study, the CIA means that S_i can be said to be "as good as randomly assigned," conditional on X_i.

Conditional on X_i, the *average causal effect* of a one year increase in schooling is $E[f_i(s) - f_i(s-1)|X_i]$, while the average causal effect of a 4-year increase in schooling is $E[f_i(s) - E[f_i(s-4)]|X_i]$. The data reveal only $Y_i = f_i(S_i)$, however, that is $f_i(s)$ for $s = S_i$. But given the CIA, conditional-on-X_i comparisons of average earnings across schooling levels have a causal interpretation. In other words,

$$E[Y_i|X_i, S_i = s] - E[Y_i|X_i, S_i = s-1]$$
$$= E[f_i(s) - f_i(s-1)|X_i]$$

for any value of s. For example, we can compare the earnings of those with 12 and 11 years of schooling to learn about the average causal effect of high school graduation:

$$E[Y_i|X_i, S_i = 12] - E[Y_i|X_i, S_i = 11] = E[f_i(12)|X_i, S_i = 12] - E[f_i(11)|X_i, S_i = 11].$$

This comparison has a causal interpretation because, given the CIA,

$$E[f_i(12)|X_i, S_i = 12] - E[f_i(11)|X_i, S_i = 11] = E[f_i(12) - f_i(11)|X_i, S_i = 12].$$

Here, the selection bias term is the average difference in the potential dropout-earnings of high school graduates and dropouts. Given the CIA, however, high school graduation is independent of potential earnings conditional on X_i, so the selection-bias vanishes. Note also that in this case, the causal effect of

graduating high school on high school graduates is the population average high school graduation effect:

$$E\left[f_i(12) - f_i(11)|X_i, s_i = 12\right] = E\left[f_i(12) - f_i(11)|X_i\right].$$

This is important . . . but less important than the elimination of selection bias in (3.2.1).

So far, we have constructed separate causal effects for each value taken on by the conditioning variable, X_i. This leads to as many causal effects as there are values of X_i, an embarrassment of riches. Empiricists almost always find it useful to boil a set of estimates down to a single summary measure, like the population average causal effect. By the law of iterated expectations, the population average causal effect of high school graduation is

$$E\left\{E\left[Y_i|X_i, s_i = 12\right] - E\left[Y_i|X_i, s_i = 11\right]\right\} \tag{3.2.3}$$
$$= E\left\{E\left[f_i(12) - f_i(11)|X_i\right]\right\}$$
$$= E\left[f_i(12) - f_i(11)\right] \tag{3.2.4}$$

In the same spirit, we might be interested in the average causal effect of high school graduation on high school graduates:

$$E\{E[Y_i|X_i, s_i = 12] - E[Y_i|X_i, s_i = 11]|s_i = 12\} \tag{3.2.5}$$
$$= E\{E[f_i(12) - f_i(11)|X_i]|s_i = 12\}$$
$$= E[f_i(12) - f_i(11)|s_i = 12]. \tag{3.2.6}$$

This parameter tells us how much high school graduates gained by virtue of having graduated. Likewise, for the effects of college graduation there is a distinction between $E[f_i(16) - f_i(12)|s_i = 16]$, the average causal effect on college graduates and $E[f_i(16) - f_i(12)]$, the population average effect.

The population average effect, (3.2.3), can be computed by averaging all of the X-specific effects using the marginal distribution of X_i, while the average effect on high school or college graduates averages the X-specific effects using the distribution of X_i in these groups. In both cases, the empirical counterpart is a matching estimator: we make comparisons across schooling groups graduates for individuals with the same covariate values, compute the difference in their earnings, and then average these differences in some way.

In practice, there are many details to worry about when implementing a matching strategy. We fill in some of the technical details on the mechanics of matching in Section 3.3.1, below. Here we note that a global drawback of the matching approach is that it is not "automatic," rather it requires two steps, matching and averaging. Estimating the standard errors of the resulting estimates may not be straightforward, either.

A third consideration is that the two-way contrast at the heart of this subsection (high school or college completers versus dropouts) does not do full justice to the problem at hand. Since s_i takes on many values, there are separate average causal effects for each possible increment in s_i, which also must be summarized in some way.[9] These considerations lead us back to regression.

Regression provides an easy-to-use empirical strategy that automatically turns the CIA into causal effects. Two routes can be traced from the CIA to regression. One assumes that $f_i(s)$ is both linear in s and the same for everyone except for an additive error term, in which case linear regression is a natural tool to estimate the features of $f_i(s)$. A more general but somewhat longer route recognizes that $f_i(s)$ almost certainly differs for different people, and, moreover, need not be linear in s. Even so, allowing for random variation in $f_i(s)$ across people, and for non-linearity for a given person, regression can be thought of as strategy for the estimation of a weighted average of the individual-specific difference, $f_i(s) - f_i(s-1)$. In fact, regression can be seen as a particular sort of matching estimator, capturing an average causal effect much like 3.2.3 or 3.2.5.

At this point, we want to focus on the conditions required for regression to have a causal interpretation and not on the details of the regression-matching analog. We therefore start with the first route, a linear constant-effects causal model. Suppose that

$$f_i(s) = \alpha + \rho s + \eta_i. \tag{3.2.7}$$

In addition to being linear, this equation says that the functional relationship of interest is the same for everyone. Again, s is written without an i subscript to index individuals, because equation (3.2.7) tells us what person i would earn for any value of s and not just the realized value, s_i. In this case, however, the only individual-specific and random part of $f_i(s)$ is a mean-zero error component, η_i, which captures unobserved factors that determine potential earnings.

Substituting the observed value s_i for s in equation (3.2.7), we have

$$Y_i = \alpha + \rho s_i + \eta_i. \tag{3.2.8}$$

Equation (3.2.8) looks like a bivariate regression model, except that equation (3.2.7) explicitly associates the coefficients in (3.2.8) with a causal relationship. Importantly, because equation (3.2.7) is a causal model, s_i may be correlated with potential outcomes, $f_i(s)$, or, in this case, the residual term in (3.2.8), η_i.

---

[9]For example, we might construct the average effect over s using the distribution of s_i. In other words, estimate $E[f_i(s) - f_i(s-1)]$ for each s by matching, and then compute the average difference

$$\sum E[f_i(s) - f_i(s-1)]P(s).$$

where $P(s)$ is the probability mass function for s_i. This is a discrete approximation to the average derivative, $E[f_i'(s_i)]$.

Suppose now that the CIA holds given a vector of observed covariates, X_i. In addition to the functional form assumption for potential outcomes embodied in (3.2.8), we decompose the random part of potential earnings, η_i, into a linear function of observable characteristics, X_i, and an error term, v_i:

$$\eta_i = X_i'\gamma + v_i,$$

where γ is a vector of population regression coefficients that is assumed to satisfy $E[\eta_i|X_i] = X_i'\gamma$. Because γ is defined by the regression of η_i on X_i, the residual v_i and X_i are uncorrelated *by construction*. Moreover, by virtue of the CIA, we have

$$E[f_i(s)|X_i, s_i] = E[f_i(s)|X_i] = \alpha + \rho s + E[\eta_i|X] = \alpha + \rho s + X_i'\gamma$$

Because mean-independence implies orthogonality, the residual in the linear causal model

$$Y_i = \alpha + \rho s_i + X_i'\gamma + v_i \tag{3.2.9}$$

is uncorrelated with the regressors, s_i and X_i, and the regression coefficient ρ is the causal effect of interest. It bears emphasizing once again that the key assumption here is that the observable characteristics, X_i, are the only reason why η_i and s_i (equivalently, $f_i(s)$ and s_i) are correlated. This is the selection-on-observables assumption for regression models discussed over a quarter century ago by Barnow, Cain, and Goldberger (1981). It remains the basis of most empirical work in Economics.

3.2.2   The Omitted Variables Bias Formula

The omitted variables bias (OVB) formula describes the relationship between regression estimates in models with different sets of control variables. This important formula is often motivated by the notion that a longer regression, i.e., one with more controls such as equation (3.2.9), has a causal interpretation, while a shorter regression does not. The coefficients on the variables included in the shorter regression are therefore said to be "biased". In fact, the OVB formula is a mechanical link between coefficient vectors that applies to short and long regressions whether or not the longer regression is causal. Nevertheless, we follow convention and refer to the difference between the included coefficients in a long regression and a short regression as being determined by the OVB formula.

To make this discussion concrete, suppose the set of relevant control variables in the schooling regression can be boiled down to a combination of family background, intelligence and motivation. Let these specific factors be denoted by a vector, A_i, which we'll refer to by the shorthand term "ability." The regression of

wages on schooling, s_i, controlling for ability can written as

$$Y_i = \alpha + \rho s_i + A_i'\gamma + \varepsilon_i, \tag{3.2.10}$$

where α, ρ, and γ are population regression coefficients, and ε_i is a regression residual that is uncorrelated with all regressors by definition. If the CIA applies given A_i, then ρ can be equated with the coefficient in the linear causal model, 3.2.7, while the residual ε_i is the random part of potential earnings that is left over after controlling for A_i.

In practice, ability is hard to measure. For example, the American Current Population Survey (CPS), a large data set widely used in applied microeconomics (and the source of U.S. government data on unemployment rates), tells us nothing about adult respondents' family background, intelligence, or motivation. What are the consequences of leaving ability out of regression (3.2.10)? The resulting "short regression" coefficient is related to the "long regression" coefficient in equation (3.2.10) as follows:

$$\frac{Cov(Y_i, s_i)}{V(s_i)} = \rho + \gamma'\delta_{As}, \tag{3.2.11}$$

where δ_{As} is the vector of coefficients from regressions of the elements of A_i on s_i. To paraphrase, the OVB formula says

Short equals long plus the effect of omitted times the regression of omitted on included.

This formula is easy to derive: plug the long regression into the short regression formula, $\frac{Cov(Y_i, s_i)}{V(s_i)}$. Not surprisingly, the OVB formula is closely related to the regression anatomy formula, 3.1.3, from Section 3.1.2. Both the OVB and regression anatomy formulas tell us that short and long regression coefficients are the same whenever the omitted and included variables are uncorrelated.[10]

We can use the OVB formula to get a sense of the likely consequences of omitting ability for schooling coefficients. Ability variables have positive effects on wages, and these variables are also likely to be positively correlated with schooling. The short regression coefficient may therefore be "too big" relative to what we want. On the other hand, as a matter of economic theory, the direction of the correlation between schooling and ability is not entirely clear. Some omitted variables may be negatively correlated with schooling, in which case the short regression coefficient will be too small.[11]

---

[10] Here is the multivariate generalization of OVB: Let β_1^s denote the coefficient vector on a $K_1 \times 1$ vector of variables, X_{1i} in a (short) regression that has no other variables and let β_1^l denote the coefficient vector on these variables in a (long) regression that includes a $K_2 \times 1$ vector of control variables, X_{2i}, with coefficient vector β_2^l. Then $\beta_1^s = \beta_1^l + E[X_{1i}X_{1i}']^{-1}E[X_{1i}X_{2i}']\beta_2^l$.

[11] As highly educated people, we like to assume that ability and schooling are positively correlated. This is not a foregone conclusion, however: Mick Jagger dropped out of the London School of Economics and Bill Gates dropped out of Harvard, perhaps because the opportunity cost of schooling for these high-ability guys was high (of course, they may also be a couple of very lucky college dropouts).

Table 3.2.1 illustrates these points using data from the NLSY. The first three entries in the table show that the schooling coefficient decreases from .132 to .114 when family background variables—in this case, parents' education—as well as a few basic demographic characteristics (age, race, census region of residence) are included as controls. Further control for individual ability, as proxied by the Armed Forces Qualification Test (AFQT) test score, reduces the schooling coefficient to .087 (AFQT is used by the military to select soldiers). The omitted variables bias formula tells us that these reductions are a result of the fact that the additional controls are positively correlated with both wages and schooling.[12]

Table 3.2.1: Estimates of the returns to education for men in the NLSY

	(1)	(2)	(3)	(4)	(5)
Controls:	None	Age dummies	Col. (2) and additional controls*	Col. (3) and AFQT score	Col. (4), with occupation dummies
	0.132	0.131	0.114	0.087	0.066
	(0.007)	(0.007)	(0.007)	(0.009)	(0.010)

Notes: Data are from the National Longitudinal Survey of Youth (1979 cohort, 2002 survey). The table reports the coefficient on years of schooling in a regression of log wages on years of schooling and the indicated controls. Standard errors are shown in parentheses. The sample is restricted to men and weighted by NLSY sampling weights. The sample size is 2434.

*Additional controls are mother's and father's years of schooling and dummy variables for race and Census region.

Although simple, the OVB formula is one of the most important things to know about regression. The importance of the OVB formula stems from the fact that if you claim an absence of omitted variables bias, then typically you're also saying that the regression you've got is the one you want. And the regression you want usually has a causal interpretation. In other words, you're prepared to lean on the CIA for a causal interpretation of the long-regression estimates.

