

Lecture Notes on Treatment Effects

(or *Completely Innocuous Econometrics*)

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Abstract

These are lecture notes that I use to accompany my MPhil teaching in Econometrics at Oxford. For more details, along with short videos to accompany these notes, see the course website: treatment-effects.com. If you spot any typos, send me a [Github pull request](#) or an email: francis.ditraglia@economics.ox.ac.uk.

Contents

1	Introduction	3
1.1	What are these notes about?	3
1.2	The Fundamental Problem of Causal Inference	4
1.3	The Potential Outcomes Framework	4
1.4	Populations, Observables, and Random Variables	5
1.5	Identification Versus Estimation	7
1.6	Our goal: identify the Average Treatment Effect	7
1.7	Quantile Treatment Effects	9
1.8	The problem to overcome: selection bias	9
1.9	Appendix: Proofs and Probability Review	11
2	Conditional Independence	14
2.1	Intuition and Notation	14
2.2	Independence versus Mean Independence	15
2.3	Randomize treatments to eliminate selection bias.	16
2.4	The Axioms of Conditional Independence	17
2.5	More Properties of Conditional Independence	19
2.6	Appendix: Technical Details	20
3	Selection on Observables	22
3.1	Does education cause political participation?	22
3.2	Selection on Observables and Overlap	23
3.3	Identification by Regression Adjustment	24
3.4	Estimation by Regression Adjustment	25
3.5	Identification by Propensity Score Weighting	25
3.6	Estimation by Propensity Score Weighting	27
3.7	Regression Adjustment versus Propensity Score Weighting	27
3.8	Don't condition on an intermediate outcome!	28
4	The Local Average Treatment Effects (LATE) Model	32
4.1	Instrumental Variables with Homogeneous Effects	32

4.2	Non-compliance and the Intent-to-Treat Effect	33
4.3	Compliers, Defiers, and Friends	35
4.4	The Local Average Treatment Effects Model	36
4.5	Who are the compliers? Why should we care?	40
4.6	LATE Appendix: Proofs	41
5	Regression Discontinuity	45
5.1	The Sharp Regression Discontinuity Design	46
5.2	The Fuzzy Regression Discontinuity Design	49
6	Difference-in-differences	54
6.1	Adding a Time Dimension	54
6.2	The Before-and-After Design	56
6.3	The Difference-in-Differences Design	58
6.4	A Regression Interpretation of DiD	60
6.5	What if the treatment is anticipated?	61

Chapter 1

Introduction

In this chapter we set the stage for the material to come, introducing the fundamental problem of causal inference, developing notation for later use, and reviewing some important facts concerning random variables.

1.1 What are these notes about?

Will earning an MPhil in Economics from Oxford increase your lifetime earnings? Does eating bacon sandwiches cause cancer? Does watching Fox News cause people to vote Republican? Will owning a dog increase your lifespan? Each of these questions concerns the **causal effect** of a **treatment** D on an **outcome** Y . The terminology “treatment” evokes a medical trial, but we will use the term much more broadly to refer to any variable D whose causal effect we hope to learn. For us, a treatment could be earning an MPhil, eating bacon sandwiches, watching Fox news, or owning a dog. These notes will focus on the case in which D is *binary*: either zero or one. If you have $D = 1$ we say that you are **treated**; if $D = 0$ we say that you are **untreated**. We will be particularly interested in methods for learning causal effects when the treatment variable is not randomly assigned, as would be the case in an **observational** rather than experimental study. So far as I know, no experiment has yet been carried out in which subjects are randomly compelled to be dog owners or forced to watch Fox News. Nonetheless papers have been written and published that attempt to estimate the causal effects of both of these treatments. We will study methods and assumptions under which observational data can be used to recover causal effects. We will also consider experiments in which subjects may fail to *comply* with their assigned treatments. In this case, the treatments that subjects actually receive are no longer randomly assigned, even if the treatments that they have been offered actually were.

1.2 The Fundamental Problem of Causal Inference

The fundamental problem of causal inference is that we can never observe a person’s **counterfactual** outcome. In other words, we can never know what her outcome *would have been* if her treatment *had been different*. After finishing her undergraduate degree, Alice earned an MPhil in Economics at Oxford. She now makes £75,000 per year. Would she still have earned as much if she had gone straight to work after finishing her undergraduate degree? Barry was a vegetarian so he never ate bacon sandwiches. He lived to the ripe old age of 90 and died in a hang-gliding accident, never having developed cancer. If he had eaten bacon sandwiches every day, would he have died of cancer at the age of 60 instead? Donald watches Fox News 10 hours a day and always votes for the Republican candidate. If he hadn’t watched Fox News, would he instead vote for the Democrats?

A counterfactual is a **within person** comparison: it asks how a given person’s outcome would have been different if her treatment had been different. Because we can never observe the same person in two different treatment states, we can never actually make this comparison. You may be wondering about a before-and-after comparison. For example, what if we looked at Alice’s wage immediately before she earned the MPhil and then immediately afterwards. Tracking the same person over time can be an extremely helpful way to untangle cause-and-effect, as you may have gathered from your exposure to panel data methods. It cannot, however, solve the fundamental problem of causal inference: comparing Alice’s wages at two *different* points in time is not the same as comparing her wage at the *same* point in time across two “parallel universes,” one in which she went straight to work and another in which she went to Oxford. Most people’s income increases as they gain additional experience, for example. Comparing Alice’s income before and after might confuse the effect of more experience in the labor force with the effect of earning an MPhil in Economics. Or perhaps Alice started the MPhil during an economic boom and finished during a severe downturn. If so, the fact that her income fell after the MPhil would tell us little of value: perhaps it would have fallen by *more* without the degree. Because the idealized within person comparison is impossible, we will need to develop methods and assumptions that allow us to substitute a **between-person** comparison.

1.3 The Potential Outcomes Framework

In order to study causal effects we need a framework that allows us to formally define them and manipulate them mathematically. Following the bulk of the treatment effects literature, we will adopt the **potential outcomes framework**, also known as the Rubin Causal Model (RCM). With each person i we associate a pair of **potential outcomes** (y_{i0}, y_{i1}) . These are precisely the counterfactual outcomes that I discussed in the pre-

ceding section. Suppose, for example, that Alice is person i . Then y_{i0} is her wage if she doesn't earn the MPhil and y_{i1} is her wage if she does. Even though we can never observe both y_{i0} and y_{i1} for the same person, we can still *imagine* that there is a fact of the matter regarding what Alice's wage would have been in a parallel universe where her treatment had been different. Using this notation, $(y_{i1} - y_{i0})$ is the causal effect *for Alice* of earning the Oxford MPhil. This need not be the same as the causal effect for Bob of earning an Oxford MPhil, or indeed the same as the causal effect of anyone else. In other words, we will allow for the possibility that treatment effects are **heterogeneous**.

While we never observe both y_{i0} and y_{i1} , we always observe one of them. If Alice is treated then we observe y_{i1} ; otherwise we observe y_{i0} . We can express this as follows

$$y_i = (1 - d_i)y_{i0} + d_iy_{i1} = y_{i0} + d_i(y_{i1} - y_{i0}) \quad (1.1)$$

where y_i is person i 's **observed outcome** and d_i is an indicator that equals one if she was treated and zero otherwise. Implicit in this equation and the potential outcomes notation that we have adopted is a very important assumption that we will maintain throughout these notes: the **stable unit treatment value assumption** (SUTVA). This requires that Alice's outcome depends only on her own treatment and not the treatments of anyone else. SUTVA is a strong assumption and it is easy to think of settings where it doesn't hold. For example, if Alice gets a flu vaccine this makes Bob less likely to get the flu regardless of whether he was vaccinated. Finding ways to relax the SUTVA assumption constitutes a very active area of research in the treatment effects literature.

1.4 Populations, Observables, and Random Variables

The first step of any causal analysis is to specify the **population of interest**. Suppose that we hope to learn the causal effect of watching Fox News on voting behavior. Whose voting behavior are we interested in? All US voters? Swing voters? Often the choice of population is dictated by circumstance. Perhaps we have access to a fantastic dataset on Pennsylvania voters but no information about voters from other states. If so, the causal claims we can make will necessarily be limited to Pennsylvania: the effect of Fox News could be markedly different, say, in Florida.

These notes assume that we have already specified a population of interest and observed a random sample from it. If our population is Pennsylvania voters, this assumes that we have observed a representative sample of n voters from the state. But what, precisely, do we observe? As discussed in the previous section, we can only observe *one* of a person's potential outcomes (y_{i0}, y_{i1}) , namely the one that corresponds to her treatment d_i , as shown in (1.1). At a bare minimum, we will always assume that both y_i and d_i are observed for each person i in our sample. Most of the methods we describe below will

in fact rely on observing some *additional* information \mathbf{w}_i . For this reason, I will refer to (y_i, d_i, \mathbf{w}_i) as the **observables** for person i .

Throughout this section and the preceding one I have used lowercase letters: y_i rather than Y_i and d_i rather than D_i , for example. I did this to emphasize that we are talking about specific values for a particular person. There is, in principle, nothing random about Alice’s treatment, her observed outcome, or her potential outcomes. Randomness enters only when we view her as merely one member of a *population* from which we will draw a random sample. From this point onwards, we will stop thinking about the values for a particular person and instead think about random variables that represent the notion of *randomly drawing someone* from the population of interest.

The idea is as follows. Suppose that 35% of voters in Pennsylvania watch Fox News ($d_i = 1$). Then if I randomly sample a single voter, there is a 35% chance that she watches Fox News. We can represent this as a *random variable* D with a Bernoulli(0.35) distribution. Similarly, if we knew the values of y_i and \mathbf{w}_i for every voter in Pennsylvania, we could construct random variables Y and \mathbf{W} that represent the idea of randomly selecting a voter and observing her values of y_i and \mathbf{w}_i . Using this abstraction, we will view the observables (y_i, d_i, \mathbf{w}_i) for any given person a *realization* from the joint distribution of a collection of random variables (Y, D, \mathbf{W}) . The thought experiment is that we reach into the state of Pennsylvania, pull out a voter at random, and observe (y_i, d_i, \mathbf{w}_i) . Viewed in this way, knowing the values of (y_i, d_i, \mathbf{w}_i) for everyone in the population is the same thing as knowing the joint distribution of (Y, D, \mathbf{W}) .

Although we can never actually observe the pair (y_{i0}, y_{i1}) for the same person, we can still *imagine* reaching into the state of Pennsylvania and learning $(y_{i0}, y_{i1}, d_i, \mathbf{w}_i)$ for a particular person. As above, we can represent this idea using a collection of random variables: $(Y_0, Y_1, D, \mathbf{W})$. Knowing $(y_{i0}, y_{i1}, d_i, \mathbf{w}_i)$ for everyone in the population would be equivalent to knowing the joint distribution of $(Y_0, Y_1, D, \mathbf{W})$. Because these random variables are constructed from the values for each individual in the population, the relationship from (1.1) continues to apply, that is

$$Y = (1 - D)Y_0 + DY_1 = Y_0 + D(Y_1 - Y_0). \quad (1.2)$$

Equation 1.2 shows that knowledge of the joint distribution of $(Y_0, Y_1, D, \mathbf{W})$ implies knowledge of the joint distribution of (Y, D, \mathbf{W}) , because Y is a function of (Y_0, Y_1, D) . The converse, however, is false: knowledge of a person’s observed outcome and her treatment does not allow us to reconstruct both of her potential outcomes.

1.5 Identification Versus Estimation

These notes mainly focus on the problem of **identifying** causal effects rather than that of estimating them. Suppose that we know the joint distribution of (Y, D, \mathbf{W}) and hope to learn the value of some quantity θ in our population of interest. As explained in the preceding section, knowing the distribution of (Y, D, \mathbf{W}) is the same as knowing the values of (y_i, d_i, \mathbf{w}_i) for everyone in the population. If this knowledge would be sufficient to uniquely pin down θ , then we say that θ is **identified**; otherwise we say that it is **unidentified**.¹ The challenge of identifying causal effects is that we observe not the joint distribution of potential outcomes (Y_0, Y_1) but only that of (Y, D, \mathbf{W}) . Our identification question is whether this observed information, combined with appropriate assumptions, will allow us determine whether D causes Y .

Identification is about populations rather than samples. Estimation, on the other hand, asks how we can use a sample of observed data to produce a “best guess” of some quantity of interest θ . In the simplest case, we assume that the researcher observes a collection of n iid draws (Y_i, D_i, \mathbf{W}_i) from the population and ask how this information can be used to construct an estimator $\hat{\theta}$ of θ with desirable properties. These notes mainly focus on identification because estimation is meaningless without it: if there is no way to learn the causal effect of D on Y from knowledge of (y_i, d_i, \mathbf{w}_i) for *everyone* in the population, there is no way to estimate it using a random sample from this population.

1.6 Our goal: identify the Average Treatment Effect

When treatment effects are heterogeneous, every person in the population could have her own, unique causal effect: $(y_{i1} - y_{i0})$. Collecting the individual treatment effects for each person in our population of interest gives rise to a *distribution* of causal effects. Using the random variables defined above, we can represent this distribution using the random variable $(Y_1 - Y_0)$. If $(Y_1 - Y_0)$ were simply a constant, i.e. if treatment effects were **homogeneous**, asking whether D causes Y would be the same thing as asking if $(Y_1 - Y_0) = 0$. The sign and magnitude of $(Y_1 - Y_0)$ would then tell us the direction and importance of the effect. When treatment effects are heterogeneous, however, the yes-or-no question “does D cause Y ?” no longer makes sense. Watching Fox News will not make Bernie Sanders vote Republican, but it might still affect the average swing voter in western Pennsylvania, for example. Faced with effects that vary across people, the natural question is “how do they vary?” In other words, what can we say about the *distribution* of $(Y_1 - Y_0)$? If we could learn the distribution of $(Y_1 - Y_0)$ across the population, we

¹Notice the use of the word *sufficient* in the definition of identification. Saying that θ is identified doesn’t mean that knowing the joint distribution of (Y, D, \mathbf{W}) is *necessary* to uniquely pin down θ . For example, uniquely determining the vector of slope coefficients from a regression of Y on (D, \mathbf{W}) would only require us to know the means, covariances, and variances of these random variables.

could answer a variety of interesting questions. For example: “what fraction of people benefit from this treatment?” or “what is the variance of treatment effects?”

Unfortunately it is impossible to learn the distribution of treatment effects. As we discussed above, the fundamental problem of causal inference is that we can never observe both y_{i1} and y_{i0} for the same person. For this reason, there is no way to identify the joint distribution of (Y_0, Y_1) . If we want to determine the correlation between height and weight, we need observations of both variables for *the same people*. So too, identifying the joint distribution between (Y_0, Y_1) would require observations of both potential outcomes for the same people. Because we observe Y_0 for a subset of the population and Y_1 for another subset, there is at least the possibility that we could learn the *marginal* distributions of Y_0 and Y_1 . What we can never learn is the *dependence* between them.

This problem severely limits our ability to characterize the distribution of $(Y_1 - Y_0)$. Suppose, for example, that we wanted to determine $\text{Var}(Y_1 - Y_0)$. By the formula for the variance of a difference,

$$\text{Var}(Y_1 - Y_0) = \text{Var}(Y_0) + \text{Var}(Y_1) - 2\text{Cov}(Y_0, Y_1).$$

Because it depends on a feature of the joint distribution of (Y_0, Y_1) —namely the covariance—the variance of the distribution of treatment effects cannot be identified. If we were willing to assume that Y_0 and Y_1 are uncorrelated, then we could indeed identify $\text{Var}(Y_1 - Y_0)$ based on knowledge of $\text{Var}(Y_0)$ and $\text{Var}(Y_1)$. In most examples, however, this assumption is untenable. Consider the problem of identifying the returns to an Oxford MPhil in Economics. More than likely, people who would earn a higher than average wage without the MPhil (high Y_0) would *also* earn a higher than average wage *with* an MPhil (high Y_1), implying a positive correlation between Y_0 and Y_1 .

It seems as though we have reached an impasse. How can we say anything useful about $(Y_1 - Y_0)$ without knowledge of the joint distribution of (Y_0, Y_1) ? Recall a fundamental property of expectation: *linearity*. The expectation of a sum equals the sum of the expectations, and the expectation of a difference equals the difference of expectations. Thus, taking expectations of both sides

$$\mathbb{E}[Y_1 - Y_0] = \mathbb{E}[Y_1] - \mathbb{E}[Y_0].$$

We call $\mathbb{E}[Y_1 - Y_0]$ the **average treatment effect** and abbreviate it ATE. The ATE measures how large the individual treatment effects ($y_{i1} - y_{i0}$) are *on average* across everyone in the population. If the ATE is positive, then the treatment is beneficial on average; if it is negative, then the treatment is harmful on average. If the ATE is zero, then the treatment has no effect on average. The primary goal of the treatment effects literature is to identify the ATE or, failing that, at least an average treatment for some

subset of the population. Undeniably the ATE is a valuable summary of $(Y_1 - Y_0)$, but it sweeps many important questions under the rug. What fraction of people would be *harmed* by the treatment? Is the treatment effect highly variable, or very similar for nearly everyone? We would love to be able to answer these questions, but unfortunately we cannot. The ATE thus represents not an ideal measure of the effect of D on Y , but the *best we can manage* given the fundamental problem of causal inference.

1.7 Quantile Treatment Effects

If you have studied quantile regression, you may have encountered the term *quantile treatment effect*. Don't let the name fool you: the fact that this quantity is called a treatment effect does not mean that it has a genuine causal interpretation. Let Q_0 be the quantile function of Y_0 and Q_1 be the quantile function of Y_1 . Then $Q_0(0.5)$ is the median of Y_0 while $Q_1(0.5)$ is the median of Y_1 . Both of these quantities are identified from the marginal distributions of the potential outcomes. Indeed, for any quantile τ , both $Q_0(\tau)$ and $Q_1(\tau)$ are identified from these marginal distributions. The difference $\delta(\tau) \equiv Q_1(\tau) - Q_0(\tau)$ is typically called the **quantile treatment effect** of D on Y . Suppose that Alice's potential outcome without treatment y_{i0} falls at the τ th quantile of the distribution of Y_0 . In other words suppose that $\tau \times 100\%$ of people have a lower value of Y_0 than Alice, and $(1 - \tau) \times 100\%$ have a higher value of Y_0 . Then $\delta(\tau)$ tells us how much higher Alice's value of y_{i1} would *need to be* in order for her to fall at the τ th quantile of the distribution of Y_1 as well. Without further assumptions, $\delta(\tau)$ lacks a causal interpretation. Giving it one requires the so-called **rank invariance** assumption. This condition requires that if Alice occupies the τ th quantile of the Y_0 distribution, then she also occupies the τ th quantile of the Y_1 distribution. Under rank invariance, $\delta(\tau)$ is the causal effect of D on Y for a person who *would have fallen* at the τ th quantile of Y_0 had she not been treated. It is difficult to think of real-world examples in which rank invariance is likely to hold. For this reason we focus on identifying the ATE in the remainder of these notes.

1.8 The problem to overcome: selection bias

We know from above that $Y = (1 - D)Y_0 + DY_1$. For a person who is treated we observe Y_1 and for a person who is not we observe Y_0 . So to estimate $ATE \equiv \mathbb{E}[Y_1] - \mathbb{E}[Y_0]$, why not simply compare the average value of Y among those with $D = 1$ to the average value of Y among those with $D = 0$? Because D is binary, this idea is *precisely* equivalent to regressing Y on D . To see this we use the following lemma.²

²For a proof, see the appendix to this chapter.