At this point, it's worth considering when the CIA is most likely to give a plausible basis for empirical work. The best-case scenario is random assignment of s_i , conditional on X_i, in some sort of (possibly natural) experiment. An example is the study of a mandatory re-training program for unemployed workers by Black, *et al.* (2003). The authors of this study were interested in whether the re-training program succeeded in raising earnings later on. They exploit the fact that eligibility for the training program they study was determined on the basis of personal characteristics and past unemployment and job histories. Workers were divided up into groups on the basis of these characteristics. While some of these groups of workers were ineligible for training, those in other groups were required to take training if they did not take

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[12] A large empirical literature investigates the consequences of omitting ability variables from schooling equations. Key early references include Griliches and Mason (1972), Taubman (1976), Griliches (1977), and Chamberlain (1978).

a job. When some of the mandatory training groups contained more workers than training slots, training opportunities were distributed by lottery. Hence, training requirements were randomly assigned conditional on the covariates used to assign workers to groups. A regression on a dummy for training plus the personal characteristics, past unemployment variables, and job history variables used to classify workers seems very likely to provide reliable estimates of the causal effect of training.[13]

In the schooling context, there is usually no lottery that directly determines whether someone will go to college or finish high school.[14] Still, we might imagine subjecting individuals of similar ability and from similar family backgrounds to an experiment that encourages school attendance. The Education Maintenance Allowance, which pays British high school students in certain areas to attend school, is one such policy experiment (Dearden, et al, 2004).

A second type of study that favors the CIA exploits detailed institutional knowledge regarding the process that determines s_i . An example is the Angrist (1998) study of the effect of voluntary military service on the later earnings of soldiers. This research asks whether men who volunteered for service in the US Armed Forces were economically better off in the long run. Since voluntary military service is not randomly assigned, we can never know for sure. Angrist therefore used matching and regression techniques to control for observed differences between veterans and nonveterans who applied to get into the all-volunteer forces between 1979 and 1982. The motivation for a control strategy in this case is the fact that the military screens soldier-applicants primarily on the basis of observable covariates like age, schooling, and test scores.

The CIA in Angrist (1998) amounts to the claim that after conditioning on all these observed characteristics veterans and nonveterans are comparable. This assumption seems worth entertaining since, conditional on X_i, variation in veteran status in the Angrist (1998) study comes solely from the fact that some qualified applicants fail to enlist at the last minute. Of course, the considerations that lead a qualified applicant to "drop out" of the enlistment process could be related to earnings potential, so the CIA is clearly not guaranteed even in this case.

3.2.3 Bad Control

We've made the point that control for covariates can make the CIA more plausible. But more control is not always better. Some variables are bad controls and should not be included in a regression model even when their inclusion might be expected to change the short regression coefficients. Bad controls are variables that are themselves outcome variables in the notional experiment at hand. That is, bad controls might just as well be dependent variables too. Good controls are variables that we can think of as having been fixed at the time the regressor of interest was determined.

The essence of the bad control problem is a version of selection bias, albeit somewhat more subtle than

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[13]This program appears to raise earnings, primarily because workers in the training group went back to work more quickly.

[14]Lotteries have been used to distribute private school tuition subsidies; see, e.g., Angrist, et al. (2002).

the selection bias discussed in Chapter (2) and Section (3.2).   To illustrate, suppose we are interested in the effects of a college degree on earnings and that people can work in one of two occupations, white collar and blue collar. A college degree clearly opens the door to higher-paying white collar jobs. Should occupation therefore be seen as an omitted variable in a regression of wages on schooling? After all, occupation is highly correlated with both education and pay. Perhaps it's best to look at the effect of college on wages for those within an occupation, say white collar only. The problem with this argument is that once we acknowledge the fact that college affects occupation, comparisons of wages by college degree status within an occupation are no longer apples-to-apples, *even if college degree completion is randomly assigned.*

Here is a formal illustration of the bad control problem in the college/occupation example.[15]   Let W_i be a dummy variable that denotes white collar workers and let Y_i denote earnings.   The realization of these variables is determined by college graduation status and potential outcomes that are indexed against C_i. We have

$$
\begin{aligned}
Y_i &= C_i Y_{1i} + (1 - C_i) Y_{0i} \\
W_i &= C_i W_{1i} + (1 - C_i) W_{0i}
\end{aligned}
$$

where $C_i = 1$ for college graduates and is zero otherwise, $\{Y_{1i}, Y_{0i}\}$ denotes potential earnings, and $\{W_{1i}, W_{0i}\}$ denotes potential white-collar status.   We assume that C_i is randomly assigned, so it is independent of all potential outcomes.   We have no trouble estimating the causal effect of C_i on either Y_i or W_i since independence gives us

$$
\begin{aligned}
E\left[Y_i | C_i = 1\right] - E\left[Y_i | C_i = 0\right] &= E\left[Y_{1i} - Y_{0i}\right], \\
E\left[W_i | C_i = 1\right] - E\left[W_i | C_i = 0\right] &= E\left[W_{1i} - W_{0i}\right].
\end{aligned}
$$

In practice, we might estimate these average treatment effects by regressing Y_i and W_i and on C_i.

Bad control means that a comparison of earnings conditional on W_i does not have a causal interpretation. Consider the difference in mean earnings between college graduates and others conditional on working at a white collar job. We can compute this in a regression model that includes W_i or by regressing Y_i on C_i in the sample where $W_i = 1$. The estimand in the latter case is the difference in means with C_i switched off and on, conditional on $W_i = 1$:

$$
E\left[Y_i | W_i = 1, C_i = 1\right] - E\left[Y_i | W_i = 1, C_i = 0\right] = E\left[Y_{1i} | W_{1i} = 1, C_i = 1\right] - E\left[Y_{0i} | W_{0i} = 1, C_i = 0\right] \quad (3.2.12)
$$

---

[15] The same problem arises in "conditional-on-positive" comparisons, discussed in detail in section (3.4.2), below.

By the joint independence of $\{Y_{1i}, W_{1i}, Y_{0i}, W_{0i}\}$ and C_i, we have

$$E\left[Y_{1i}|W_{1i} = 1, C_i = 1\right] - E\left[Y_{0i}|W_{0i} = 1, C_i = 0\right] = E\left[Y_{1i}|W_{1i} = 1\right] - E\left[Y_{0i}|W_{0i} = 1\right].$$

This expression illustrates the apples-to-oranges nature of the bad-control problem:

$$E\left[Y_{1i}|W_{1i} = 1\right] - E\left[Y_{0i}|W_{0i} = 1\right]$$

$$= \underbrace{E\left[Y_{1i} - Y_{0i}|W_{1i} = 1\right]}_{\text{causal effect on college grads}} + \underbrace{\left\{E\left[Y_{0i}|W_{1i} = 1\right] - E\left[Y_{0i}|W_{0i} = 1\right]\right\}}_{\text{selection bias}}.$$

In other words, the difference in wages between those with and without a college degree conditional on working in a white collar job equals the causal effect of college on those with $W_{1i} = 1$ (people who work at a white collar job when they have a college degree) and a selection-bias term which reflects the fact that college changes the composition of the pool of white collar workers.

The selection-bias in this context can be positive or negative, depending on the relation between occupational choice, college attendance, and potential earnings. The main point is that even if $Y_{1i} = Y_{0i}$, so that there is no causal effect of college on wages, the conditional comparison in (3.2.12) will not tell us this (the regression of Y_i on W_i and C_i has exactly the same problem). It is also incorrect to say that the conditional comparison captures the part of the effect of college that is "not explained by occupation." In fact, the conditional comparison does not tell us much that is useful without a more elaborate model of the links between college, occupation, and earnings.[16]

As an empirical illustration, we see that the addition of two-digit occupation dummies indeed reduces the schooling coefficient in the NLSY models reported in Table 3.2.1, in this case from .087 to .066. However, it's hard to say what we should make of this decline. The change in schooling coefficients when we add occupation dummies may simply be an artifact of selection bias. So we would do better to control only for variables that are not themselves caused by education.

A second version of the bad control scenario involves *proxy control*, that is, the inclusion of variables that might partially control for omitted factors, but are themselves affected by the variable of interest. A simple version of the proxy-control scenario goes like this: Suppose you are interested in a long regression, similar to equation (3.2.10),

$$Y_i = \alpha + \rho S_i + \gamma a_i + \varepsilon_i, \tag{3.2.13}$$

where for the purposes of this discussion we've replaced the vector of controls A_i, with a scalar ability measure a_i. Think of this as an IQ score that measures innate ability in eighth grade, before any relevant

---

[16]In this example, selection bias is probably negative, that is $E\left[Y_{0i}|W_{1i} = 1\right] < E\left[Y_{0i}|W_{0i} = 1\right]$. It seems reasonable to think that any college graduate can get a white collar job, so $E\left[Y_{0i}|W_{1i} = 1\right]$ is not too far from $E[Y_{0i}]$. But someone who gets a white collar without benefit of a college degree (i.e., $W_{0i} = 1$) is probably special, i.e., has a better than average Y_{0i}.

schooling choices are made (assuming everyone completes eighth grade). The error term in this equation satisfies $E[S_i \varepsilon_i] = E[a_i \varepsilon_i] = 0$ by definition. Since a_i is measured before S_i is determined, it is a good control.

Equation (3.2.13) is the regression of interest, but unfortunately, data on a_i are unavailable. However, you have a second ability measure collected later, after schooling is completed (say, the score on a test used to screen job applicants). Call this variable "late ability," a_{li}. In general, schooling increases late ability relative to innate ability. To be specific, suppose

$$a_{li} = \pi_0 + \pi_1 S_i + \pi_2 a_i. \tag{3.2.14}$$

By this, we mean to say that both schooling and innate ability increase late or measured ability. There is almost certainly some randomness in measured ability as well, but we can make our point more simply via the deterministic link, (3.2.14).

You're worried about OVB in the regression of Y_i on S_i alone, so you propose to regress Y_i on S_i and late ability, a_{li} since the desired control, a_i, is unavailable. Using (3.2.14) to substitute for a_i in (3.2.13), the regression on S_i and a_{li} is

$$Y_i = (\alpha - \gamma \frac{\pi_0}{\pi_2}) + (\rho - \gamma \frac{\pi_1}{\pi_2})S_i + \frac{\gamma}{\pi_2} a_{li} + \varepsilon_i. \tag{3.2.15}$$

In this scenario, γ, π_1, and π_2 are all positive, so $\rho - \gamma \frac{\pi_1}{\pi_2}$ is too small unless π_1 turns out to be zero. In other words, use of a proxy control that is increased by the variable of interest generates a coefficient below the desired effect. Importantly, π_1 can be investigated to some extent: if the regression of a_{li} on S_i is zero, you might feel better about assuming that π_1 is zero in (3.2.14).

There is an interesting ambiguity in the proxy-control story that is not present in the first bad-control story. Control for outcome variables is simply misguided; you do not want to control for occupation in a schooling regression if the regression is to have a causal interpretation. In the proxy-control scenario, however, your intentions are good. And while proxy control does not generate the regression coefficient of interest, it may be an improvement on no control at all. Recall that the motivation for proxy control is equation (3.2.13). In terms of the parameters in this model, the OVB formula tells us that a regression on S_i with no controls generates a coefficient of $\rho + \gamma \delta_{as}$, where δ_{as} is slope coefficient from a regression of a_i on S_i. The schooling coefficient in (3.2.15) might be closer to ρ than the coefficient you estimate with no control at all. Moreover, assuming δ_{as} is positive, you can safely say that the causal effect of interest lies between these two.

One moral of both the bad-control and the proxy-control stories is that when thinking about controls, timing matters. Variables measured before the variable of interest was determined are generally good controls. In particular, because these variables were determined before the variable of interest, they cannot themselves

be outcomes in the causal nexus. In many cases, however, the timing is uncertain or unknown. In such cases, clear reasoning about causal channels requires explicit assumptions about what happened first, or the assertion that none of the control variables are themselves caused by the regressor of interest.[17]

3.3 Heterogeneity and Nonlinearity

As we saw in the previous section, a linear causal model in combination with the CIA leads to a linear CEF with a causal interpretation. Assuming the CEF is linear, the population regression is it. In practice, however, the assumption of a linear CEF is not really necessary for a causal interpretation of regression. For one thing, as discussed in Section 3.1.2, we can think of the regression of Y_i on X_i and S_i as providing the best linear approximation to the underlying CEF, regardless of its shape. Therefore, if the CEF is causal, the fact that regression approximates it gives regression coefficients a causal flavor. This claim is a little vague, however, and the nature of the link between regression and the CEF is worth exploring further. This exploration leads us to an understanding of regression as a computationally attractive matching estimator.

3.3.1 Regression Meets Matching

The past decade or two has seen increasing interest in matching as an empirical tool. Matching as a strategy to control for covariates is typically motivated by the CIA, as for causal regression in the previous section. For example, Angrist (1998) used matching to estimate the effects of volunteering for the military service on the later earnings of soldiers. These matching estimates have a causal interpretation assuming that, conditional on the individual characteristics the military uses to select soldiers (age, schooling, test scores), veteran status is independent of potential earnings.

An attractive feature of matching strategies is that they are typically accompanied by an explicit statement of the conditional independence assumption required to give matching estimates a causal interpretation. At the same time, we have just seen that the causal interpretation of a regression coefficient is based on exactly the same assumption. In other words, matching and regression are both control strategies. Since the core assumption underlying causal inference is the same for the two strategies, it's worth asking whether or to what extent matching really differs from regression. Our view is that regression can be motivated as a computational device for a particular sort of weighted matching estimator, and therefore the differences between regression and matching are unlikely to be of major empirical importance.