Lemma 1.1. *Let W be a binary random variable with $\mathbb{P}(W = 1) = p$. Then for any random variable X , we have $\text{Cov}(X, W) = p(1 - p) [\mathbb{E}(X|W = 1) - \mathbb{E}(X|W = 0)]$ provided that the requisite expectations exist.*

Since D is binary, $\text{Var}(D) = \mathbb{P}(D = 1) [1 - \mathbb{P}(D = 1)]$. Thus, applying [Lemma 1.1](#),

$$\beta_{OLS} \equiv \frac{\text{Cov}(D, Y)}{\text{Var}(D)} = \mathbb{E}(Y|D = 1) - \mathbb{E}(Y|D = 0). \quad (1.3)$$

Does β_{OLS} equal the ATE? To find out, we substitute [\(1.2\)](#) into [\(1.3\)](#) yielding

$$\begin{aligned} \beta_{OLS} &= \mathbb{E}(Y|D = 1) - \mathbb{E}(Y|D = 0) \\ &= \mathbb{E}[(1 - D)Y_0 + DY_1|D = 1] - \mathbb{E}[(1 - D)Y_0 + DY_1|D = 0] \\ &= \mathbb{E}[Y_1|D = 1] - \mathbb{E}[Y_0|D = 0]. \end{aligned}$$

These manipulations show that β_{OLS} *may not* equal the ATE. The unconditional mean $\mathbb{E}(Y_1)$ need not equal the conditional mean $\mathbb{E}(Y_1|D = 1)$, and similarly $\mathbb{E}(Y_0)$ need not equal $\mathbb{E}(Y_0|D = 0)$, because D may be *related* to the potential outcomes. This problem is called **selection bias**. To better understand it, consider the following example: let $D = 1$ if you graduated from university and let Y be your income at age 30. Adding and subtracting $\mathbb{E}(Y_0|D = 1)$ from the expression for β_{OLS} , we have

$$\beta_{OLS} = \underbrace{\mathbb{E}(Y_1 - Y_0|D = 1)}_{\text{TOT}} + \underbrace{[\mathbb{E}(Y_0|D = 1) - \mathbb{E}(Y_0|D = 0)]}_{\text{Difference in Outside Options}}. \quad (1.4)$$

The first term in [\(1.4\)](#) is the average causal effect of the **treatment on the treated** abbreviated TOT. This measures causal effect of graduating from university on income averaged over all the people in the population who *chose* to graduate from university. When treatment effects are heterogeneous the TOT need not equal the ATE. Mark Zuckerberg famously dropped out of Harvard University in his sophomore year ($D = 0$) but is currently one of the highest earning people on the planet. Presumably his decision to leave university was motivated by a belief that his personal treatment effect $y_{i1} - y_{i0}$ was *negative*: the time he would have spent studying could be put to more lucrative use developing Facebook. If people have some knowledge of their personal treatment effects and are to some extent free to choose their treatment, then we would expect $\mathbb{E}(Y_1 - Y_0|D = 1)$ to be *higher* than the ATE and $\mathbb{E}(Y_1 - Y_0|D = 0)$ to be *lower*.³

The second term in [\(1.4\)](#) measures the difference in average values of Y_0 between the treated and the untreated. In the university and income example, this measures the average **difference in outside options** between those who ultimately chose to attend

³By the Law of Iterated Expectations ([Lemma 1.2](#)), the ATE $\mathbb{E}(Y_1 - Y_0)$ is a convex combination of $\mathbb{E}(Y_1 - Y_0|D = 1)$ and $\mathbb{E}(Y_1 - Y_0|D = 0)$, so it necessarily lies *between* them.

university and those who did not.⁴ If higher ability people are more likely to graduate from university ($D = 1$) and also have a higher-paying outside option Y_0 , say because ability has a direct effect on income, the second term in (1.4) will be *positive*. Thus, even if the TOT is equal to the ATE, β_{OLS} will not in general identify the average causal effect of D on Y when individuals can choose their treatment status.

Once you start looking for it, you will find examples of selection bias *everywhere*. People who are admitted to hospitals are more likely to die in the next year than people who are not. This isn't because hospitals kill people: it's because sick people are more likely to go to hospitals. Dog owners are less likely to die over a five year horizon, but this may simply reflect the fact that healthy people are more likely to get a dog than sick people: taking care of an animal is a lot of work! Watching Fox News may cause you to vote Republican, or perhaps voting Republican causes you to watch Fox News.

1.9 Appendix: Proofs and Probability Review

The mathematical level of these notes is fairly modest. I assume throughout, however, that you are familiar with basic properties of random variables, expectation, variance, and covariance. In case you need to refresh your memory, this section lists some important properties that are used throughout the document.

Proof of Lemma 1.1. Let $p = \mathbb{P}(W = 1) = \mathbb{E}(W)$ and define $m_0 = \mathbb{E}(X|W = 0)$ and $m_1 = \mathbb{E}(X|W = 1)$ By the shortcut formula and iterated expectations,

$$\begin{aligned}\text{Cov}(X, W) &= \mathbb{E}(XW) - \mathbb{E}(X)\mathbb{E}(W) = \mathbb{E}[W\mathbb{E}(X|W)] - \mathbb{E}(X)p \\ &= \mathbb{E}(X|W = 1)p - \mathbb{E}(X)p = pm_1 - p\mathbb{E}(X)\end{aligned}$$

Applying iterated expectations a second time,

$$\mathbb{E}(X) = \mathbb{E}[\mathbb{E}(X|W)] = m_0(1 - p) + pm_1$$

and substituting this equation into the expression for $\text{Cov}(X, W)$,

$$\begin{aligned}\text{Cov}(X, W) &= pm_1 - p[m_0(1 - p) + pm_1] = (p - p^2)m_1 - p(1 - p)m_0 \\ &= p(1 - p)(m_1 - m_0) = p(1 - p)[\mathbb{E}(X|W = 1) - \mathbb{E}(X|W = 0)]\end{aligned}$$

□

⁴Some authors call the second term in (1.4) the “selection bias.” In contrast I reserve this phrase for the *overall* difference between β_{OLS} and the ATE that arises when people are free to choose their treatments.

Lemma 1.2 (The Law of Iterated Expectations).

$$\mathbb{E}[Y] = \mathbb{E}_X [\mathbb{E}(Y|X)], \quad \mathbb{E}[Y|Z] = \mathbb{E}_{X|Z} [\mathbb{E}(Y|X, Z)]$$

Lemma 1.3 (Taking out what is known). *If f is a measurable function, then*

$$\mathbb{E}[f(X)Y|X] = f(X)\mathbb{E}[Y|X]$$

Lemma 1.4 (The Law of Total Probability). *For discrete random variables X and Y*

$$\mathbb{P}(Y = y) = \sum_{all\ x} \mathbb{P}(Y = y|X = x)\mathbb{P}(X = x)$$

Lemma 1.5 (Linearity of Expectation). *For RVs X, Y, Z and constants a, b, c*

$$\mathbb{E}[aX + bY + c] = a\mathbb{E}[X] + b\mathbb{E}[Y] + c, \quad \mathbb{E}[aX + bY + c|Z] = a\mathbb{E}[X|Z] + b\mathbb{E}[Y|Z] + c$$

Lemma 1.6 (Bayes' Theorem).

$$\mathbb{P}(A|B) = \frac{\mathbb{P}(B|A)\mathbb{P}(A)}{\mathbb{P}(B)}, \quad \mathbb{P}(A|B, C) = \frac{\mathbb{P}(B|A, C)\mathbb{P}(A|C)}{\mathbb{P}(B|C)}$$

Definition 1.1 (Variance and Conditional Variance).

$$\text{Var}(X) \equiv \mathbb{E} [(X - \mathbb{E} \{X\})^2], \quad \text{Var}(X|Z) \equiv \mathbb{E} [(X - \mathbb{E} \{X|Z\})^2 | Z]$$

Definition 1.2 (Covariance and Conditional Covariance).

$$\begin{aligned} \text{Cov}(X, Y) &\equiv \mathbb{E} [(X - \mathbb{E} \{X\}) (Y - \mathbb{E} \{Y\})] \\ \text{Cov}(X, Y|Z) &\equiv \mathbb{E} [(X - \mathbb{E} \{X|Z\}) (Y - \mathbb{E} \{Y|Z\}) | Z] \end{aligned}$$

Lemma 1.7 (Shortcut Rule for Variance and Covariance).

$$\begin{aligned} \text{Var}(X) &= \mathbb{E}[X^2] - \mathbb{E}[X]^2 \\ \text{Var}(X|Z) &= \mathbb{E}[X^2|Z] - \mathbb{E}[X|Z]^2 \\ \text{Cov}(X, Y) &= \mathbb{E}[XY] - \mathbb{E}[X]\mathbb{E}[Y] \\ \text{Cov}(X, Y|Z) &= \mathbb{E}[XY|Z] - \mathbb{E}[X|Z]\mathbb{E}[Y|Z] \end{aligned}$$

Lemma 1.8 (Properties of Variance and Covariance).

- (i) $\text{Cov}(X, X) = \text{Var}(X)$
- (ii) $\text{Var}(aX + c) = a^2 \text{Var}(X)$

$$(iii) \quad \text{Var}(aX + bY + c) = a^2 \text{Var}(X) + b^2 \text{Var}(Y) + 2ab \text{Cov}(X, Y)$$

$$(iv) \quad \text{Cov}(aX + bY + c, Z) = a \text{Cov}(X, Z) + b \text{Cov}(Y, Z)$$

Lemma 1.9 (Properties of Conditional Variance and Covariance).

$$(i) \quad \text{Var}(X|X) = 0$$

$$(ii) \quad \text{Cov}(X, Y|X) = 0$$

$$(iii) \quad \text{Cov}(X, X|Z) = \text{Var}(X|Z)$$

$$(iv) \quad \text{Var}(aX + c|Z) = a^2 \text{Var}(X|Z)$$

$$(v) \quad \text{Var}(aX + bY + c) = a^2 \text{Var}(X) + b^2 \text{Var}(Y) + 2ab \text{Cov}(X, Y)$$

$$(vi) \quad \text{Cov}(aX + bY + c, Z|W) = a \text{Cov}(X, Z|W) + b \text{Cov}(Y, Z|W)$$

Lemma 1.10 (The Law of Total Variance).

$$\text{Var}(Y) = \mathbb{E} [\text{Var}(Y|X)] + \text{Var}(\mathbb{E}[Y|X])$$

Lemma 1.11 (The Law of Total Covariance).

$$\text{Cov}(X, Y) = \mathbb{E} [\text{Cov}(X, Y|Z)] + \text{Cov}[\mathbb{E}(X|Z), \mathbb{E}(Y|Z)]$$

Chapter 2

Conditional Independence

To understand the literature on treatment effects, you will need to develop some familiarity with the notion of **conditional independence** and its properties. This chapter provides an overview. We begin by defining independence and the closely related idea of conditional independence, and go on to explain the consequences that these notions have for *expectations*. This allows us to propose our first solution to the problem of selection bias: randomly assigning individuals to treatment.