To flesh out this idea, it helps to look more deeply into the mathematical structure of the matching and regressions *estimands*, i.e., the population quantities that these methods attempt to estimate. For regression, of course, the estimand is a vector of population regression coefficients. The matching estimand is typically

---

[17]Griliches and Mason (1972) is a seminal exploration of the use of early and late ability controls in schooling equations. See also Chamberlain (1977, 1978) for closely related studies. Rosenbaum (1984) offers an alternative discussion of the proxy control idea using very different notation, outside of a regression framework.

a particular weighted average of contrasts or comparisons across cells defined by covariates. This is easiest to see in the case of discrete covariates, as in the military service example, and for a discrete regressor such as veteran status, which we denote here by the dummy, D_i. Since treatment takes on only two values, we can use the notation $Y_{1i}=f_i(1)$ and $Y_{0i}=f_i(0)$ to denote potential outcomes. A parameter of primary interest in this context is the average effect of treatment on the treated, $E[Y_{1i}-Y_{0i}|D_i = 1]$. This tells us the difference between the average earnings of soldiers, $E[Y_{1i}|D_i = 1]$, an observable quantity, and the counterfactual average earnings they would have obtained if they had not served, $E[Y_{0i}|D_i = 1]$. Simply comparing the observed earnings differential by veteran status is a biased measure of the effect of treatment on the treated unless D_i is independent of Y_{0i}. Specifically,

$$
\begin{aligned}
E\left[Y_i|D_i = 1\right] - E\left[Y_i|D_i = 0\right] &= E\left[Y_{1i} - Y_{0i}|D_i = 1\right] \\
&\quad + \left\{E\left[Y_{0i}|D_i = 1\right] - E\left[Y_{0i}|D_i = 0\right]\right\}.
\end{aligned}
$$

In other words, the observed earnings difference by veteran status equals the average effect of treatment on the treated plus selection bias. This parallels the discussion of selection bias in Chapter 2.

Given the CIA, selection bias disappears after conditioning on X_i, so the effect of treatment on the treated can be constructed by iterating expectations over X_i:

$$
\begin{aligned}
\delta_{TOT} &\equiv E[Y_{1i} - Y_{0i}|D_i = 1] \\
&= E\{E[Y_{1i}|X_i, D_i = 1] - E[Y_{0i}|X_i, D_i = 1]|D_i = 1\}.
\end{aligned}
$$

Of course, $E[Y_{0i}|X_i, D_i = 1]$ is counterfactual. By virtue of the CIA, however,

$$
E[Y_{0i}|X_i, D_i = 0] = E[Y_{0i}|X_i, D_i = 1].
$$

Therefore,

$$
\begin{aligned}
\delta_{TOT} &= E\left\{E\left[Y_{1i}|X_i, D_i = 1\right] - E\left[Y_{0i}|X_i, D_i = 0\right]|D_i = 1\right\} \\
&= E[\delta_X|D_i = 1],
\end{aligned}
\tag{3.3.1}
$$

where

$$
\delta_X \equiv E[Y_i|X_i, D_i = 1] - E[Y_i|X_i, D_i = 0],
$$

is the random X-specific difference in mean earnings by veteran status at each value of X_i.

The matching estimator in Angrist (1998) uses the fact that X_i is discrete to construct the sample analog

of the right-hand-side of (3.3.1). In the discrete case, the matching estimand can be written

$$E[\text{Y}_{1i} - \text{Y}_{0i}|\text{D}_i = 1] = \sum_x \delta_x P(\text{X}_i = x|\text{D}_i = 1), \tag{3.3.2}$$

where $P(\text{X}_i = x|\text{D}_i = 1)$ is the probability mass function for X_i given $\text{D}_i = 1$.[18] In this case, X_i, takes on values determined by all possible combinations of year of birth, test-score group, year of application to the military, and educational attainment at the time of application. The test score in this case is from the AFQT, used by the military to categorize the mental abilities of applicants (we included this as a control in the schooling regression discussed in Section 3.2.2). The Angrist (1998) matching estimator simply replaces δ_X by the sample veteran-nonveteran earnings difference for each combination of covariates, and then combines these in a weighted average using the empirical distribution of covariates among veterans.[19]

Note also that we can just as easily construct the unconditional average treatment effect,

$$
\begin{aligned}
\delta_{ATE} &= E\{E[\text{Y}_{1i}|\text{X}_i, \text{D}_i = 1] - E[\text{Y}_{0i}|\text{X}_i, \text{D}_i = 0]\} \\
&= \sum_x \delta_x P(\text{X}_i = x) \\
&= E[\text{Y}_{1i} - \text{Y}_{0i}],
\end{aligned} \tag{3.3.3}
$$

which is the expectation of δ_X using the marginal distribution of X_i instead of the distribution among the treated. δ_{TOT} tells us how much the typical soldier gained or lost as a consequence of military service, while δ_{ATE} tells us how much the typical applicant to the military gained or lost (since the Angrist, 1998, population consists of applicants.)

The US military tends to be fairly picky about it's soldiers, especially after downsizing at the end of the Cold War. For the most part, the military now takes only high school graduates with test scores in the upper half of the test score distribution. The resulting positive screening generates positive selection bias in naive comparisons of veteran and non-veteran earnings. This can be seen in Table 3.3.1, which reports differences-in-means, matching, and regression estimates of the effect voluntary military service on the 1988-91 Social Security-taxable earnings of men who applied to join the military between 1979 and 1982. The matching estimates were constructed from the sample analog of (3.3.2). Although white veterans earn $1,233 more than nonveterans, this difference becomes negative once differences in covariates are matched away. Similarly, while non-white veterans earn $2,449 more than nonveterans, controlling for covariates reduces this to $840.

---

[18] This matching estimator is discussed by Rubin (1977) and used by Card and Sullivan (1988) to estimate the effect of subsidized training on employment.

[19] With continuous covariates, exact matching is impossible and some sort of approximation is required, a fact that leads to bias. See Abadie and Imbens (2006), who derive the implications of approximate matching for the limiting distirbution of matching estimators.

Table 3.3.1: Uncontrolled, matching, and regression estimates of the effects of voluntary military service on earnings

Race	Average earnings in 1988-1991	Differences in means by veteran status	Matching estimates	Regression estimates	Regression minus matching
	(1)	(2)	(3)	(4)	(5)
Whites	14537	1233.4	-197.2	-88.8	108.4
		(60.3)	(70.5)	(62.5)	(28.5)
Non-whites	11664	2449.1	839.7	1074.4	234.7
		(47.4)	(62.7)	(50.7)	(32.5)

Notes: Adapted from Angrist (1998, Tables II and V). Standard errors are reported in parentheses. The table shows estimates of the effect of voluntary military service on the 1988-1991 Social Security- taxable earnings of men who applied to enter the armed forces between 1979 and 1982. The matching and regression estimates control for applicants' year of birth, education at the time of application, and AFQT score. There are 128,968 whites and 175,262 non-whites in the sample.

Table (3.3.1) also shows regression estimates of the effect of voluntary military service, controlling for the same set of covariates that were used to construct the matching estimates. These are estimates of δ_R in the equation

$$Y_i = \sum_x d_{ix}\beta_x + \delta_R D_i + \varepsilon_i, \tag{3.3.4}$$

where d_{ix} is a dummy that indicates $X_i = x$, β_x is a regression-effect for $X_i = x$, and δ_R is the regression estimand. Note that this regression model allows a separate parameter for every value taken on by the covariates. This model can therefore be said to be saturated-in-X_i, since it includes a parameter for every value of X_i (it is not "fully saturated," however, because there is a single additive effect for D_i with no $D_i \cdot X_i$ interactions).

Despite the fact that the matching and regression estimates control for the same variables, the regression estimates in Table 3.3.1 are somewhat larger than the matching estimates for both whites and nonwhites. In fact, the differences between the matching and regression results are statistically significant. At the same time, the two estimation strategies present a broadly similar picture of the effects of military service. The reason the regression and matching estimates are similar is that regression, too, can be seen as a sort of matching estimator: the regression estimand differs from the matching estimands only in the weights used to sum the covariate-specific effects, δ_X into a single effect. In particular, matching uses the distribution of covariates among the treated to weight covariate-specific estimates into an estimate of the effect of treatment on the treated, while regression produces a variance-weighted average of these effects.

To see this, start by using the regression anatomy formula to write the coefficient on D_i in the regression of Y_i on X_i and D_i as

$$\delta_R = \frac{Cov(Y_i, \tilde{D}_i)}{V(\tilde{D}_i)} \tag{3.3.5}$$

$$= \frac{E[(D_i - E[D_i|X_i])Y_i]}{E[(D_i - E[D_i|X_i])^2]}$$

$$= \frac{E\{(D_i - E[D_i|X_i])E[Y_i|D_i, X_i]\}}{E[(D_i - E[D_i|X_i])^2]}. \tag{3.3.6}$$

The second equality in this set of expressions uses the fact that saturating the model in X_i means $E[D_i|X_i]$ is linear. Hence, $\tilde{D}_i$, which is defined as the residual from a regression of D_i on X_i, is the difference between D_i and $E[D_i|X_i]$. The third equality uses the fact that the regression of Y_i on D_i and X_i is the same as the regression of Y_i on $E[Y_i|D_i,X_i]$.

To simplify further, we expand the CEF, $E[Y_i|D_i,X_i]$, to get

$$E[Y_i|D_i, X_i] = E[Y_i|D_i = 0, X_i] + \delta_X D_i.$$

If covariates are unnecessary - in other words, the CIA holds unconditionally, as if in a randomized trial - this CEF becomes

$$E[Y_i|D_i, X_i] = E[Y_i|D_i = 0] + E[Y_{1i} - Y_{0i}]D_i,$$

from which we conclude that the regression of Y_i on D_i estimates the population average treatment effect in this case (e.g., as in the experiment discussed in Section 2.3). But here we are interested in the more general scenario where conditioning X_i is necessary to eliminate selection bias.

To evaluate the more general regression estimand, (3.3.5), we begin by substituting for $E[Y_i|D_i,X_i]$ in the numerator. This gives

$$E\{(D_i - E[D_i|X_i])E[Y_i|D_i, X_i]\} = E\{(D_i - E[D_i|X_i])E[Y_i|D_i = 0, X_i]\} + E\{(D_i - E[D_i|X_i])D_i\delta_X\}.$$

The first term on the right-hand side is zero because $E[Y_i|D_i = 0,X_i]$ is a function of X_i and is therefore uncorrelated with $(D_i - E[D_i|X_i])$. For the same reason, the second term simplifies to

$$E\{(D_i - E[D_i|X_i])D_i\delta_X\} = E\{(D_i - E[D_i|X_i])^2\delta_X\}.$$

At this point, we've shown

$$\delta_R = \frac{E[(D_i - E[D_i|X_i])^2\delta_X]}{E[(D_i - E[D_i|X_i])^2]} = \frac{E\{E[(D_i - E[D_i|X_i])^2|X_i]\delta_X\}}{E\{E[(D_i - E[D_i|X_i])^2|X_i]\}} = \frac{E[\sigma_D^2(X_i)\delta_X]}{E[\sigma_D^2(X_i)]}, \tag{3.3.7}$$

where

$$\sigma_D^2(X_i) = E[(D_i - E[D_i|X_i])^2|X_i]$$

is the conditional variance of D_i given X_i. This establishes that the regression model, (3.3.4), produces a treatment-variance weighted average of δ_X.

Because the regressor of interest, D_i is a dummy variable, one last step can be taken. In this case, $\sigma_D^2(X_i) = P(D_i = 1|X_i)(1 - P(D_i = 1|X_i))$, so

$$\delta_R = \frac{\sum_x \delta_x \left[P(D_i = 1|X_i = x)(1 - P(D_i = 1|X_i = x))\right] P(X_i = x)}{\sum_x \left[P(D_i = 1|X_i = x)(1 - P(D_i = 1|X_i = x))\right] P(X_i = x)}$$

This shows that the regression estimand weights each covariate-specific treatment effect by $[P(X_i = x|D_i = 1)(1 - P(X_i = x|D_i = 1))]P(X_i = x)$. In contrast, the matching estimand for the effect of treatment on the treated can be written

$$E[Y_{1i} - Y_{0i}|D_i = 1] = \sum_x \delta_x P(X_i = x|D_i = 1) = \frac{\sum_x \delta_x P(D_i = 1|X_i = x)P(X_i = x)}{\sum_x P(D_i = 1|X_i = x)P(X_i = x)}$$

because

$$P(X_i = x|D_i = 1) = \frac{P(D_i = 1|X_i = x) \cdot P(X_i = x)}{P(D_i = 1)}.$$

So the weights used to construct $E[Y_{1i} - Y_{0i}|D_i = 1]$ are proportional to the probability of treatment at each value of the covariates.

The point of this derivation is that the treatment-on-the-treated estimand puts the most weight on covariate cells containing those who are most likely to be treated. In contrast, regression puts the most weight on covariate cells where the conditional variance of treatment status is largest. As a rule, this variance is maximized when $P(D_i = 1|X_i = x) = \frac{1}{2}$, in other words, for cells where there are equal numbers of treated and control observations. Of course, the difference in weighting schemes is of little importance if δ_x does not vary across cells (though weighting still affects the statistical efficiency of estimators). In this example, however, men who were most likely to serve in the military appear to benefit least from their service. This is probably because those most likely to serve were most qualified, but therefore also had the highest civilian earnings potential and so benefited least from military service. This fact leads matching estimates of the effect of military service to be smaller than regression estimates based on the same vector of control variables.[20]

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[20] It's no surprise that regression gives the most weight to cells where $P(D_i = 1|X_i = x) = 1/2$ since regression is efficient for a homoskedastic constant-effects linear model. We should expect an efficient estimator to give the most weight to cells where the common treatment effect is estimated most precisely. With homoskedastic residuals, the most precise treatment effects

Importantly, neither the regression nor the covariate-matching estimands give any weight to covariate cells that do not contain both treated and control observations. Consider a value of X_i, say x^*, where either no one is treated or everyone is treated. Then, δ_{x^*} is undefined, while the regression weights, $[P(D_i = 1|X_i = x^*)(1 - P(D_i = 1|X_i = x^*))]$, are zero. In the language of the econometric literature on matching, both the regression and matching estimands impose *common support*, that is, they are limited to covariate values where both treated and control observations are found.[21]

The step from *estimand* to *estimator* is a little more complicated. In practice, both regression and matching estimators are implemented using modelling assumptions that implicitly involve a certain amount of extrapolation across cells. For example, matching estimators often combine covariates cells with few observations. This violates common support if the cells being combined do not each have both treated and non-treated observations. Regression models that are not saturated in X_i may also violate common support, since covariate cells without both treated and control observations can end up contributing to the estimates by extrapolation. Here too, however, we see a symmetry between the matching and regression strategies: they are in the same class, in principle, and require the same sort of compromises in practice.[22]

Even More on Regression and Matching: Ordered and Continuous Treatments[★]

Does the pseudo-matching interpretation of regression outlined above for a binary treatment apply to models with ordered and continuous treatments? The long answer is fairly technical and may be more than you want to know. The short answer is, to one degree or another, "yes."

As we've already discussed, one interpretation of regression is that the population OLS slope vector provides the MMSE linear approximation to the CEF. This, of course, works for ordered and continuous regressors as well as for binary. A related property is the fact that regression coefficients have an "average derivative" interpretation. In multivariate regression models, this interpretation is unfortunately complicated by the fact that the OLS slope vector is a matrix-weighted average of the gradient of the CEF. Matrix-weighted averages are difficult to interpret except in special cases (see Chamberlain and Leamer, 1976). An important special case when the average derivative property is relatively straightforward is in regression models for an ordered or continuous treatment with a saturated model for covariates. To avoid lengthy derivations, we simply explain the formulas. A derivation is sketched in the appendix to this chapter. For additional details, see the appendix to Angrist and Krueger (1999).

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come from cells where the probability of treatment equals $1/2$.

[21] The *support* of a random variable is the set of realizations that occur with positive probability. See Heckman, Ichimura, Smith, and Todd (1998) and Smith and Todd (2001) for a discussion of common support in matching.

[22] Matching problems involving finely distributed X-variables are often solved by aggregating values to make coarser groupings or by pairing observations that have similar, though not necessarily identical values. See Cochran (1965), Rubin (1973), or Rosenbaum (1995, Chapter 3) for discussions of this approach. With continuously-distributed covariates, matching estimators are biased because matches are imperfect. Abadie and Imbens (2008) have recently shown that a regression-based bias correction can eliminate the (asymptotic) bias from imperfect matches.

For the purposes of this discussion, the treatment intensity, s_i, is assumed to be a continuously distributed random variable, not necessarily non-negative. Suppose that the CEF of interest can be written $h(t) \equiv E[Y_i|s_i = t]$ with derivative $h'(t)$. Then

$$\frac{E[Y_i(s_i - E[s_i])]}{E[s_i(s_i - E[s_i])]} = \frac{\int h'(t)\,\mu_t dt}{\int \mu_t dt} \tag{3.3.8}$$

where

$$\mu_t \equiv \{E[s_i|s_i \geq t] - E[s_i|s_i < t]\}\{P(s_i \geq t)[1 - P(s_i \geq t)\}, \tag{3.3.9}$$

and the integrals in (3.3.8) run over the possible values of s_i. This formula weights each possible value of s_i in proportion to the difference in the conditional mean of s_i above and below that value. More weight is also given to points close to the median of s_i since $P(s_i \geq t) \cdot [1 - P(s_i \geq t)]$ is maximized at $P(s_i \geq t) = 1/2$.

With covariates, X_i, the weights in (3.3.8) become X-specific. A covariate-averaged version of the same formula applies to the multivariate regression coefficient of Y_i on s_i, after partialling out X_i. In particular,

$$\frac{E[Y_i(s_i - E[s_i|X_i])]}{E[s_i(s_i - E[s_i|X])]} = \frac{E\left[\int h'_X(t)\mu_{tX} dt\right]}{E\left[\int \mu_{tX} dt\right]}, \tag{3.3.10}$$

where $h'_X(t) \equiv \frac{\partial E[Y_i|X_i, s_i = t]}{\partial t}$ and $\mu_{tX} \equiv \{E[s_i|X_i, s_i \geq t] - E[s_i|X_i, s_i < t]\}\{P(s_i \geq t|X_i)[1 - P(s_i \geq t|X_i)]\}$. It bears emphasizing that equation (3.3.10) reflects two types of averaging: an integral that averages *along* the length of a nonlinear CEF at fixed covariate values, and an expectation that averages *across* covariate cells. An important point in this context is that population regression coefficients contain no information about the effect of s_i on the CEF for values of X_i where $P(s_i \geq t|X_i)$ equals 0 or 1. This includes values of X_i where s_i is fixed. In the same spirit, it's worth noting that if s_i is a dummy variable, we can extract equation (3.3.7) from the more general formula, (3.3.10).

Angrist and Krueger (1999) construct the average weighting function for a schooling regression with state of birth and year of birth covariates. Although equations (3.3.8) and (3.3.10) may seem arcane or at least non-obvious, in this example the average weights, $E[\mu_{tX}]$, turn out to be a reasonably smooth symmetric function of t, centered at the mode of s_i.

The implications of (3.3.8) or (3.3.10) can be explored further given a model for the distribution of regressors. Suppose, for example, that s_i is Normally distributed. Let $z_i = \frac{s_i - E(s_i)}{\sigma_s}$, where σ_s is the standard deviation of s_i, so that z_i is standard Normal. Then

$$E[s_i|s_i \geq t] = E(s_i) + \sigma_s E\left[z_i|z_i \geq \frac{t - E(s_i)}{\sigma_s}\right] = E(s_i) + \sigma_s E\left[z_i|z_i \geq t^*\right].$$

From truncated Normal formulas (see, e.g., Johnson and Kotz, 1970), we know that

$$E[z_i|z_i > t^*] = \frac{\phi(t^*)}{[1 - \Phi(t^*)]} \text{ and } E[z_i|z_i < t^*] = \frac{-\phi(t^*)}{\Phi(t^*)}.$$

where $\phi(\cdot)$ and $\Phi(\cdot)$ are the standard Normal density and distribution function. Substituting in the formula for μ_t, (3.3.9), we have

$$\mu_t = \sigma_s \left\{ \frac{\phi(t^*)}{[1 - \Phi(t^*)]} - \frac{-\phi(t^*)}{\Phi(t^*)} \right\} [1 - \Phi(t^*)]\Phi(t^*) = \sigma_s \phi(t^*).$$

We have therefore shown that

$$\frac{Cov(\text{Y}_i, \text{S}_i)}{V(\text{S}_i)} = E[h'(\text{S}_i)].$$

In other words, the regression of Y_i on S_i is the (unweighted!) population average derivative, $E[h'(\text{S}_i)]$, when S_i is Normally distributed. Of course, this result is a special case of a special case.[23] Still, it seems reasonable to imagine that Normality might not matter very much. And in our empirical experience, the average derivatives (also called "marginal effects") constructed from parametric nonlinear models for limited dependent variables (e.g., Probit or Tobit) are usually indistinguishable from the corresponding regression coefficients, regardless of the distribution of regressors. We expand on this point in Section 3.4.2, below.

3.3.2 Control for Covariates Using the Propensity Score

The most important result in regression theory is the omitted variables bias formula: coefficients on included variables are unaffected by the omission of variables when the variables omitted are uncorrelated with the variables included. The propensity score theorem, due to Rosenbaum and Rubin (1983), extends this idea to estimation strategies that rely on matching instead of regression, where the causal variable of interest is a treatment dummy.[24]

The propensity score theorem states that if potential outcomes are independent of treatment status conditional on a multivariate covariate vector, X_i, then potential outcomes are independent of treatment status conditional on a scalar function of covariates, the propensity score, defined as $p(\text{X}_i) \equiv E[\text{D}_i|\text{X}_i]$. Formally, we have

Theorem 3.3.1 *The Propensity-Score Theorem.*

Suppose the CIA holds for Y_{ji}; $j = 0, 1$. *Then* $\text{Y}_{ji} \perp\!\!\!\perp \text{D}_i | p(\text{X}_i)$.

---

[23] More specialized results in this spirit appear in Ruud (1986), who considers distribution-free estimation of limited-dependent-variable models with Normally distributed regressors.

[24] Propensity-score methods can be adapted to multi-valued treatments, though this has yet to catch on. See Imbens (2000) for an effort in this direction.

Proof. The claim is true if $P[\mathrm{D}_i = 1|\mathrm{Y}_{ji}, p(\mathrm{X}_i)]$ does not depend on Y_{ji}.

$$
\begin{aligned}
P[\mathrm{D}_i = 1|\mathrm{Y}_{ji}, p(\mathrm{X}_i)] &= E[\mathrm{D}_i|\mathrm{Y}_{ji}, p(\mathrm{X}_i)] \\
&= E\{E[\mathrm{D}_i|\mathrm{Y}_{ji}, p(\mathrm{X}_i), \mathrm{X}_i]|\mathrm{Y}_{ji}, p(\mathrm{X}_i)\} \\
&= E\{E[\mathrm{D}_i|\mathrm{Y}_{ji}, \mathrm{X}_i]|\mathrm{Y}_{ji}, p(\mathrm{X}_i)\} \\
&= E\{E[\mathrm{D}_i|\mathrm{X}_i]|\mathrm{Y}_{ji}, p(\mathrm{X}_i)\}, \text{by the CIA.}
\end{aligned}
$$

But $E\{E[\mathrm{D}_i|\mathrm{X}_i]|\mathrm{Y}_{ji}, p(\mathrm{X}_i)\} = E\{p(\mathrm{X}_i)|\mathrm{Y}_{ji}, p(\mathrm{X}_i)\}$, which is clearly just $p(\mathrm{X}_i)$. ∎

Like the OVB formula for regression, the propensity score theorem says you need only control for covariates that affect the probability of treatment. But it also says something more: the only covariate you really need to control for is the probability of treatment itself. In practice, the propensity score theorem is usually used for estimation in two steps: first, $p(\mathrm{X}_i)$ is estimated using some kind of parametric model, say, Logit or Probit. Then estimates of the effect of treatment are computed either by matching on the fitted values from this first step, or by a weighting scheme described below (see, Imbens, 2004, for an overview).

In practice there are many ways to use the propensity score theorem for estimation. Direct propensity-score matching works like covariate matching, except that we match on the score instead of the covariates directly. By the propensity score theorem and the CIA,

$$
E[\mathrm{Y}_{1i} - \mathrm{Y}_{0i}|\mathrm{D}_i = 1] = E\left\{E[\mathrm{Y}_i|p(\mathrm{X}_i), \mathrm{D}_i = 1] - E[\mathrm{Y}_i|p(\mathrm{X}_i), \mathrm{D}_i = 0]|\mathrm{D}_i = 1\right\}.
$$

Estimates of the effect of treatment on the treated can therefore be obtained by stratifying on an estimate of $p(\mathrm{X}_i)$ and substituting conditional sample averages for expectations or by matching each treated observation to controls with the same or similar values of the propensity score (both of these approaches were used by Dehejia and Wahba, 1999). Alternately, a model-based or non-parametric estimate of $E[\mathrm{Y}_i|p(\mathrm{X}_i), \mathrm{D}_i]$ can be substituted for these conditional mean functions and the outer expectation replaced with a sum (as in Heckman, Ichimura, and Todd, 1998).

The somewhat niftier weighting approach to propensity-score estimation skips the cumbersome matching step by exploiting the fact that the CIA implies $E\left[\frac{\mathrm{Y}_i \mathrm{D}_i}{p(\mathrm{X}_i)}\right] = E[\mathrm{Y}_{1i}]$ and $E[\frac{\mathrm{Y}_i(1-\mathrm{D}_i)}{(1-p(\mathrm{X}_i))}] = E[\mathrm{Y}_{0i}]$. Therefore, given a scheme for estimating $p(\mathrm{X}_i)$, we can construct estimates of the average treatment effect from the sample analog of

$$
\begin{aligned}
E[\mathrm{Y}_{1i} - \mathrm{Y}_{0i}] &= E\left[\frac{\mathrm{Y}_i \mathrm{D}_i}{p(\mathrm{X}_i)} - \frac{\mathrm{Y}_i(1 - \mathrm{D}_i)}{1 - p(\mathrm{X}_i)}\right] \\
&= E\left[\frac{(\mathrm{D}_i - p(\mathrm{X}_i))\mathrm{Y}_i}{p(\mathrm{X}_i)(1 - p(\mathrm{X}_i))}\right].
\end{aligned} \tag{3.3.11}
$$

This last expression is an estimand of the form suggested by Newey (1990) and Robins, Mark, and Newey

(1992). We can similarly calculate the effect of treatment on the treated from the sample analog of:

$$E[\text{Y}_{1i} - \text{Y}_{0i}|\text{D}_i = 1] = E\left[\frac{(\text{D}_i - p(\text{X}_i))\text{Y}_i}{(1 - p(\text{X}_i))P(\text{D}_i)}\right]. \tag{3.3.12}$$

The idea that you can correct for non-random sampling by weighting by the reciprocal of the probability of selection dates back to Horvitz and Thompson (1952). Of course, to make this approach feasible, and for the resulting estimates to be consistent, we need a consistent estimator for $p(\text{X}_i)$

The Horvitz-Thompson version of the propensity-score approach is appealing since the estimator is essentially automated, with no cumbersome matching required. The Horvitz-Thompson approach also highlights the close link between propensity-score matching and regression, much as discussed for covariate matching in section 3.3.1. Consider again the regression estimand, δ_R, for the population regression of Y_i on D_i, controlling for a saturated model for covariates. This estimand can be written

$$\delta_R = \frac{E[(\text{D}_i - p(\text{X}_i))\text{Y}_i]}{E[p(\text{X}_i)(1 - p(\text{X}_i))]}. \tag{3.3.13}$$

The two Horvitz-Thompson matching estimands and the regression estimand are all members of the class of weighted average estimands considered by Hirano, Imbens, and Ridder (2003):

$$E\left\{g(\text{X}_i)\left[\frac{\text{Y}_i\text{D}_i}{p(\text{X}_i)} - \frac{\text{Y}_i(1 - \text{D}_i)}{(1 - p(\text{X}_i))}\right]\right\}, \tag{3.3.14}$$

where $g(\text{X}_i)$ is a known weighting function (To go from estimand to estimator, replace $p(\text{X}_i)$ with a consistent estimator, and expectations with sums). For the average treatment effect, set $g(\text{X}_i) = 1$; for the effect on the treated, set $g(\text{X}_i) = \frac{p(\text{X}_i)}{P(\text{D}_i)}$; and for regression set

$$g(\text{X}_i) = \frac{p(\text{X}_i)(1 - p(\text{X}_i))}{E[p(\text{X}_i)(1 - p(\text{X}_i))]}.$$

This similarity highlights once again the fact that regression and matching—including propensity score matching—are not really different animals, at least not until we specify a model for the propensity score.