The remainder of the chapter discusses a set of *axioms* that allow us to manipulate conditional independence relationships. Defining conditional independence and deriving its axioms for *all possible* kinds of random variables requires some measure theory. If have the appropriate background, I recommend reading the technical appendix, [section 2.6](#), alongside the rest of the chapter. If you are not familiar with measure theory, don't worry: you will be able to understand everything except the technical appendix. There are only two terms from measure theory that I use in the body of the chapter. The first is that of a **measurable function**. If you haven't encountered this term before, it is just a particular way of saying that a function is “well-behaved.” Any continuous function is measurable, as is any discontinuous function with a finite or countable number of discontinuities. The second is the terminology “ W is Y -measurable.” In words, this simply means that if we know the realization of the random variable Y then we also know the realization of the random variable W .

2.1 Intuition and Notation

Two continuous random variables X and Y are **independent** if and only if their joint density equals the product of their marginal densities: $f(x, y) = f(x)f(y)$ for all x, y in the support sets of X and Y .¹ By the definition of a conditional density, $f(y|x) = f(x, y)/f(x)$ so an *equivalent* definition of statistical independence is $f(y|x) = f(y)$ for

¹For discrete RVs, replace densities with mass functions throughout, e.g. $p(x, y) = p(x)p(y)$.

all x, y in the support sets of X and Y . In other words, X and Y are independent if and only if knowing X provides *no additional information* about Y : the conditional density of Y given X is the same as the marginal density of Y . Of course we could just have easily reversed the roles of X and Y : an additional equivalent definition of conditional independence is $f(x|y) = f(x)$.

A closely related property is **conditional independence**. Two continuous random variables X and Y are conditionally independent given a third random variable Z if and only if $f(x, y|z) = f(x|z)f(y|z)$ for all x, y, z in the support sets of X, Y, Z . Using the definition of a conditional density, $f(y|x, z) = f(x, y|z)/f(x|z)$, this is equivalent to $f(y|x, z) = f(y|z)$. Reversing the roles of y and x , it is *also* equivalent to $f(x|y, z) = f(x|z)$.² If X and Y are conditionally independent given Z , this means that any dependence between X and Y comes solely from the fact that both are dependent on Z . In words: if we already know Z , then knowing X tells us nothing additional about Y , and vice-versa. We define conditional independence for continuous random *vectors* analogously: \mathbf{X} and \mathbf{Y} are conditionally independent given \mathbf{Z} if $f(\mathbf{x}, \mathbf{y}|\mathbf{z}) = f(\mathbf{x}|\mathbf{z})f(\mathbf{y}|\mathbf{z})$, or equivalently if $f(\mathbf{y}|\mathbf{x}, \mathbf{z}) = f(\mathbf{y}|\mathbf{z})$ or $f(\mathbf{x}|\mathbf{y}, \mathbf{z}) = f(\mathbf{x}|\mathbf{z})$. For discrete random vectors, replace densities with mass functions.³

Independence, conditional and unconditional, is such an important concept in statistics and econometrics that it has its own symbol: “ \perp .” If we write $X \perp Y$ this means that X is independent of Y ; if we write $X \perp Y|Z$, this means that X is independent of Y , given Z . The same notation is used for random variables and random vectors.

2.2 Independence versus Mean Independence

Because our goal is to identify average treatment effects, we will be particularly interested in the consequences that conditional independence has for *means*.

Lemma 2.1. *Let X, Y, Z be random variables. If $X \perp Y|Z$ then*

- (i) $\mathbb{E}[XY|Z] = \mathbb{E}[X|Z]\mathbb{E}[Y|Z]$
- (ii) $\mathbb{E}[Y|X, Z] = \mathbb{E}[Y|Z]$
- (iii) $\mathbb{E}[X|Y, Z] = \mathbb{E}[X|Z]$.

Proof. The general case follows as a corollary of [Proposition 2.1](#). Here we will assume that X, Y, Z are continuous random variables. Results for discrete RVs follow by

²There are in fact many equivalent definitions of conditional independence. For full details see the Technical Appendix ([section 2.6](#)).

³For a fully general definition of conditional independence, see the Technical Appendix ([section 2.6](#)).

replacing integrals with sums. For (i), use $f(x, y|z) = f(x|z)f(y|z)$ and the definition of conditional expectation to write

$$\begin{aligned} E[XY|Z = z] &= \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} xyf(x, y|z) dx dy = \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} xyf(x|z)f(y|z) dx dy \\ &= \int_{-\infty}^{\infty} yf(y|z) \left(\int_{-\infty}^{\infty} xf(x|z)dx \right) dy = E[X|Z = z] \int_{-\infty}^{\infty} yf(y|z)dy \\ &= E[X|Z = z]E[Y|Z = z]. \end{aligned}$$

For (ii), use $f(y|x, z) = f(y|z)$ and the definition of conditional expectation to write

$$E[Y|X = x, Z = z] = \int_{-\infty}^{\infty} yf(y|x, z) dy = \int_{-\infty}^{\infty} yf(y|z) dy = E[Y|Z = z].$$

The argument for (iii) is nearly identical, combining $f(x|y, z) = f(x|z)$ with the definition of conditional expectation. \square

Properties (ii) and (iii) of the lemma are often called **mean independence**. It is important to remember that conditional independence implies mean independence but *not the other way around*. Conditional independence is the stronger assumption. There is also a version of [Lemma 2.1](#) that holds without conditioning on Z : $X \perp\!\!\!\perp Y$ implies that $E[XY] = E[X]E[Y]$, $E[Y|X] = E[Y]$, and $E[X|Y] = E[X]$. A good exercise would be to prove these implications for yourself if X and Y are continuous. Similar results also hold for random *vectors*: if $\mathbf{X} \perp\!\!\!\perp \mathbf{Y} | \mathbf{Z}$ then $E[\mathbf{Y} | \mathbf{X}, \mathbf{Z}] = E[\mathbf{Y} | \mathbf{Z}]$ and $E[\mathbf{X} | \mathbf{Y}, \mathbf{Z}] = E[\mathbf{X} | \mathbf{Z}]$. Moreover, if $\mathbf{X} \perp\!\!\!\perp \mathbf{Y}$ then $E[\mathbf{Y} | \mathbf{X}] = E[\mathbf{Y}]$ and $E[\mathbf{X} | \mathbf{Y}] = E[\mathbf{X}]$.

2.3 Randomize treatments to eliminate selection bias.

Now that we know something about mean independence, we can propose our first solution to the problem of selection bias, as described in [section 1.8](#) above. Suppose that, instead of arising naturally from the decisions people make, treatments were *randomly assigned* to people, independently of any of their characteristics. In this case, D would be *independent* of (Y_0, Y_1) . By an argument nearly identical to that in [Lemma 2.1](#) only without the “ Z ” this would imply that $E(Y_0|D) = E(Y_0)$ and $E(Y_1|D) = E(Y_1)$. Thus,

$$\begin{aligned} \beta_{OLS} &= E(Y|D = 1) - E(Y|D = 0) \\ &= E(Y_1|D = 1) - E(Y_0|D = 0) \\ &= E(Y_1 - Y_0) = \text{ATE} \end{aligned}$$

if $D \perp\!\!\!\perp (Y_0, Y_1)$. In words: there is no selection bias in a randomized experiment in which subjects are not free to choose their treatment.⁴ Because randomized experiments are immune to selection bias, experimental studies are considered by many to be a “gold standard” against which other kinds of studies, such as those based on observational data, are to be judged. Valuable though they can be when applied carefully and interpreted correctly, however, randomized controlled trials are no panacea. For a recent critique, see [Deaton and Cartwright \(2018\)](#).

2.4 The Axioms of Conditional Independence

Now that we understand what conditional independence means, we have to learn how to work with it mathematically. Our approach will be *axiomatic*: we will state a number of abstract properties that the independence operator $\perp\!\!\!\perp$ satisfies and see how to use these to derive new properties. The result will be a kind of “algebra” of conditional independence: we will learn a number of rules with which we can manipulate a given conditional independence assumption to transform it into new conditional independence assumptions. All of the axioms of conditional independence can be rigorously proved from first principles: see the Technical Appendix for details ([section 2.6](#)). The names attached to axioms (i) and (iii)–(v) are taken from Pearl (1988). Axiom (ii) has not been given a name in the literature, so I have christened it the “redundancy” property. Note that when we write $W = h(Y)$ where h is a measurable function, this is equivalent to saying that W is Y -measurable: in other words, knowing the realization of Y tells us with certainty the realization of W .

Theorem 2.1 (Axioms of Conditional Independence). *Let X, Y, Z, W be random variables defined on a common probability space, and let h be a measurable function. Then:*

- (i) (*Symmetry*): $X \perp\!\!\!\perp Y | Z \implies Y \perp\!\!\!\perp X | Z$.
- (ii) (*Redundancy*): $X \perp\!\!\!\perp Y | Y$.
- (iii) (*Decomposition*): $X \perp\!\!\!\perp Y | Z$ and $W = h(Y) \implies X \perp\!\!\!\perp W | Z$.
- (iv) (*Weak Union*): $X \perp\!\!\!\perp Y | Z$ and $W = h(Y) \implies X \perp\!\!\!\perp Y | (W, Z)$.
- (v) (*Contraction*): $X \perp\!\!\!\perp Y | Z$ and $X \perp\!\!\!\perp W | (Y, Z) \implies X \perp\!\!\!\perp (Y, W) | Z$.

We begin with some important discussion of what these properties mean, how they can be used, and how they relate to properties used by other authors.

⁴This rules out settings in which some experimental subjects refuse to comply with the treatment they have been randomly assigned. We take up this more challenging case in a later chapter.

Random Variables vs. Vectors All of the results from above and the Technical Appendix, including [Proposition 2.1](#) and [Theorem 2.1](#), hold regardless of whether X, Y, Z, W are real-valued random variables, random vectors, or arbitrary collections of random variables and vectors. This is important, as it is typically necessary to find “clever” choices of X, Y, Z, W when applying the axioms of conditional independence. Often this requires defining one or more of these to be a *collection* of random variables, as we will see in many of the examples below.

Conditional vs. Unconditional Axioms Axioms (i) and (iii)–(v) are stated conditional on Z , but these same statements also hold *unconditionally* by dropping Z .⁵ Because it is easier to put these unconditional versions of the axioms into words, I omit explicit conditioning on Z in some of the verbal explanations below.

Symmetry The symmetry property says that if learning Y does not give us any information about X , then learning X does not give us any information about Y . This is actually somewhat surprising, as the equality $\mathbb{E}(\mathbb{1}\{A_X\} | Y, Z) = \mathbb{E}(\mathbb{1}\{A_X\} | Z)$ does *not* treat X and Y symmetrically. Symmetry only becomes intuitively clear after establishing [Proposition 2.1](#).

Redundancy The redundancy property says that if I already know Y , then learning Y *a second time* provides no additional information about X . Since $X \perp\!\!\!\perp Y | Y$ implies $Y \perp\!\!\!\perp X | Y$ by symmetry, another way of interpreting this condition is that, conditional on itself, a random variable Y is independent of *any other random variable*. In fact we can establish a more general result using similar reasoning, namely $X \perp\!\!\!\perp W | Y$ if W is Y -measurable. A proof of this fact using the axioms of conditional independence appears in the following section.

Decomposition The decomposition property says that if learning Y provides no information about X , then learning a *function* of Y likewise provides no information about X . If W is a measurable function of Y then it contains *at most* the same information content as Y . A common use of decomposition is to *drop* a random variable from a conditional independence statement. For example, suppose that $X_1 \perp\!\!\!\perp (X_2, X_3) | Z$. Since X_2 is (X_2, X_3) -measurable, it follows that $X_1 \perp\!\!\!\perp X_2 | Z$. Analogously, $X_1 \perp\!\!\!\perp X_3 | Z$. This *consequence* of the decomposition axiom is what some authors call “the decomposition property.”

Weak Union The weak union property says that if learning Y provides no information about X , then learning Y after having *already learned* a function of Y likewise provides no

⁵Formally, this is equivalent to taking $\sigma(Z) = \emptyset$.

information about X . In effect, weak union allows us to *add* something to our conditioning set. A common application of this property is to move a random variable from the “left” of the conditioning bar to the “right.” For example, suppose that $X_1 \perp\!\!\!\perp (X_2, X_3) | Z$. Since X_2 is (X_2, X_3) -measurable, weak union gives $X_1 \perp\!\!\!\perp (X_2, X_3) | (X_3, Z)$. It follows by decomposition that $X_1 \perp\!\!\!\perp X_2 | (X_3, Z)$. Naturally, the same logic shows that $X_1 \perp\!\!\!\perp X_3 | (X_2, Z)$. This *consequence* of the weak union and decomposition axioms is what some authors call the “weak union property.”

Contraction The contraction property is a bit complicated to put into words. In effect, it allows us to move a random variable from the “right” of the conditioning bar to the “left”. For example, suppose that $X_1 \perp\!\!\!\perp X_2 | (X_3, X_4)$ and we want to show that $X_1 \perp\!\!\!\perp (X_2, X_3) | X_4$. If $X_1 \perp\!\!\!\perp X_3 | X_4$, then contraction will give us our desired result.

2.5 More Properties of Conditional Independence

The axioms of conditional independence from [Theorem 2.1](#) provide a simple but powerful way to deduce new conditional independence relationships from old ones.

Corollary 2.1. $X \perp\!\!\!\perp Y | Z$ implies $(X, Z) \perp\!\!\!\perp Y | Z$.

Proof of Corollary 2.1. By symmetry,

$$Y \perp\!\!\!\perp X | Z \tag{2.1}$$

and by redundancy,

$$Y \perp\!\!\!\perp (X, Z) | (X, Z). \tag{2.2}$$

Now, applying the decomposition property to [\(2.2\)](#)

$$Y \perp\!\!\!\perp Z | (X, Z) \tag{2.3}$$

and hence, applying the contraction property to [\(2.1\)](#) and [\(2.3\)](#), we obtain $Y \perp\!\!\!\perp (X, Z) | Z$. The result follows by symmetry. \square

Another simple result that can be derived from the axioms of conditional probability is the following extension of the redundancy property. This does not appear in any references that I have seen, but it is easy to establish using the axioms of conditional independence.

Corollary 2.2. Let $W = h(Y)$ where h is a measurable function. Then $X \perp\!\!\!\perp W | Y$.

Proof of Corollary 2.2. By redundancy $X \perp\!\!\!\perp Y | Y$. By decomposition, taking Y to be “ Z ,” this yields $X \perp\!\!\!\perp W | Y$. \square

The well known-result that $X \perp\!\!\!\perp Y|Z$ implies $f(X) \perp\!\!\!\perp g(Y)|Z$ also follows directly from the axioms of conditional independence.

Corollary 2.3. *Let f and g be measurable functions. Then $X \perp\!\!\!\perp Y|Z \implies f(X) \perp\!\!\!\perp g(Y)|Z$.*

Proof of Corollary 2.3. By decomposition, $X \perp\!\!\!\perp g(Y)|Z$. Hence, by symmetry $g(Y) \perp\!\!\!\perp X|Z$. Applying decomposition a second time, $g(Y) \perp\!\!\!\perp f(X)|Z$. The result follows by a final application of symmetry. \square

2.6 Appendix: Technical Details

Definition 2.1 (Conditional Independence). Let X, Y, Z be random variables defined on a common probability space $(\Omega, \mathcal{A}, \mathbb{P})$. We say that X is conditionally independent of Y given Z (with respect to \mathbb{P}), written $X \perp\!\!\!\perp Y|Z$ if for all events $A_X \in \sigma(X)$ we have $\mathbb{E}(\mathbb{1}\{A_X\} | Y, Z) = \mathbb{E}(\mathbb{1}\{A_X\} | Z)$, \mathbb{P} -almost surely.

Proposition 2.1 (Equivalent Definitions of Conditional Independence). *Let X, Y, Z be random variables defined on a common probability space $(\Omega, \mathcal{A}, \mathbb{P})$. Then the following statements are equivalent:*

- (i) $X \perp\!\!\!\perp Y|Z$
- (ii) For all real, bounded, measurable functions f , $\mathbb{E}[f(X)|Y, Z] = \mathbb{E}[f(X)|Z]$
- (iii) For all, real, bounded, measurable functions f, g , $\mathbb{E}[f(X)g(Y)|Z] = \mathbb{E}[f(X)|Z] \mathbb{E}[g(Y)|Z]$
- (iv) For all $A_X \in \sigma(X)$ and all $A_Y \in \sigma(Y)$, $\mathbb{E}[\mathbb{1}\{A_X \cap A_Y\} | Z] = \mathbb{E}[\mathbb{1}\{A_X\} | Z] \mathbb{E}[\mathbb{1}\{A_Y\} | Z]$

where all equalities of conditional expectations are understood to hold \mathbb{P} -almost surely.

Proof of the Symmetry Property. The symmetry property follows immediately from the alternative definition of conditional independence given in [Proposition 2.1](#) (iii). \square

Proof of the Redundancy Property. Let f and g be real-valued, bounded, measurable functions. Since $g(Y)$ is Y -measurable,

$$\mathbb{E}[f(X)g(Y)|Y] = \mathbb{E}[f(X)|Y]g(Y) = \mathbb{E}[f(X)|Y] \mathbb{E}[g(Y)|Y]$$

so the result follows by [Proposition 2.1](#) (iii). \square

Proof of the Decomposition Property. Let f be a real-valued, bounded, measurable function. Since W is a measurable function of Y , we have $\sigma(W) \subseteq \sigma(Y)$ and consequently $\sigma(W, Z) \subseteq \sigma(Y, Z)$. Hence, by the *tower property* of conditional expectation,

$$\mathbb{E}[f(X)|W, Z] = \mathbb{E}\{\mathbb{E}[f(X)|Y, Z] | W, Z\}.$$

But since $X \perp\!\!\!\perp Y|Z$, **Proposition 2.1** (ii) gives $\mathbb{E}[f(X)|Y, Z] = \mathbb{E}[f(X)|Z]$. And because $\mathbb{E}[f(X)|Z]$ is (W, Z) -measurable,

$$\mathbb{E}\{\mathbb{E}[f(X)|Z] | W, Z\} = \mathbb{E}[f(X)|Z] \mathbb{E}[1 | W, Z] = \mathbb{E}[f(X)|Z].$$

Thus, $\mathbb{E}[f(X)|W, Z] = \mathbb{E}[f(X)|Z]$ so the result follows by **Proposition 2.1** (ii). \square

Proof of the Weak Union Property. Let f be a real-valued, bounded, measurable function. Since W is a measurable function of Y , we have $\sigma(W) \subseteq \sigma(Y)$. As a result, it follows that $\sigma(Y, W, Z) = \sigma(Y, Z)$ and hence $\mathbb{E}[f(X)|Y, W, Z] = \mathbb{E}[f(X)|Y, Z]$. Now, since $X \perp\!\!\!\perp Y|Z$, **Proposition 2.1** (ii) gives $\mathbb{E}[f(X)|Y, Z] = \mathbb{E}[f(X)|Z]$. Finally, since $X \perp\!\!\!\perp Y|Z$ and W is Y -measurable, the decomposition property, **Theorem 2.1** (iii), gives $X \perp\!\!\!\perp W|Z$ and hence $\mathbb{E}[f(X)|Z] = \mathbb{E}[f(X)|Z, W]$. Hence, the result follows by **Proposition 2.1** (ii). \square

Proof of the Contraction Property. Let f be a real, bounded, measurable function. Now, since $X \perp\!\!\!\perp W|(Y, Z)$ we have $\mathbb{E}[f(X)|Y, W, Z] = \mathbb{E}[f(X)|Y, Z]$ by **Proposition 2.1** (ii). Similarly, since $X \perp\!\!\!\perp Y|Z$ we have $\mathbb{E}[f(X)|Y, Z] = \mathbb{E}[f(X)|Z]$. Combining these equalities gives $\mathbb{E}[f(X)|Y, W, Z] = \mathbb{E}[f(X)|Z]$ so the result follows by **Proposition 2.1** (i). \square

Chapter 3

Selection on Observables

As we saw in [chapter 2](#), there is no selection bias when D is randomly assigned: a simple comparison of mean outcomes between treated and untreated individuals identifies the ATE. In many examples, however, carrying out a randomized controlled trial may be infeasible, unethical, or even impossible. In this chapter we will consider an assumption called *selection on observables* that allows us to identify the ATE from observational data by conditioning on observed characteristics \mathbf{X} . We'll consider two different approaches to identification that both rely on the selection on observables assumption: one based on regression adjustment and another based on propensity score weighting.