A big question here is how best to model and estimate $p(\text{X}_i)$, or how much smoothing or stratification to use when estimating $E[\text{Y}_i|p(\text{X}_i), \text{D}_i]$, especially if the covariates are continuous The regression analog of this question is how to parametrize the control variables (e.g., polynomials or main effects and interaction terms if the covariates are coded as discrete). The answer to this is inherently application-specific. A growing empirical literature suggests that a Logit model for the propensity score with a few polynomial terms in continuous covariates works well in practice, though this cannot be a theorem (see, e.g., Dehejia and Wahba, 1999).

A developing theoretical literature has produced some thought-provoking theorems on efficient use of the

propensity score. First, from the point of view of asymptotic efficiency, there is usually a cost to matching on the propensity score instead of full covariate matching. We can get lower asymptotic standard errors by matching on any covariate that explains outcomes, whether or not it turns up in the propensity score. This we know from Hahn's (1998) investigation of the maximal precision that it is possible to obtain for estimates of treatment effects under the CIA, with and without knowledge of the propensity score. For example, in Angrist (1998), there is an efficiency gain from matching on year of birth, even if the probability of serving in the military is unrelated to birth year, because earnings are related to birth year. A regression analog for this point is the result that even in a scenario with no omitted variables bias, the long regression generates more precise estimates of the coefficients on the variables included in a short regression whenever these variables have some predictive power for outcomes because these covariates lead to a smaller residual variance (see Section 3.1.3).

Hahn's (1998) results raise the question of why we should ever bother with estimators that use the propensity score. A philosophical argument is that the propensity score rightly focuses researcher attention on models for treatment assignment, something about which we may have reasonably good information, instead of the typically more complex and mysterious process determining outcomes. This view seems especially compelling when treatment assignment is the outcome of human institutions or government regulations while the process determining outcomes is more anonymous (e.g., a market). For example, in a time series evaluation of the causal effects of monetary policy, Angrist and Kuersteiner (2004) argue that we know more about how the Federal Reserve sets interests rates than about the process determining GDP. In the same spirit, it may also be easier to validate a model for treatment assignment than to validate a model for outcomes (see, e.g., Rosenbaum and Rubin, 1985, for a version of this argument).

A more precise though purely statistical argument for using the propensity score is laid out in Angrist and Hahn (2004). This paper shows that even though there is no asymptotic efficiency gain from the use of estimators based on the propensity score, there will often be a gain in precision in finite samples. Since all real data sets are finite, this result is empirically relevant. Intuitively, if the covariates omitted from the propensity score explain little of the variation in outcomes (in a purely statistical sense), it may then be better to ignore them than to bear the statistical burden imposed by the need to estimate their effects. This is easy to see in studies using data sets such as the NLSY where there are hundreds of covariates that might predict outcomes. In practice, we focus on a small subset of all possible covariates. This subset is chosen with an eye to what predicts treatment as well as outcomes.

Finally, Hirano, Imbens, and Ridder (2003) provide an alternative asymptotic resolution of the "propensity score paradox" generated by Hahn's (1998) theorems. They show that even though estimates of treatment effects based on a known propensity score are inefficient, for models with continuous covariates, a Horvitz-Thompson-type weighting estimator is efficient when weighting uses a *non-parametric estimate* of the score. The fact that the propensity score is estimated and the fact that it is estimated non-parametrically

are both key for the Hirano, Imbens, and Ridder conclusions.

Do the Hirano, Imbens, and Ridder (2003) results resolve the propensity-score paradox? For the moment, we prefer the finite-sample resolution given by Angrist and Hahn (2004). Their results highlight the fact that it is the researchers' willingness to impose some restrictions on the score which gives propensity-score-based inference its conceptual and statistical power. In Angrist (1998), for example, an application with high-dimensional though discrete covariates, the unrestricted non-parametric estimator of the score is just the empirical probability of treatment in each covariate cell. With this nonparametric estimator plugged in for $p(\mathrm{X}_i)$, it's straightforward to show that the sample analogs of (3.3.11) and (3.3.12) are algebraically equivalent to the corresponding full-covariate matching estimators. Hence, it's no surprise that score-based estimation comes out efficient, since full-covariate matching is the asymptotically efficient benchmark. An essential element of propensity score methods is the use of prior knowledge for dimension reduction. The statistical payoff is an improvement in finite-sample behavior. If you're not prepared to smooth, restrict, or otherwise reduce the dimensionality of the matching problem in a manner that has real empirical consequences, then you might as well go for full covariate matching or saturated regression control.

3.3.3 Propensity-Score Methods vs. Regression

Propensity-score methods shift attention from the estimation of $E[\mathrm{Y}_i|\mathrm{X}_i,\mathrm{D}_i]$ to the estimation of the propensity score, $p(\mathrm{X}_i) \equiv E[\mathrm{D}_i|\mathrm{X}_i]$. This is attractive in applications where the latter is easier to model or motivate. For example, Ashenfelter (1978) showed that participants in government-funded training programs often have suffered a marked pre-program dip in earnings, a pattern found in many later studies. If this dip is the only thing that makes trainees special, then we can estimate the causal effect of training on earnings by controlling for past earnings dynamics. In practice, however, it's hard to match on earnings dynamics since earnings histories are both continuous and multi-dimensional. Dehejia and Wahba (1999) argue in this context that the causal effects of training programs are better estimated by conditioning on the propensity score than by conditioning on the earnings histories themselves.

The propensity-score estimates reported by Dehejia and Wahba are remarkably close to the estimates from a randomized trial that constitute their benchmark. Nevertheless, we believe regression should be the starting point for most empirical projects. This is not a theorem; undoubtedly, there are circumstances where propensity score matching provides more reliable estimates of average causal effects. The first reason we don't find ourselves on the propensity-score bandwagon is practical: there are many details to be filled in when implementing propensity-score matching - such as how to model the score and how to do inference - these details are not yet standardized. Different researchers might therefore reach very different conclusions, even when using the same data and covariates. Moreover, as we've seen with the Horvitz-Thompson estimands, there isn't very much theoretical daylight between regression and propensity-score weighting. If the regression model for covariates is fairly flexible, say, close to saturated, regression can be seen as a type

of propensity-score weighting, so the difference is mostly in the implementation. In practice you may be far from saturation, but with the right covariates this shouldn't matter.

The face-off between regression and propensity-score matching is illustrated here using the same National Supported Work (NSW) sample featured in Dehejia and Wahba (1999).[25]   The NSW is a mid-1970s program that provided work experience to a sample with weak labor-force attachment. Somewhat unusually for it's time, the NSW was evaluated in a randomized trial. Lalonde's (1986) path-breaking analysis compared the results from the NSW randomized study to econometric results using non-experimental control groups drawn from the PSID and the CPS. He came away pessimistic because plausible non-experimental methods generated a wide range of results, many of which were far from the experimental estimates. Moreover, Lalonde argued, an objective investigator, not knowing the results of the randomized trial, would be unlikely to pick the best econometric specifications and observational control groups.

In a striking second take on the Lalonde (1986) findings, Dehejia and Wahba (1999) found that they could come close to the NSW experimental results by matching the NSW treatment group to observational control groups selected using the propensity score. They demonstrated this using various comparison groups. Following Dehejia and Wahba (1999), we look again at two of the CPS comparison groups, first, a largely unselected sample (CPS-1) and then a narrower comparison group selected from the recently unemployed (CPS-3).

Table 3.3.2 (a replication of Table 1 in Dehejia and Wahba, 1999) reports descriptive statistics for the NSW treatment group, the randomly selected NSW control group, and our two observational control groups. The NSW treatment group and the randomly selected NSW control groups are younger, less educated, more likely to be nonwhite, and have much lower earnings than the general population represented by the CPS-1 sample. The CPS-3 sample matches the NSW treatment group more closely but still shows some differences, particularly in terms of race and pre-program earnings.

Table 3.3.3 reports estimates of the NSW treatment effect. The dependent variable is annual earnings in 1978, a year or two after treatment. Rows of the table show results with alternative sets of controls: none; all the demographic variables in Table 3.3.2; lagged (1975) earnings; demographics plus lagged earnings; demographics and two lags of earnings. All estimates are from regressions of 1978 earnings on a treatment dummy plus controls (the raw treatment-control difference appears in the first row).

Estimates using the experimental control group, reported in column 1, are in the order of $1,600-1,800. Not surprisingly, these estimates vary little across specifications. In contrast, the raw earnings gap between NSW participants and the CPS-1 sample, reported in column 2, is roughly $-8,500, suggesting this comparison is heavily contaminated by selection bias. The addition of demographic controls and lagged earnings narrows the gap considerably; the estimated treatment effect reaches (positive) $800 in the last row. The results

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[25] An similar but more extended propensity-score face-off appears in the exchange beween Smith and Todd (2005) and Dehejia (2005).

are even better in column 3, which uses the narrower CPS-3 comparison group. The characteristics of this group are much closer to the those of NSW participants; consistent with this, the raw earnings difference is only $-635. The fully-controlled estimate, reported in the last row, is close to $1,400, not far from the experimental treatment effect.

A drawback of the process taking us from CPS-1 to CPS-3 is the *ad hoc* nature of the rules used to construct the smaller and more carefully-selected CPS-3 comparison group. The CPS-3 selection criteria can be motivated by the NSW program rules, which favor individuals with low earnings and weak labor-force attachment, but in practice, there are many ways to implement this. We'd therefore like a more systematic approach to pre-screening. In a recent paper, Crump, Hotz, Imbens and Mitnik (2006) suggest that the propensity score be used for systematic sample-selection as a *precursor* to regression estimation. This contrasts with our earlier discussion of the propensity score as the basis for an *estimator*.

We implemented the Crump, *et al.* (2006) suggestion by first estimating the propensity score on a pooled NSW-treatment and observational-comparison sample, and then picking only those observations with $0.1 < p(X_i) < 0.9$. In other words, the estimation sample is limited to observations with a predicted probability of treatment equal to at least 10 percent, but no more than 90 percent. This ensures that regressions are estimated with a sample including only covariate cells with there are at least a few treated and control observations. Estimation using screened samples therefore requires no extrapolation to cells without "common support", i.e. to cells where there is no overlap in the covariate distribution between treatment and controls. Descriptive statistics for samples screened on the score (estimated using the full set of covariates listed in the table) appear in the last two columns of Table 3.3.2. The covariate means in screened CPS-1 and CPS-3 are much closer to the NSW means in column 1 than are the covariate means from unscreened samples.

We explored the common-support screener further using alternative sets of covariates, but with the same covariates used for both screening and the estimation of treatment effects at each iteration. The resulting estimates are displayed in the final two columns of Table 3.3.3. Controlling for demographic variables or lagged earnings alone, these results differ little from those in columns 2-3. With both demographic variables and a single lag of earnings as controls, however, the screened CPS-1 estimates are quite a bit closer to the experimental estimates than are the unscreened results. Screened CPS-1 estimates with two lags of earnings remain close to the experimental benchmark. On the other hand, the common-support screener improves the CPS-3 results only slightly with a single lag of earnings and seems to be a step backward with two.

This investigation boosts our (already strong) faith in regression. Regression control for covariates does a good job of eliminating selection bias in the CPS-1 sample in spite of a huge baseline gap. Restricting the sample using our knowledge of program admissions criteria yields even better regression estimates with CPS-3, about as good as Dehejia and Wahba's (1999) propensity score matching results with two lags of earnings. Systematic pre-screening to enforce common support seems like a useful adjunct to regression

estimation with CPS-1, a large and coarsely-selected initial sample. The estimates in screened CPS-1 are as good as unscreened CPS-3. We note, however, that the standard errors for estimates using propensity-score-screened samples have not been adjusted to reflect sampling variance in our estimates of the score. An advantage of pre-screening using prior information, as in the step from CPS-1 to CPS-3, is that no such adjustment is necessary.

3.4   Regression Details

3.4.1   Weighting Regression

Few things are as confusing to applied researchers as the role of sample weights. Even now, 20 years post-Ph.D., we read the section of the Stata manual on weighting with some dismay. Weights can be used in a number of ways, and how they are used may well matter for your results. Regrettably, however, the case for or against weighting is often less than clear-cut, as are the specifics of how the weights should be programmed. A detailed discussion of weighting pros and cons is beyond the scope of this book. See Pfefferman (1993) and Deaton (1997) for two perspectives. In this brief subsection, we provide a few guidelines and a rationale for our approach to weighting.

A simple rule of thumb for weighting regression is use weights when they make it more likely that the regression you are estimating is close to the population target you are trying to estimate. If, for example, the target (or *estimand*) is the population regression function, and the sample to be used for estimation is non-random with sampling weights, w_i, equal to the inverse probability of sampling observation i, then it makes sense to use weighted least squares, weighting by w_i (for this you can use Stata *pweights or a SAS WEIGHT* statement). Weighting by the inverse sampling probability generates estimates that are consistent for the population regression function even if the sample you have to work with is not a simple random sample.

A related weighting scenario is grouped data. Suppose that you would like to regress Y_i on X_i in a random sample, presumably because you want to learn about the population regression vector $\beta = E[X_i X_i']^{-1} E[X_i Y_i]$. Instead of a random sample, however, you have data grouped at the level of X_i. That is, you have estimates of $E[Y_i | X_i = x]$ for each x, estimated using data from a random sample. Let this average be denoted $\bar{y}_x$, and suppose you also know n_x, where n_x/N is the relative frequency of x in the underlying random sample. As we saw in Section 3.1.2, the regression of $\bar{y}_x$ on x, weighted by n_x is the same as the random-sample regression. Therefore, if your goal is to get back to the microdata regression, it makes sense to weight by group size. We note, however, that macroeconomists, accustomed to working with published averages and ignoring the underlying microdata, might disagree, or perhaps take the point in principle but remain disinclined to buck tradition in their discipline, which favors the unweighted analysis of aggregates.

If, on the other hand, the rationale for weighting has something to do with heteroskedasticity, as in many

Table 3.3.2: Covariate means in the NSW and observational control samples

	NSW		Full Samples		P-score Screened Samples	
Variable	Treated	Control	CPS-1	CPS-3	CPS-1	CPS-3
	(1)	(2)	(3)	(4)	(5)	(6)
Age	25.82	25.05	33.23	28.03	25.63	25.97
Years of schooling	10.35	10.09	12.03	10.24	10.49	10.42
Black	0.84	0.83	0.07	0.20	0.96	0.52
Hispanic	0.06	0.11	0.07	0.14	0.03	0.20
Dropout	0.71	0.83	0.30	0.60	0.60	0.63
Married	0.19	0.15	0.71	0.51	0.26	0.29
1974 earnimigs	2,096	2,107	14,017	5,619	2,821	2,969
1975 earnings	1,532	1,267	13,651	2,466	1,950	1,859
Number of Obs.	185	260	15,992	429	352	157

Notes: Adapted from Dehejia and Wahba (1999), Table 1. The samples in the first four columns are as described in Dehejia and Wahba (1999). The samples in the last two columns are limited to observations with a propensity score between .1 and .9. Propensity score estimates use all the covariates listed in the table.

Table 3.3.3: Regression estimates of NSW training effects using alternate controls

Specification	Full Samples			P-Score Screened Samples	
	NSW	CPS-1	CPS-3	CPS-1	CPS-3
	(1)	(2)	(3)	(4)	(5)
Raw Difference	1,794 (633)	-8,498 (712)	-635 (657)		
Demographic controls	1,670 (639)	-3,437 (710)	771 (837)	-3,361 (811) [139/497]	890 (884) [154/154]
1975 Earnings	1,750 (632)	-78 (537)	-91 (641)	no obs. [0/0]	166 (644) [183/427]
Demographics, 1975 Earnings	1,636 (638)	623 (558)	1,010 (822)	1,201 (722) [149/357]	1,050 (861) [157/162]
Demographics, 1974 and 1975 Earnings	1,676 (639)	794 (548)	1,369 (809)	1,362 (708) [151/352]	649 (853) [147/157]

Notes: The table reports regression estimates of training effects using the Dehejia-Wahba (1999) data with alternative sets of controls. The demographic controls are age, years of schooling, and dummies for Black, Hispanic, high school dropout, and married.

Standard Errors are reported in parentheses, Observation counts are reported in brackets [treated/control]

textbook discussions of weighting, we are even less sympathetic to weighting than the macroeconomists. The argument for weighting under heteroskedasticity goes roughly like this: suppose you are interested in a linear CEF, $E[\text{Y}_i|\text{X}_i] = \text{X}_i'\beta$. The error term, defined as $e_i \equiv \text{Y}_i - \text{X}_i'\beta$, may be heteroskedastic. That is, the conditional variance function, $E[e_i^2|\text{X}_i]$ need not be constant. In this case, while the population regression function is still equal to $E[\text{X}_i\text{X}_i']^{-1}E[\text{X}_i\text{Y}_i]$, the sample analog is inefficient. A more precise estimator of the linear CEF is weighted least squares, i.e., minimize the sum of squared errors weighted by an estimate of $E[e_i^2|\text{X}_i]^{-1}$.

As noted in Section 3.1.3, an inherently heteroskedastic scenario is the LPM, where Y_i is a dummy variable. Assuming the CEF is in fact linear, as it will be if the model is saturated, then $P[\text{Y}_i = 1|\text{X}_i] = \text{X}_i'\beta$ and therefore $E\left[e_i^2|\text{X}_i\right] = \text{X}_i'\beta\left(1 - \text{X}_i'\beta\right)$, which is obviously a function of X_i. This is an example of model-based heteroskedasticity where in principle, the conditional variance function is easily constructed from estimates of the underlying regression function. The efficient weighted least squares estimator—a special case of generalized least squares (GLS)—is to weight by $\left[\text{X}_i'\beta(1 - \text{X}_i'\beta)\right]^{-1}$. In practice, because the CEF has been assumed to be linear, these weights can be estimated in a first pass by OLS.

There are two reason why we prefer not to weight in this case (though we would use a heteroskedasticity-consistent covariance matrix). First, in practice, the estimate of $E[e_i^2|\text{X}_i]$ may not be very good. If the conditional variance model is a poor approximation and/or the estimates of it are very noisy (in the LPM, this might mean the CEF is not really linear), weighted least squares estimates may have worse finite-sample properties than unweighted estimates. The inferences you draw based on asymptotic theory may therefore be misleading, and the hoped for efficiency gain may not materialize[26]. Second, if the CEF is not linear, the weighted least squares estimator is no more likely to estimate the CEF than is the unweighted estimator. Moreover, the unweighted estimator still estimates something easy to interpret: it estimates the MMSE linear approximation to the population CEF.

Of course, the GLS estimator also provides some sort of approximation, but the nature of this approximation depends on the weights. At a minimum, this makes it harder to compare your results to estimates by other researchers, and opens up additional avenues for specification searches when results depend on weighting. Finally, an old caution comes to mind: "if it ain't broke, don't fix it." The interpretation of the population regression vector is unaffected by heteroskedasticity, so why worry about it? Any efficiency gain from weighting is likely to be modest, and incorrect or poorly estimated weights can do more harm than good.

3.4.2   Limited Dependent Variables and Marginal Effects

Many empirical studies involve variables that take on only a limited number of values. An example is the Angrist and Evans (1998) investigation of the effect of childbearing on female labor supply, discussed in

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[26]Altonji and Segal (1996) discuss this point in a generalized method-of-moments context.

Section 3.4.2 in this chapter and in the chapter on instrumental variables, below. This study is concerned with the causal effects of childbearing on parents' work and earnings.   Because childbearing is likely to be correlated with potential earnings, the study reports instrumental variables estimates based on sibling-sex composition and multiple births, as well as OLS estimates.   Almost every outcome in this study is either binary (like employment status) or non-negative (like hours worked, weeks worked, and earnings). Should the fact that a dependent variable is limited affect empirical practice? Many econometrics textbooks argue that, while OLS is fine for continuous dependent variables, when the outcome of interest is a limited dependent variable (LDV), linear regression models are inappropriate and nonlinear models such as Probit and Tobit are preferred. In contrast, our view of regression as inheriting its legitimacy from the CEF makes LDVness seem less central.

As always, a useful benchmark is a randomized experiment, where regression is simply a treatment-control difference. Consider regressions of various outcome variables on a randomly assigned regressor that indicates one of the treatment groups in the Rand Health Insurance Experiment (HIE; Manning, et al, 1987). In this ambitious experiment, probably the most expensive in American social science, the Rand Corporation set up a small health insurance company that charged no premium. Nearly 6,000 participants in the study were randomly assigned to health insurance plans with different features.

One of the most important features of any insurance plan is the portion of health care costs the insured individual is expected to pay. The HIE randomly assigned individuals to many different plans. One plan provided entirely free care, while the others included various combinations of co-payments, expenditure caps, and deductibles so that patients covered some of their health care costs out-of-pocket. The main purpose of the experiment was to learn whether the use of medical care is sensitive to cost and, if so, whether this affects health.   The HIE results showed that those offered free or low-cost medical care used more of it, but they were not, for the most part, any healthier as a result.   These findings helped pave the way for cost-sensitive health insurance plans and managed care.

Most of the outcomes in the HIE are LDVs. These include dummies indicating whether an experimental subject incurred any medical expenditures or was hospitalized in a given year and non-negative outcomes such as the number of face-to-face doctor visits and gross annual medical expenses (whether paid by patient or insurer). The expenditure variable is zero for about 20 percent of the sample. Results for two of the HIE treatment groups are reproduced in Table 3.4.1, derived from the estimates reported in Table 2 of Manning, *et al.* (1987). Table 3.4.1 shows average outcomes in the free care and individual deductible groups. The latter group faced a deductible of $150 per person or $450 per family per year for outpatient care, after which all costs were covered (There was no charge for inpatient care). The overall sample size in these two groups was a little over 3,000.

To simplify the LDV discussion, suppose that the comparison between free care and deductible plans is

Table 3.4.1: Average outcomes in two of the HIE treatment groups

Plan	Face-to-face visits	Outpatient Expenses (1984$)	Admis-sions	Prob. Any Medical (%)	Prob. Any Inpatient (%)	Total Expenses (1984$)
Free	4.55	340	.128	86.8	10.3	749
	(.168)	(10.9)	(.0070)	(.817)	(.45)	(39)
Individual	3.02	235	.115	72.3	9.6	608
Deductible	(.171)	(11.9)	(.0076)	(1.54)	(.55)	(46)
Deductible	-1.53	-105	-0.013	-14.5	-0.7	-141
minus free	(.240)	(16.1)	(.0103)	(1.74)	(.71)	(60)

Notes: Adapted from Manning (1987), Table 2. All standard errors (shown in parentheses) are corrected for intertemporal and intrafamily correlations. Amounts are in June 1984 dollars. Visits are face-to-face contacts with MD, DO, or other health providers; excludes visits only for radiology, anesthesiology or pathology services. Visits and expenses exclude dental care and outpatient psychotherapy.

the only comparison of interest and that treatment was determined by simple random assignment.[27] Let $D_i = 1$ denote assignment to the deductible group. By virtue of random assignment, the difference in means between those with $D_i = 1$ and $D_i = 0$ identifies the effect of treatment on the treated. As in our earlier discussion of experiments (Chapter 2):

$$E\left[Y_i|D_i = 1\right] - E\left[Y_i|D_i = 0\right] \tag{3.4.1}$$
$$= E\left[Y_{1i}|D_i = 1\right] - E\left[Y_{0i}|D_i = 1\right]$$
$$= E\left[Y_{1i} - Y_{0i}\right]$$

because D_i is independent of potential outcomes. Also, as before, $E\left[Y_i|D_i = 1\right] - E\left[Y_i|D_i = 0\right]$ is the slope coefficient in a regression of Y_i on D_i.

Equation (3.4.1) suggests that the estimation of causal effects in experiments presents no special challenges whether Y_i is binary, non-negative, or continuously distributed. The interpretation of the right-hand side changes for different sorts of dependent variables, but you do not need to *do* anything special to get the average causal effect. For example, one of the HIE outcomes is a dummy denoting any medical expenditure.

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[27] The HIE was considerably more complicated than described here. There were 14 different treatments, including assignment to a prepaid HMO-like service. The experimental design did not use simple random assignment, but rather a more complicated assignment scheme meant to ensure covariate balance acrosss groups.

Since the outcome here is a Bernoulli trial, we have

$$E[Y_{1i} - Y_{0i}] = E[Y_{1i}] - E[Y_{0i}] = P[Y_{1i} = 1] - P[Y_{0i} = 1]. \tag{3.4.2}$$

This relation might affect the language we use to describe the results but not the underlying calculation. In the HIE, for example, comparisons across experimental groups, as on the left hand side of (3.4.1), show that 87 percent of those assigned to the free-care group used at least some care in a given year, while only 72 percent of those assigned to the deductible plan used care. The relatively modest \$150 deductible therefore had a marked effect on use of care. The difference between these two rates, $-.15 (s.e. = .017)$ is an estimate of $E[Y_{1i} - Y_{0i}]$, where Y_i is a dummy indicating any medical expenditure. Because the outcome here is a dummy variable, the average causal effect is also a causal effect on usage rates or probabilities.