3.1 Does education cause political participation?

This example is based on [Kam and Palmer \(2008\)](#). University graduates are more likely to vote, volunteer for political campaigns, contact their elected representatives, and participate in demonstrations. Does this show that education *causes* political participation? Let $D = 1$ if you attended university and $D = 0$ otherwise. Further let Y be an index of political participation, where high values indicate greater participation and lower values indicate less. It seems hard to believe that D could be independent of the potential outcomes (Y_0, Y_1) in this example. University graduates differ from non-graduates in myriad ways that could also influence political participation. People from wealthy backgrounds are more likely to graduate from college. They are also more likely to have the leisure time required for political participation; if you are struggling to make ends meet it will be hard to find time to attend a political rally. Because it seems far-fetched to imagine anyone carrying out an experiment that forced some people to attend college and others not to, observational data is the best we can hope for if our goal is to identify the causal effect of education on political participation.

The assumption that $(Y_0, Y_1) \perp\!\!\!\perp D$ is clearly untenable, so what could we use instead? Our main reason for doubting that a simple comparison of mean political participation

across groups could be given a causal interpretation was that university graduates are different from non-graduates in more ways than their education level. But perhaps if we were to *condition* on these differences, effectively holding them fixed, we could find a way to make progress. In other words, even if (Y_0, Y_1) are not independent of D , perhaps there is a collection of observable individual characteristics \mathbf{X} such that $(Y_0, Y_1) \perp\!\!\!\perp D | \mathbf{X}$. For example, perhaps by conditioning on sex, race, family background and so on we could break the dependence between college graduation and the potential outcomes. This idea is called *selection on observables* because it assumes that selection bias operates solely through characteristics that we can observe.

3.2 Selection on Observables and Overlap

The methods explored in this chapter rely on two assumptions. First is *selection on observables*, as outlined in the previous section. The precise version of this condition that we will rely on below is as follows.

Assumption 3.1 (Selection on Observables).

$$\mathbb{E}(Y_0 | \mathbf{X}, D) = \mathbb{E}(Y_0 | \mathbf{X}), \quad \text{and} \quad \mathbb{E}(Y_1 | \mathbf{X}, D) = \mathbb{E}(Y_1 | \mathbf{X}).$$

Assumption 3.1 says that the potential outcomes (Y_0, Y_1) are *mean independent* of the treatment D conditional on \mathbf{X} . This is weaker than but implied by the conditional independence assumption, namely $(Y_0, Y_1) \perp\!\!\!\perp D | \mathbf{X}$, described in the previous section.¹ Because our goal is to identify a mean, the ATE, we only require a mean independence assumption. To introduce our second assumption we require the following definition.

Definition 3.1 (Propensity Score). The probability $p(\mathbf{X}) \equiv \mathbb{P}(D = 1 | \mathbf{X})$ of treatment conditional on an observed random vector \mathbf{X} is called the propensity score.

Assumption 3.2 (Overlap). $0 < p(\mathbf{x}) < 1$ for all \mathbf{x} in the support of \mathbf{X} .

Assumption 3.2 states that the propensity score is *strictly* between zero and one for any value that the covariates \mathbf{X} could take on. Since $p(\mathbf{x}) \equiv \mathbb{P}(D = 1 | \mathbf{X} = \mathbf{x})$, this requires that, among people with any fixed value \mathbf{x} of the covariates \mathbf{X} , some are treated ($D = 1$) and some are untreated ($D = 0$).

Both **Assumption 3.1** and **Assumption 3.2** are crucial for the methods described below. Unfortunately the two are somewhat at odds with each other. The more observed controls \mathbf{X} that we condition on, the more plausible the selection on observables assumption (**Assumption 3.1**) becomes.² At the same time, conditioning on a richer set of

¹See part (ii) of **Lemma 2.1**.

²But beware of *bad controls*! See **section 3.8** for details.

controls makes it *harder* to satisfy the overlap condition. Suppose that \mathbf{X} includes race, sex, whether or not you attended an independent secondary school, year of birth, and post code. It is distinctly possible that *every* white male who attended an independent secondary school and was born in 1995 to a wealthy North Oxford family in fact graduated from university. If so, the overlap assumption fails for this particular value of \mathbf{x} . A common although not entirely satisfactory solution to the failure of [Assumption 3.2](#) is to redefine the population of interest by restricting attention to only those values \mathbf{x} for which overlap holds. For example, we might be forced to exclude people born to wealthy North Oxford families from our population of interest. Note that if we take this route, we will identify a *different ATE* than the one we initially set out to recover: one that corresponds to the restricted population.

3.3 Identification by Regression Adjustment

Our first approach to identifying the ATE using [Assumption 3.1](#) and [Assumption 3.2](#) is called *regression adjustment*. The idea is to compare mean values of Y between treated and untreated individuals within *strata* defined by a common value \mathbf{x} of the covariates. This yields a conditional ATE given that $\mathbf{X} = \mathbf{x}$. This quantity, which we denote $ATE(\mathbf{x})$, is the average treatment effect for a certain kind of person, namely someone with covariates equal to \mathbf{x} , e.g. a white male born to a wealthy North Oxford family in 1995. To convert this into an unconditional ATE we average $ATE(\mathbf{x})$ over the distribution of \mathbf{X} in the population using the law of iterated expectations ([Lemma 1.2](#)).

Theorem 3.1. *Under [Assumption 3.1](#) and [Assumption 3.2](#),*

$$ATE \equiv \mathbb{E}(Y_1 - Y_0) = \mathbb{E}_{\mathbf{X}} [\mathbb{E}(Y|\mathbf{X}, D = 1)] - \mathbb{E}_{\mathbf{X}} [\mathbb{E}(Y|\mathbf{X}, D = 0)].$$

Proof. Since $Y = Y_0 + D(Y_1 - Y_0)$, under selection on observables

$$\begin{aligned} \mathbb{E}(Y|\mathbf{X}, D) &= \mathbb{E}(Y_0|\mathbf{X}, D) + D [\mathbb{E}(Y_1|\mathbf{X}, D) - \mathbb{E}(Y_0|\mathbf{X}, D)] \\ &= \mathbb{E}(Y_0|\mathbf{X}) + D [\mathbb{E}(Y_1|\mathbf{X}) - \mathbb{E}(Y_0|\mathbf{X})] \end{aligned}$$

where the first equality follows by the properties of conditional expectation, and the second from [Assumption 3.1](#). Substituting $D = 0$ and $D = 1$ into the preceding expression,

$$\mathbb{E}(Y|\mathbf{X}, D = 0) = \mathbb{E}(Y_0|\mathbf{X}), \quad \mathbb{E}(Y|\mathbf{X}, D = 1) = \mathbb{E}(Y_1|\mathbf{X})$$

which in turn implies that

$$ATE(\mathbf{X}) = \mathbb{E}(Y_1 - Y_0|\mathbf{X}) = \mathbb{E}(Y|\mathbf{X}, D = 1) - \mathbb{E}(Y|\mathbf{X}, D = 0).$$

The overlap assumption ([Assumption 3.2](#)) implies that $\text{ATE}(\mathbf{X})$ is well-defined for all points in the support of \mathbf{X} , since it ensures that there are individuals with $D = 1$ and $D = 0$ for any value of the covariates. Hence, taking the expectation of both sides,

$$\text{ATE} = \mathbb{E}_{\mathbf{X}} [\text{ATE}(\mathbf{X})] = \mathbb{E}_{\mathbf{X}} [\mathbb{E}(Y|\mathbf{X}, D = 1)] - \mathbb{E}_{\mathbf{X}} [\mathbb{E}(Y|\mathbf{X}, D = 0)]$$

by the law of iterated expectations. □

3.4 Estimation by Regression Adjustment

Let $\hat{\mu}_0(\mathbf{X})$ be a consistent estimator of $\mathbb{E}(Y|\mathbf{X}, D = 0)$ and $\hat{\mu}_1(\mathbf{X})$ be a consistent estimator of $\mathbb{E}(Y|\mathbf{X}, D = 1)$. Then, under general conditions,

$$\widehat{\text{ATE}}_{RA} \equiv \frac{1}{n} \sum_{i=1}^n [\hat{\mu}_1(\mathbf{X}_i) - \hat{\mu}_0(\mathbf{X}_i)]$$

is a consistent estimator of the ATE, where *RA* stands for *regression adjustment*. The question remains: how do we obtain $\hat{\mu}_0(\cdot)$ and $\hat{\mu}_1(\cdot)$? If \mathbf{X} is discrete and takes on a small number of values, we can simply calculate the sample mean of Y at each combination of $(D = 0, \mathbf{X} = \mathbf{x})$ for $\hat{\mu}_0(\mathbf{x})$ and at each combination of $(D = 1, \mathbf{X} = \mathbf{x})$ for $\hat{\mu}_1(\mathbf{x})$. If \mathbf{X} contains any continuous variables, or is discrete but takes on a large number of values, however, this approach fails. Non-parametric methods, either series or kernel-based, provide an alternative but perform poorly when the dimension of \mathbf{X} is large. Model-based approaches are also possible, e.g. assuming that $\mathbb{E}[Y|D = d, \mathbf{X}]$ is linear in \mathbf{X} for a given value of d . If the model is a poor description of the true conditional mean function, however, this can produce misleading results. Model-based approaches can also mask failures of the overlap assumption: they will always generate a value for $\mathbb{E}[Y|D = d, \mathbf{X} = \mathbf{x}]$ even if there are no individuals in the dataset with $(D = d, \mathbf{X} = \mathbf{x})$. The model extrapolates from values that are actually contained in the dataset. Whichever method is used to construct estimates $\hat{\mu}_0(\cdot)$ and $\hat{\mu}_1(\cdot)$, a simple way to carry out inference that correctly accounts for this first-stage estimation step is to bootstrap pairs (Y_i, \mathbf{X}_i) .

3.5 Identification by Propensity Score Weighting

Our second approach to identifying the ATE using [Assumption 3.1](#) and [Assumption 3.2](#) is called *propensity score weighting*. Whereas regression adjustment compares average values of Y between treated and untreated individuals with the same value of \mathbf{X} , propensity score weighting calculates the average value of Y across *everyone in the population* with weights that depend on each person's actual treatment D and her predicted probability of treatment: the propensity score.