Recognizing that the outcome variable here is a probability, suppose instead that you use Probit to fit the CEF in this case. No harm in trying! The Probit model is usually motivated by the assumption that participation is determined by a latent variable, Y_i^*, that satisfies

$$Y_i^* = \beta_0^* + \beta_1^* D_i - \nu_i, \tag{3.4.3}$$

where ν_i is distributed $N(0, \sigma^2)$. Note that this variable cannot be actual medical expenditure since expenditure is non-negative and therefore non-Normal, while Normally distributed variables are continuously distributed on the Real line and can therefore be negative. Given the latent index model,

$$Y_i = 1[Y_i^* > 0],$$

the CEF can be written

$$E[Y_i | D_i] = \Phi\left[\frac{\beta_0^* + \beta_1^* D_i}{\sigma}\right],$$

where $\Phi[\cdot]$ is the Normal CDF. Therefore

$$E[Y_i | D_i] = \Phi\left[\frac{\beta_0^*}{\sigma}\right] + \left\{\Phi\left[\frac{\beta_0^* + \beta_1^*}{\sigma}\right] - \Phi\left[\frac{\beta_0^*}{\sigma}\right]\right\} D_i.$$

This is a linear function of the regressor, D_i, so the slope coefficient in the regression of Y_i on D_i is exactly the difference in Probit fitted values, $\Phi\left[\frac{\beta_0^* + \beta_1^*}{\sigma}\right] - \Phi\left[\frac{\beta_0^*}{\sigma}\right]$. Note, however, that the *Probit Coefficients*, $\frac{\beta_0^*}{\sigma}$ and $\frac{\beta_1^*}{\sigma}$ do not give us the size of effect of D_i on participation until we feed them back into the Normal CDF (though they do have the right sign).

One of the most important outcomes in the HIE is gross medical expenditure, in other words, health care costs. Did subjects who faced a deductible use less care, as measured by the cost? In the HIE, the average difference in expenditures between the deductible and free-care groups was -141 dollars $(s.e. = 60)$, about

19% of the expenditure level in the free-care group. This calculation suggests that making patients pay a portion of costs reduces expenditures quite a bit, though the estimate is not very precise.

Because expenditure outcomes are non-negative random variables, and sometimes equal to zero, their expectation can be written

$$E[Y_i|D_i] = E[Y_i|Y_i > 0, D_i]P[Y_i > 0|D_i].$$

The difference in expenditure outcomes across treatment groups is

$$E[Y_i|D_i = 1] - E[Y_i|D_i = 0] \tag{3.4.4}$$

$$= E[Y_i|Y_i > 0, D_i = 1]P[Y_i > 0|D_i = 1] - E[Y_i|Y_i > 0, D_i = 0]P[Y_i > 0|D_i = 0]$$

$$= \underbrace{\{P[Y_i > 0|D_i = 1] - P[Y_i > 0|D_i = 0]\}}_{\text{participation effect}}E[Y_i|Y_i > 0, D_i = 1]$$

$$+ \underbrace{\{E[Y_i|Y_i > 0, D_i = 1] - E[Y_i|Y_i > 0, D_i = 0]\}}_{\text{COP effect}}P[Y_i > 0|D_i = 0].$$

So the overall difference in average expenditure can be broken up into two parts: the difference in the probability that expenditures are positive (often called a participation effect), and the difference in means conditional on participation, a conditional-on-positive (COP) effect. Again, however, this has no special implications for the estimation of causal effects; equation (3.4.1) remains true: the regression of Y_i on D_i gives the population average treatment effect for expenditures.

Good COP, Bad COP: Conditional-on-positive effects

Because the effect on a non-negative random variable like expenditure has two parts, some applied researchers feel they should look at these parts separately. In fact, many use a "two-part model," where the first part is an evaluation of effect on participation and the second part looks at the COP effects (see, e.g., Duan, *et al.*, 1983 and 1984 for such models applied to the HIE). The first part of (3.4.4) raises no special issues, because, as noted above, the fact that Y_i is a dummy means only that average treatment effects are also differences in probabilities. The problem with the two-part model is that the COP effects do not have a causal interpretation, even in a randomized trial. This is exactly the same selection problem raised in Section 3.2.3, on bad control.

To analyze the COP effect further, write

$$E[Y_i|Y_i > 0, D_i = 1] - E[Y_i|Y_i > 0, D_i = 0] \tag{3.4.5}$$

$$= E[Y_{1i}|Y_{1i} > 0] - E[Y_{0i}|Y_{0i} > 0]$$

$$= \underbrace{E[Y_{1i} - Y_{0i}|Y_{1i} > 0]}_{\text{causal effect}} + \underbrace{\{E[Y_{0i}|Y_{1i} > 0] - E[Y_{0i}|Y_{0i} > 0]\}}_{\text{selection bias}}.$$

This decomposition shows that the COP effect is composed of two terms: a causal effect for the subpopulation

that uses medical care when it is free and the difference in Y_{0i} between those who use medical care when it is free and those who use medical care when they have to pay something.   This second term is a form of selection bias, though it is more subtle than the selection bias in Chapter 2.

Here selection bias arises because the experiment changes the *composition* of the group with positive expenditures.   The $Y_{0i} > 0$ population probably includes some low-cost users who would opt out of care if they had to pay a deductible. In other words, it is larger and probably has lower costs on average than the $Y_{1i} > 0$ group.   The selection bias term is therefore positive, with the result that COP effects are closer to zero than the negative causal effect, $E[Y_{1i}-Y_{0i}|Y_{1i} > 0]$.   This is a version of the bad control problem from Section 3.2.3: in a causal-effects setting, $Y_i > 0$ is an outcome variable and therefore unkosher for conditioning unless the treatment has no effect on the likelihood that Y_i is positive.

One resolution of the non-causality of COP effects relies on censored regression models like Tobit.   These models postulate a latent expenditure outcome for nonparticipants (e.g., Hay and Olsen, 1984).   A traditional Tobit formulation for the expenditure problem stipulates that the observed Y_i is generated by

$$Y_i = 1[Y_i^* > 0]Y_i^*$$

where Y_i^* is a Normally distributed latent expenditure variable that can take on negative values.   Because Y_i^* is not an LDV, Tobit proponents feel comfortable linking this to D_i with a traditional linear model, say, equation (3.4.3).   In this case, β_1^* is the causal effect of D_i on latent expenditure, Y_i^*. This equation is defined for everyone, whether Y_i is positive or not.   There is no COP-style selection problem if we are happy to study effects on Y_i^*.

But we are not happy with effects on Y_i^*. The first problem is that "latent health care expenditure" is a puzzling construct.[28] Health care expenditure really is zero for some people; this is not a statistical artifact or due to some kind of censoring.   So the notion of latent and potentially negative Y_i^* is hard to grasp. There is no data on Y_i^* and there never will be.   A second problem is that the link between the parameter β_1^* in the latent model and causal effects on the observed outcome, Y_i, turns on distributional assumptions about the latent variable.   To establish this link we evaluate the expectation of Y_i given D_i to find

$$E[Y_i|D_i] = \Phi\left[\frac{\beta_0^* + \beta_1^* D_i}{\sigma}\right][\beta_0^* + \beta_1^* D_i] + \sigma\phi\left[\frac{\beta_0^* + \beta_1^* D_i}{\sigma}\right] \tag{3.4.6}$$

where σ is the standard deviation of ν_i (see, e.g. McDonald and Moffitt, 1980).   This expression involves the assumed Normality and homoskedasticity of ν_i and the assumption that Y_i can be represented as $1[Y_i^* > 0]Y_i^*$, as well as the latent coefficients.

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[28] A generalization of Tobit is the sample selection model, where the latent variable determining participation is not the same as the latent expenditure variable. See, e.g., Maddala (1983). The same conceptual problems related to the interpretation of effects on latent variables arise in the sample selection model as with Tobit.

The Tobit CEF provides us with an expression for a treatment effect on observed expenditure. Specifically,

$$E\left[Y_i|D_i=1\right] - E\left[Y_i|D_i=0\right] \tag{3.4.7}$$

$$= \left\{\Phi\left[\frac{\beta_0^* + \beta_1^*}{\sigma}\right][\beta_0^* + \beta_1^*] + \sigma\phi\left[\frac{\beta_0^* + \beta_1^*}{\sigma}\right]\right\} - \left\{\Phi\left[\frac{\beta_0}{\sigma}\right][\beta_0^*] + \sigma\phi\left[\frac{\beta_0^*}{\sigma}\right]\right\}$$

a rather daunting expression. But since the only conditioning variable is a dummy variable, D_i, none of this is necessary for the estimation of $E[Y_i|D_i=1] - E[Y_i|D_i=0]$. The slope coefficient from an OLS regression of Y_i on D_i recovers the CEF difference on the left hand side of (3.4.7) whether or not you adopt a Tobit model to explain the underlying structure.

COP effects are sometimes motivated by a researcher's sense that when the outcome distribution has a mass point - that is, it piles up on particular values like zero - or a heavily skewed distribution, or both, then an analysis of effects on averages misses something. Analyses of effects on averages indeed miss some things, like changes in the probability of specific values, or a shift in quantiles away from the median. But why not look at these distribution effects directly? A sensible alternative to COP effects looks directly at effects on distributions or quantiles. Distribution outcomes include the likelihood that annual medical expenditures exceed zero, 100 dollars, 200 dollars, and so on. This puts $1[Y_i > c]$ for different choices of c on the left-hand side of the regression of interest. Econometrically, these outcomes are all in the category of equation (3.4.2). The idea of looking directly at distribution effects with linear probability models is illustrated by Angrist (2001), in an analysis of the effects of childbearing on hours worked. Alternately, if quantiles provide a focal point, we can use quantile regression to model them. Chapter 7 discusses this idea in detail.

Do Tobit-type latent-variable models ever make sense? Yes, if the data you are working with are truly censored. True censoring means the latent variable has an empirical counterpart that is the outcome of primary interest. A leading example from labor economics is CPS earnings data, which topcodes (censors) very high values of earnings to protect respondent confidentiality. Typically, we're interested in the causal effect of schooling on earnings as it appears on respondents' tax returns, not their CPS-topcoded earnings. Chamberlain (1994) shows that in some years, CPS topcoding reduces the measured returns to schooling considerably, and proposes an adjustment for censoring based on a Tobit-style adaptation of quantile regression. The use of quantile regression to model censored data is also discussed in Chapter 7.[29]

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[29] We should note that our favorite regression example - a regression of log wages on schooling - may have a COP problem since the sample of log wages naturally omits those with zero earnings. This leads to COP-style selection bias if education affects the probability of working. In practice, therefore, we focus on samples of prime-age males where participation rates are high and reasonably stable across schooling groups (e.g., white men aged 40-49 in Figure 3.1.1).

Covariates lead to nonlinearity

True censoring as with the CPS topcode is rare, a fact that leaves limited scope for constructive applications of Tobit-type models in applied work. At this point, however, we have to hedge a bit. Part of the neatness in the discussion of experiments comes from the fact that $E[\text{Y}_i|\text{D}_i]$ is necessarily a linear function of D_i so that regression and the CEF are one and the same. In fact, this CEF is linear for any function of Y_i, including the distribution indicators, $1[\text{Y}_i > c]$. In practice, of course, the explanatory variable of interest isn't always a dummy, and there are usually additional covariates in the CEF, in which case, $E[\text{Y}_i|\text{X}_i,\text{D}_i]$ is almost certainly nonlinear for LDVs. Intuitively, as predicted means get close to the dependent variable boundaries, say because some covariate cells are close to the boundaries, the derivatives of the CEF for LDVs get smaller (think, for example, of the how the Normal CDF flattens at extreme values).

The upshot is that in LDV models with covariates, regression need not fit the CEF perfectly. It remains true, however, that the underlying CEF has a causal interpretation if the CIA holds. And if the CEF has a causal interpretation, it seems fair to say that regression has a causal interpretation as well, because it still provides the MMSE approximation to the CEF. Moreover, if the model for covariates is saturated, then regression also estimates a weighted average treatment effect similar to (3.3.1) and (3.3.3). Likewise, if the regressor of interest is multi-valued or continuous, we get a weighted average derivative, as described by the formulas in subsection 3.3.1.

And yet, we don't often have enough data for the saturated-covariate regression specification to be very attractive. Regression will therefore miss some features of the CEF. For one thing, it may generate fitted values outside the LDV boundaries. This fact bothers some researchers and has certainly generated a lot of bad press for the linear probability model. One attractive feature of nonlinear models like Probit and Tobit is that they produce CEFs that respect LDV boundaries. In particular, Probit fitted values are always between zero and one, while Tobit fitted values are positive (this is not obvious from equation 3.4.6). We might therefore prefer nonlinear models on simple curve-fitting grounds.

Point conceded. It's important to emphasize, however, that the output from nonlinear models must be converted into *marginal effects* to be useful. Marginal effects are the (average) changes in CEF implied by a nonlinear model. Without marginal effects, it's hard to talk about the impact on observed dependent variables. Continuing to assume the regressor of interest is D_i, population average marginal effects can be constructed either by differencing

$$E\{E[\text{Y}_i|\text{X}_i, \text{D}_i = 1] - E[\text{Y}_i|\text{X}_i, \text{D}_i = 0]\},$$

or by differentiation: $E\left\{\frac{\partial E[\text{Y}_i|\text{X}_i,\text{D}_i]}{\partial \text{D}_i}\right\}$. Most people use derivatives when dealing with continuous or multi-valued regressors as well.