Theorem 3.2. Under *Assumption 3.1* and *Assumption 3.2*,

$$ATE \equiv \mathbb{E}(Y_1 - Y_0) = \mathbb{E} \left[\frac{\{D - p(\mathbf{X})\} Y}{p(\mathbf{X}) \{1 - p(\mathbf{X})\}} \right].$$

Proof. Since D is binary, $D^2 = D$, $(1 - D)^2 = (1 - D)$, and $D(1 - D) = 0$. Hence,

$$\begin{aligned} DY &= D^2 Y_1 + D(1 - D) Y_0 = DY_1 \\ (1 - D)Y &= (1 - D)DY_1 + (1 - D)^2 Y_0 = (1 - D)Y_0 \end{aligned}$$

since $Y = DY_1 + (1 - D)Y_0$. Thus,

$$\mathbb{E} \left[\frac{DY}{p(\mathbf{X})} \middle| \mathbf{X} \right] = \frac{1}{p(\mathbf{X})} \mathbb{E} [DY_1 | \mathbf{X}] \quad (3.1)$$

$$\mathbb{E} \left[\frac{(1 - D)Y}{1 - p(\mathbf{X})} \middle| \mathbf{X} \right] = \frac{1}{1 - p(\mathbf{X})} \mathbb{E} [(1 - D)Y_0 | \mathbf{X}]. \quad (3.2)$$

Now, by iterated expectations and *Assumption 3.1*,

$$\begin{aligned} \mathbb{E}[DY_1 | \mathbf{X}] &= \mathbb{E}_{D|\mathbf{X}} [\mathbb{E}(DY_1 | D, \mathbf{X})] = \mathbb{E}_{D|\mathbf{X}} [D \mathbb{E}(Y_1 | D, \mathbf{X})] = \mathbb{E}_{D|\mathbf{X}} [D \mathbb{E}(Y_1 | \mathbf{X})] \\ &= \mathbb{E}[D | \mathbf{X}] \mathbb{E}[Y_1 | \mathbf{X}] = p(\mathbf{X}) \mathbb{E}[Y_1 | \mathbf{X}] \end{aligned}$$

where the final equality uses $\mathbb{E}[D | \mathbf{X}] = \mathbb{P}(D = 1 | \mathbf{X})$. Similarly,

$$\begin{aligned} \mathbb{E}[(1 - D)Y_0 | \mathbf{X}] &= \mathbb{E}_{D|\mathbf{X}} [\mathbb{E}\{(1 - D)Y_0 | D, \mathbf{X}\}] = \mathbb{E}_{D|\mathbf{X}} [(1 - D) \mathbb{E}(Y_0 | D, \mathbf{X})] \\ &= \mathbb{E}_{D|\mathbf{X}} [(1 - D) \mathbb{E}(Y_0 | \mathbf{X})] = \mathbb{E}[1 - D | \mathbf{X}] \mathbb{E}[Y_0 | \mathbf{X}] \\ &= [1 - p(\mathbf{X})] \mathbb{E}[Y_0 | \mathbf{X}] \end{aligned}$$

Substituting these expressions for $\mathbb{E}[DY_1 | \mathbf{X}]$ and $\mathbb{E}[(1 - D)Y_0 | \mathbf{X}]$ into (3.1) and (3.2)

$$\mathbb{E} \left[\frac{DY}{p(\mathbf{X})} \middle| \mathbf{X} \right] = \mathbb{E}(Y_1 | \mathbf{X}), \quad \mathbb{E} \left[\frac{(1 - D)Y}{1 - p(\mathbf{X})} \middle| \mathbf{X} \right] = \mathbb{E}(Y_0 | \mathbf{X})$$

so we see that

$$\begin{aligned} ATE(\mathbf{X}) &\equiv \mathbb{E}(Y_1 - Y_0 | \mathbf{X}) = \mathbb{E} \left[\frac{DY}{p(\mathbf{X})} - \frac{(1 - D)Y}{1 - p(\mathbf{X})} \middle| \mathbf{X} \right] \\ &= \mathbb{E} \left[\frac{DY \{1 - p(\mathbf{X})\} - (1 - D)Y p(\mathbf{X})}{p(\mathbf{X}) \{1 - p(\mathbf{X})\}} \middle| \mathbf{X} \right] \\ &= \mathbb{E} \left[\frac{DY - DY p(\mathbf{X}) - Y p(\mathbf{X}) + DY p(\mathbf{X})}{p(\mathbf{X}) \{1 - p(\mathbf{X})\}} \middle| \mathbf{X} \right] \\ &= \mathbb{E} \left[\frac{\{D - p(\mathbf{X})\} Y}{p(\mathbf{X}) \{1 - p(\mathbf{X})\}} \middle| \mathbf{X} \right]. \end{aligned}$$

Therefore, taking iterated expectations,

$$\text{ATE} = \mathbb{E}_{\mathbf{X}} [\text{ATE}(\mathbf{X})] = \mathbb{E}_{\mathbf{X}} \left(\mathbb{E} \left[\frac{\{D - p(\mathbf{X})\} Y}{p(\mathbf{X}) \{1 - p(\mathbf{X})\}} \middle| \mathbf{X} \right] \right) = \mathbb{E} \left[\frac{\{D - p(\mathbf{X})\} Y}{p(\mathbf{X}) \{1 - p(\mathbf{X})\}} \right].$$

□

3.6 Estimation by Propensity Score Weighting

Suppose we already have a consistent estimator $\hat{p}(\cdot)$ of the propensity score. Then,

$$\widehat{\text{ATE}}_{PSW} \equiv \frac{1}{n} \sum_{i=1}^n \frac{[D_i - \hat{p}(\mathbf{X}_i)] Y_i}{\hat{p}(\mathbf{X}_i) [1 - \hat{p}(\mathbf{X}_i)]}$$

where *PSW* stands for *propensity score weighting* is a consistent estimator of the ATE under [Assumption 3.1](#), [Assumption 3.2](#), and appropriate regularity conditions. But how can we estimate the propensity score? If \mathbf{X} is discrete and only takes on a small number of values, we can estimate the propensity score directly using the sample fraction of observations with $\mathbf{X} = \mathbf{x}$. This approach is no longer possible when any of the elements of \mathbf{X} is continuous and can perform poorly even for discrete \mathbf{X} if some values \mathbf{x} are shared by only a small number of people in the sample. A common model-based approach is to fit a “flexible” logit model, including levels, squares, and interactions of \mathbf{X} . Although fairly widespread and convenient, this approach has the potential to mask failures of overlap: the logit model will never give $p(\mathbf{X}) = 0$ or 1 regardless of whether there are values of \mathbf{x} for which everyone in the sample is either treated or untreated. Moreover, the particular logit model that we specify could be a poor reflection of the true propensity score. Another approach uses non-parametric methods, either series or kernel based, to estimate the propensity score. While less prone to mis-specification than model-based approaches, non-parametric methods perform poorly when \mathbf{X} is high-dimensional. Regardless of the particular method used, inference for propensity score weighting is somewhat complicated by the first-stage estimation of $\hat{p}(\mathbf{X})$. An easy solution is to bootstrap pairs (\mathbf{X}_i, Y_i) .

3.7 Regression Adjustment versus Propensity Score Weighting

In theory, both [Theorem 3.1](#) and [Theorem 3.2](#) identify the *same* quantity, namely the ATE.³ In practice, however, because they require us to use the data in different ways, estimators based on regression adjustment and propensity score weighting will differ,

³If [Assumption 3.2](#) fails and we are forced to restrict attention individuals with values of \mathbf{X} for which overlap holds, then both theorems identify the ATE for this *restricted* population.

sometimes substantially. Recall that regression adjustment requires us to model and estimate the conditional mean of Y given $(D = 0, \mathbf{X})$ and $(D = 1, \mathbf{X})$ whereas propensity score weighting requires us to model and estimate the conditional probability that $D = 1$ given \mathbf{X} . A particular challenge for propensity score weighting is values of $\hat{p}(\mathbf{X}_i)$ that are close to zero or one, as this causes the fraction in $\widehat{\text{ATE}}_{PSW}$ to become unstable.

3.8 Don't condition on an intermediate outcome!

The key message of this chapter is that conditioning on the right information can allow us to identify causal effects even when treatment is not randomly assigned. The key message of this section is that conditioning on the *wrong* information can lead us to draw erroneous causal conclusions even when treatment *is indeed* randomly assigned. This problem is commonly known as **bad control** or **conditioning on an intermediate outcome**. We'll use a simple example to explain the problem and how to avoid it. For simplicity our discussion will be limited to a binary covariate X that is potentially a “bad control.” Very similar reasoning applies to any covariate, binary or not.

Gwynnaeth attended a bilingual French and English high school in Canada. She is now a university senior lecturer and earns a good living. Did attending a bilingual high school *cause* her earnings to be higher than they otherwise would have been? Let Y be a person's wage, and define $D = 1$ if she attends a bilingual high school and zero otherwise. Gwynnaeth chose to attend a bilingual high school: her D was not randomly assigned. But imagine that we were to carry out an experiment in which we *did* randomly assign D , sending half of a group of students to a bilingual high school and the rest to a regular high school. Since $D \perp\!\!\!\perp (Y_0, Y_1)$, we have $\mathbb{E}(Y_0|D) = \mathbb{E}(Y_0)$ and $\mathbb{E}(Y_1|D) = \mathbb{E}(Y_1)$. Thus,

$$\mathbb{E}(Y|D = 1) - \mathbb{E}(Y|D = 0) = \mathbb{E}(Y_1|D = 1) - \mathbb{E}(Y_0|D = 0) = \mathbb{E}(Y_1 - Y_0) = \text{ATE}$$

since $Y = (1 - D)Y_0 + DY_1$. Because students in this hypothetical experiment are randomly assigned to high schools, we don't need to condition on *anything* to identify the average treatment effect D on Y : a simple comparison of means suffices. But what would happen if we nevertheless *did* choose to condition on something?