How close do OLS regression estimates come to the marginal effects induced by a nonlinear model like

Probit or Tobit? We first derive the marginal effects, and then show an empirical example. The Probit CEF for a model with covariates is

$$E[\text{Y}_i|\text{X}_i, \text{D}_i] = \Phi\left[\frac{\text{X}_i'\beta_0^* + \beta_1^*\text{D}_i}{\sigma}\right].$$

The average finite difference is therefore

$$E\left\{\Phi\left[\frac{\text{X}_i'\beta_0^* + \beta_1^*}{\sigma}\right] - \Phi\left[\frac{\text{X}_i'\beta_0^*}{\sigma}\right]\right\}. \tag{3.4.8}$$

In practice, this can also be approximated by the average derivative,

$$E\left\{\Phi\left[\frac{\text{X}_i'\beta_0^* + \beta_1^*\text{D}_i}{\sigma}\right]\right\} \cdot (\beta_1^*/\sigma)$$

(Stata computes marginal effects both ways but defaults to (3.4.8) for dummy regressors).

Similarly, generalizing equation (3.4.6) to a model with covariates, we have

$$E[\text{Y}_i|\text{X}_i, \text{D}_i] = \Phi\left[\frac{\text{X}_i'\beta_0^* + \beta_1^*\text{D}_i}{\sigma}\right]\left[\text{X}_i'\beta_0^* + \beta_1^*\text{D}_i\right] + \sigma\phi\left[\frac{\text{X}_i'\beta_0^* + \beta_1^*\text{D}_i}{\sigma}\right]$$

for a non-negative LDV. Tobit marginal effects are almost always cast in terms of the average derivative, which can be shown to be the surprisingly simple expression

$$E\left\{\phi\left[\frac{\text{X}_i'\beta_0^* + \beta_1^*\text{D}_i}{\sigma}\right]\right\} \cdot \beta_1^*. \tag{3.4.9}$$

See, e.g., Wooldridge (2006). One immediate implication of (3.4.9) is that the Tobit coefficient, β_1^* is always too big relative to the effect of D_i on Y_i. Intuitively, this is because - given the linear model for latent Y_i^* - the latent outcome always changes when D_i switches on or off. But real Y_i need not change: for many people, it's zero either way.

Table 3.4.2 compares regression and nonlinear marginal effects for a regression of female employment and hours of work, both LDVs, on measures of fertility. The estimates were constructed using one of the 1980 Census samples used by Angrist and Evans (1998) This sample includes married women aged 21-35 with at least two children. The childbearing variables consist of either a dummy indicating additional childbearing beyond two, or the total number of births. The covariates include linear terms in mothers' age, age at first birth, race dummies (black and Hispanic), and mother's education (dummies for high school graduates, some college, and college graduates). The covariate model is not saturated, rather there are linear effects and no interactions, so the underlying CEF in this example is surely nonlinear.

Probit marginal effects for the effect of a dummy variable indicating more than two children are indistinguishable from OLS estimates of the same relation. This can be seen in columns 2, 3, and 4 of Table 3.4.2,

the first row of which compares the estimates from different methods for the full 1980 sample.   The OLS estimate of the effect of a third child is -.162, while the corresponding Probit marginal effects are -.163 and -.162.   These were estimated using (3.4.8) in the first case and

$$E \left\{ \Phi \left[\frac{X_i' \beta_0^* + \beta_1^*}{\sigma} \right] - \Phi \left[\frac{X_i' \beta_0^*}{\sigma} \right] | D_i = 1 \right\}$$

in the second (hence, a marginal effect on the treated).

Tobit marginal effects for the relation between fertility and hours worked are also quite close to the corresponding OLS estimates, though not indistinguishable.   This can be seen in columns 5 and 6.   Compare, for example, the Tobit estimates of -6.56 and -5.87 with the OLS estimate of -5.92 in column 2.   Although one Tobit estimate is 10 percent larger in absolute value, this seems unlikely to be of substantive importance. The remaining columns of the table compare OLS to marginal effects for an ordinal childbearing variable instead of a dummy.   These calculations all use derivatives to compute marginal effects (labeled MFX). Here too, the OLS and nonlinear marginal effects estimates are similar for both Probit and Tobit.

It is sometimes said that Probit models can be expected to generated marginal effects close to OLS when the fitted values are close to .5 because the nonlinear CEF is roughly linear in the middle.   We therefore replicated the comparison of OLS and marginal effects in a subsample with relatively high average employment rates, non-white women over 30 who attended college and whose first birth was before age 20. Although the average employment rate is 83 percent in this group, the OLS estimates and marginal effects are again similar.

Table 3.4.2: Comparison of alternative estimates of the effect of childbearing on LDVs

		More than two children					Number of children			
		OLS	Probit		Tobit		OLS	Probit MFX	Tobit MFX	
Dependent variable	Mean	OLS	Avg effect, full sample	Avg effect on treated	Avg effect, full sample	Avg effect on treated	OLS	Avg effect, full sample	Avg effect, full sample	Avg effect on treated
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Panel A: Full Sample										
Employment	.528	-.162	-.163	-.162	-	-	-.113	-.114	-	-
	(.499)	(.002)	(.002)	(.002)			(.001)	(.001)		
Hours worked	16.7	-5.92	-	-	-6.56	-5.87	-4.07	-	-4.66	-4.23
	(18.3)	(.074)			(.081)	(.073)	(.047)		(.054)	(.049)
Panel B: Non-white College Attendees over 30, first birth before age 20										
Employment	.832	-.061	-.064	-.070	-	-	-.054	-.048	-	-
	(.374)	(.028)	(.028)	(.031)			(.016)	(.013)		
Hours worked	30.8	-4.69	-	-	-4.97	-4.90	-2.83	-	-3.20	-3.15
	(16.0)	(1.18)			(1.33)	(1.31)	(.645)		(.670)	(.659)

Notes: The table reports OLS estimates, average treatment effects, and marginal effects (MFX) for the effect of childbearing on mothers' labor supply. The sample in Panel A includes 254,654 observations and is the same as the married-women-1980-Census sample used by Angrist and Evans (1998). Covariates include age, age at first birth, and dummies for boys at first and second birth. The sample in Panel B includes 746 nonwhites with at least some college aged over 30 whose first birth was before age 20. Standard errors are reported in parentheses in column 1. Standard deviations are shown in parentheses in other columns. The sample used to estimate average effects on the treated includes women with more than two children.

The upshot of this discussion is that while a nonlinear model may fit the CEF for LDVs more closely than a linear model, when it comes to marginal effects this probably matters little. This optimistic conclusion is not a theorem, but as in the empirical example here, it seems to be fairly robustly true.

Why then, should we bother with nonlinear models and marginal effects? One answer is that the marginal effects are easy enough to compute now that they are automated in packages like Stata. But there are a number of decisions to make along the way (e.g., the weighting scheme, derivatives versus finite differences) while OLS is standardized. Nonlinear life also promises to get considerably more complicated when we start to think about IV and panel data. Finally, extra complexity comes into the inference step as well, since we need standard errors for marginal effects. The principle of Occam's razor advises, "Entities should not be multiplied unnecessarily." In this spirit, we quote our former teacher, Angus Deaton (1997), pondering the nonlinear regression function generated by Tobit-type models:

> Absent knowledge of F [the distribution of the errors], this regression function does not even identify the β's [Tobit coefficients] - see Powell (1989) - but more fundamentally, we should ask how it has come about that we have to deal with such an awkward, difficult, and non-robust object.

3.4.3 Why is Regression Called Regression and What Does Regression-to-the-mean Mean?

The term regression originates with Francis Galton's (1886) study of height. Galton, who worked with samples of roughly-normally-distributed data on parents and children, noted that the CEF of a child's height given his parents' height is linear, with parameters given by the bivariate regression slope and intercept. Since height is stationary (its distribution is not changing [much] over time), the bivariate regression slope is also the correlation coefficient, i.e., between zero and one.

The single regressor in Galton's set-up, x_i, is average parent height and the dependent variable, Y_i, is the height the of adult children. The regression slope coefficient, as always, is $\beta_1 = \frac{Cov(\mathrm{Y}_i, x_i)}{V(x_i)}$, and the intercept is $\alpha = E[\mathrm{Y}_i] - \beta_1 E[\mathrm{X}_i]$. But because height is not changing across generations, the mean and variance of Y_i and x_i are the same. Therefore,

$$\begin{aligned} \beta_1 &= \frac{Cov(\mathrm{Y}_i, x_i)}{V(x_i)} = \frac{Cov(\mathrm{Y}_i, x_i)}{\sqrt{V(x_i)}\sqrt{V(\mathrm{Y}_i)}} = \rho_{xy} \\ \alpha &= E[\mathrm{Y}_i] - \beta_1 E[\mathrm{X}_i] = \mu(1 - \beta_1) = \mu(1 - \rho_{xy}) \end{aligned}$$

where ρ_{xy} is the intergenerational correlation coefficient in height and $\mu = E[\mathrm{Y}_i] = E[\mathrm{X}_i]$ is population average height. From this we get the linear CEF

$$E[\mathrm{Y}_i | x_i] = \mu(1 - \rho_{xy}) + \rho_{xy} x_i,$$

so the height of a child given his parents' height is therefore a weighted average of his parents' height and the population average height. The child of tall parents will therefore not be as tall as they are, on average. Likewise, for the short. To be specific, Pischke, who is 6' 3", can expect his children to be tall, though not as tall as he is. Thankfully, however, Angrist, who is 5'6", can expect his children to be taller than he is. Galton called this property, "regression toward mediocrity in hereditary stature." Today, we call this "regression to the mean."

Galton, who was Charles Darwin's cousin, is also remembered for having founded the Eugenics Society, dedicated to breeding better people. Indeed, his interest in regression came largely from this quest. We conclude from this that the value of scientific ideas should not be judged by their author's politics.

Galton does not seem to have shown much interest in multiple regression, our chief concern in this chapter. Indeed, the regressions in Galton's work are mechanical properties of distributions of stationary random variables, almost identities, and certainly not causal. Galton, would have said so himself because he objected to the Lamarckian idea (later promoted in Stalin's Russia) that acquired traits could be inherited.

The idea that regression can be used for statistical control satisfyingly originates in an inquiry into the determinants of poverty rates by George Udny Yule (1899). Yule, a statistician and student of Karl Pearson's (Pearson was Galton's protégé) realized that Galton's regression coefficient could be extended to multiple variables by solving the least squares normal equations that had been derived long before by Legendre and Gauss. Yule's (1899) paper appears to be the first publication containing multivariate regression estimates. His model links changes in poverty rates in an area to changes in the administration of the English Poor Laws, while controlling for population growth and the age distribution in the area. He was particularly interested in whether out-relief, the practice of providing income support for poor people without requiring them to move to the poorhouse, did not itself contribute to higher poverty rates. This is a well-defined causal question of a sort that still occupies us today.[30]

Finally, we note that the history of regression is beautifully detailed in the book by Steven Stigler (1986). Stigler is a famous statistician at the University of Chicago, but not quite as famous as his father, the economist and Nobel laureate, George Stigler.

3.5 Appendix: Derivation of the average derivative formula

Begin with the regression of Y_i on S_i :

$$\frac{Cov(\mathrm{Y}_i, \mathrm{S}_i)}{V(\mathrm{S}_i)} = \frac{E[h(\mathrm{S}_i)(\mathrm{S}_i - E[\mathrm{S}_i])]}{E[\mathrm{S}_i(\mathrm{S}_i - E[\mathrm{S}_i])]}.$$

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[30]Yule's first applied paper on the poor laws was published in 1895 in the *Economic Journal*, where Pischke is proud to serve as co-editor. The theory of multiple regression that goes along with this appears in Yule (1897).

Let $\kappa_{-\infty} = \lim_{t \to -\infty} h(t)$. By the fundamental theorem of calculus, we have:

$$h(\mathrm{s}_i) = \kappa_{-\infty} + \int_{-\infty}^{\mathrm{s}_i} h'(t)\, dt.$$

Substituting for $h(\mathrm{s}_i)$, the numerator becomes

$$E[h(\mathrm{s}_i)(\mathrm{s}_i - E[\mathrm{s}_i])] = \int_{-\infty}^{+\infty} \int_{-\infty}^{s} h'(t)(s - E[\mathrm{s}_i]) g(s)\, dt\, ds$$

where $g(s)$ is the density of s_i at s. Reversing the order of integration, we have

$$E[h(\mathrm{s}_i)(\mathrm{s}_i - E[\mathrm{s}_i])] = \int_{-\infty}^{+\infty} h'(t) \int_{t}^{+\infty} (s - E[\mathrm{s}_i]) g(s)\, ds\, dt.$$

The inner integral is easily seen to be equal to $\mu_t \equiv \{E[\mathrm{s}_i|\mathrm{s}_i \geq t] - E[\mathrm{s}_i|\mathrm{s}_i < t]\}\{P(\mathrm{s}_i \geq t)[1 - P(\mathrm{s}_i \geq t)]\}$, which is clearly non-negative. Setting $\mathrm{s}_i = \mathrm{Y}_i$, the denominator can similarly be shown to be the integral of these weights. We therefore have a weighted average derivative representation of the bivariate regression coefficient, $\frac{Cov(\mathrm{Y}_i, \mathrm{s}_i)}{V(\mathrm{s}_i)}$, equation (3.3.8) in the text. A similar formula for a regression with covariates, X_i, is derived in the appendix to Angrist and Krueger (1999).