Given that she is a university senior lecturer, it will come as no surprise that Gwynnaeth attended university herself. Let $X = 1$ if a person attended university and zero otherwise. Should we condition on X to estimate the ATE in our hypothetical experiment? *Absolutely not!* College attendance X is an **intermediate outcome** aka a **bad control**. Because D causes X as well as Y , the treatment D is no longer randomly assigned if we *condition* on X . In other words, conditioning on X introduces selection bias that was not present unconditionally. We will examine this in two ways: first intuitively using a simple stylized model, and then mathematically, building on our earlier derivations. Consider

the following stylized model:

- (i) Two factors increase a person's wage: knowledge K and innate ability A .
- (ii) Attending a bilingual high school increases K more than attending a regular one.
- (iii) The top 30% of people in the population distribution of $(K + A)$ attend university.

Because D was randomly assigned it is independent of A . This is no longer true, however, conditional on X . First consider the group of people from our experiment who attended university ($X = 1$). Among them, those who didn't attend a bilingual high school ($D = 0$) will have *higher* average ability than those who did ($D = 1$). Why is this the case? Our second assumption was that those who didn't attend a bilingual school end up with a lower value of K , on average, than those who did. Thus, for them to make it into the top 30% of $(K + A)$ requires a *higher* value of A . Putting it another way, if you did attend a bilingual school, then you can make it into the top 30% of $(K + A)$ with a lower value of A . Because those with $(D = 1, X = 1)$ have lower ability than those with $(D = 0, X = 1)$ and lower ability implies lower wages,

$$\mathbb{E}[Y|D = 1, X = 1] - \mathbb{E}[Y|D = 0, X = 1] < \mathbb{E}[Y_1|X = 1] - \mathbb{E}[Y_0|X = 1] = \text{ATE}(X = 1).$$

A similar argument shows that, that among those who did not attend university, those with $D = 1$ will have lower average ability than those with $D = 0$.⁴ It follows that

$$\mathbb{E}[Y|D = 1, X = 0] - \mathbb{E}[Y|D = 0, X = 0] < \mathbb{E}[Y_1|X = 0] - \mathbb{E}[Y_0|X = 0] = \text{ATE}(X = 0).$$

In this simple model, conditioning on university attendance would lead us to *understate* the true treatment effect. Now that we understand the basic intuition, we'll take a more mathematical look at the problem of a bad control. The following lemma and discussion are a special case of [Wooldridge \(2005\)](#).

Lemma 3.1. *Let X be a binary RV and suppose that $\mathbb{E}(Y_j) = \mathbb{E}(Y_j|D)$ for $j = 0, 1$. If $\mathbb{E}(Y_j|X, D) = \mathbb{E}(Y_j|X)$ for $j = 0, 1$ then at least one of the following must hold:*

- (i) $X \perp\!\!\!\perp D$
- (ii) $\mathbb{E}(Y_j|X) = \mathbb{E}(Y_j)$ for $j = 0, 1$

Proof of Lemma 3.1. Since Y_j is mean independent of D for $j = 0, 1$ and X is binary, the law of iterated expectations gives

$$\begin{aligned} \mathbb{E}(Y_1) &= \mathbb{E}(Y_1|D) = \mathbb{E}_{X|D} [\mathbb{E}(Y_1|D, X)] \\ &= \mathbb{E}(Y_1|D, X = 0)\mathbb{P}(X = 0|D) + \mathbb{E}(Y_1|D, X = 1)\mathbb{P}(X = 1|D) \end{aligned}$$

⁴If you did not make it into the top 30% of the distribution of $(K + A)$ in spite of receiving the extra boost to K that comes from $D = 1$, then you must have had a low value of A .

and similarly for Y_0 . Further imposing that (Y_0, Y_1) are mean independent of D given X

$$\mathbb{E}(Y_0) = \mathbb{E}(Y_0|X=0)\mathbb{P}(X=0|D=d) + \mathbb{E}(Y_0|X=1)\mathbb{P}(X=1|D=d) \quad (3.3)$$

$$\mathbb{E}(Y_1) = \mathbb{E}(Y_1|X=0)\mathbb{P}(X=0|D=d) + \mathbb{E}(Y_1|X=1)\mathbb{P}(X=1|D=d). \quad (3.4)$$

The left-hand sides of (3.3) and (3.4) do not depend on the value d that the treatment D takes on. Thus, to avoid a contradiction between $\mathbb{E}(Y_j|D) = \mathbb{E}(Y_j)$ and $\mathbb{E}(Y_j|X, D) = \mathbb{E}(Y_j|X)$, the RHS *cannot* depend on d either. There are only two ways that this is possible. The first is if $X \perp\!\!\!\perp D$ so that $\mathbb{P}(X=x|D=d) = \mathbb{P}(X=x)$ and

$$\begin{aligned} \mathbb{E}(Y_0) &= \mathbb{E}(Y_0|X=0)\mathbb{P}(X=0) + \mathbb{E}(Y_0|X=1)\mathbb{P}(X=1) \\ \mathbb{E}(Y_1) &= \mathbb{E}(Y_1|X=0)\mathbb{P}(X=0) + \mathbb{E}(Y_1|X=1)\mathbb{P}(X=1). \end{aligned}$$

If X and D are *dependent*, then the only way that the RHS (3.3) and (3.4) could *not* involve d is if $\mathbb{E}(Y_1|X=0) = \mathbb{E}(Y_1|X=1) = \mathbb{E}(Y_1)$ and similarly for Y_0 , so that

$$\begin{aligned} \mathbb{E}(Y_0|X=0)\mathbb{P}(X=0|D=d) + \mathbb{E}(Y_0|X=1)\mathbb{P}(X=1|D=d) &= \mathbb{E}(Y_0) \\ \mathbb{E}(Y_1|X=0)\mathbb{P}(X=0|D=d) + \mathbb{E}(Y_1|X=1)\mathbb{P}(X=1|D=d) &= \mathbb{E}(Y_1) \end{aligned}$$

since $\mathbb{P}(X=0|D=d) + \mathbb{P}(X=1|D=d) = 1$ for any value of d . □

Lemma 3.1 tells us that if treatment is randomly assigned, then any covariate X that is both related to treatment *and* affects the average potential outcomes is *necessarily* a bad control. Given that D is mean independent of (Y_0, Y_1) , such an X cannot satisfy $\mathbb{E}(Y_j|X, D) = \mathbb{E}(Y_j|X)$, the selection on observables assumption. This means that we *cannot* identify the ATE by conditioning on X and using, for example, regression adjustment or propensity score weighting. In our example from above, college attendance (X) was both affected by attending a bilingual high school (D) and in turn affected wages. Given that D was randomly assigned, the lemma shows that college attendance is a bad control in the wages and high-school experiment.

Lemma 3.1 does *not* say that conditioning on a covariate that is related to D and Y is always bad. Indeed the whole point of this chapter is to try to eliminate selection bias by finding covariates that *are* related to D and Y . The lemma concerns a setting where we have already solved the selection problem by randomly assigning D . It tells us when conditioning on X would *introduce* selection bias that was not there to begin with. This may strike you as odd: why would we bother to condition on X if we already knew that the treatment had been randomly assigned? There are two answers to this question. First, it is fairly common in practice for researchers to condition on covariates when analyzing experimental data, either to estimate conditional ATEs for people with different characteristics or to reduce the variance of their overall ATE estimator by “projecting

out” sources of noise in Y . [Lemma 3.1](#) tells us that this is perfectly fine *provided that these covariates were measured before assigning the treatment*: because D is randomly assigned, we know that any *pre-existing* characteristics of individuals, e.g. sex or age, will be independent of treatment and hence cannot be bad controls.

Second, the reasoning used in our proof of [Lemma 3.1](#) also applies to settings in which D is not randomly assigned. Suppose that we have a set of “good controls” \mathbf{W} that satisfy [Assumption 3.1](#), i.e. (Y_0, Y_1) are mean independent of D given \mathbf{W} . Now suppose that we are considering adding an *additional* binary variable X to our set of controls. We should only add X if (Y_0, Y_1) are mean independent of D given the *full* set of controls (\mathbf{W}, X) . Suppose this is the case. Then, by iterated expectations and our two of mean independence assumptions,

$$\begin{aligned}\mathbb{E}(Y_1|\mathbf{W}) &= \mathbb{E}(Y_1|\mathbf{W}, D) = \mathbb{E}_{X|\mathbf{W}, D} [\mathbb{E}(Y_1|\mathbf{W}, D, X)] \\ &= \mathbb{E}(Y_1|\mathbf{W}, D, X = 0)\mathbb{P}(X = 0|\mathbf{W}, D) + \mathbb{E}(Y_1|\mathbf{W}, D, X = 1)\mathbb{P}(X = 1|\mathbf{W}, D) \\ &= \mathbb{E}(Y_1|\mathbf{W}, X = 0)\mathbb{P}(X = 0|\mathbf{W}, D) + \mathbb{E}(Y_1|\mathbf{W}, X = 1)\mathbb{P}(X = 1|\mathbf{W}, D)\end{aligned}$$

and similarly for Y_0 , yielding

$$\begin{aligned}\mathbb{E}(Y_0|\mathbf{W}) &= \mathbb{E}(Y_0|\mathbf{W}, X = 0)\mathbb{P}(X = 0|\mathbf{W}, D) + \mathbb{E}(Y_0|\mathbf{W}, X = 1)\mathbb{P}(X = 1|\mathbf{W}, D) \\ \mathbb{E}(Y_1|\mathbf{W}) &= \mathbb{E}(Y_1|\mathbf{W}, X = 0)\mathbb{P}(X = 0|\mathbf{W}, D) + \mathbb{E}(Y_1|\mathbf{W}, X = 1)\mathbb{P}(X = 1|\mathbf{W}, D).\end{aligned}$$

The left hand sides of these equations do not depend on D . By reasoning similar to that used in the proof of [Lemma 3.1](#), the only way that the right hand sides could *not* depend on D is if either $X \perp\!\!\!\perp D|\mathbf{W}$ or $\mathbb{E}(Y_j|\mathbf{W}, X) = \mathbb{E}(Y_j|\mathbf{W})$. If X is determined after D , it is unlikely that the first of these conditions hold. If X satisfies the second condition, then conditioning on it is completely irrelevant in any case: it will neither help us to identify ATEs, conditional or unconditional, nor will it improve the precision of our estimates.

Chapter 4

The Local Average Treatment Effects (LATE) Model

In [section 1.8](#) we showed that an OLS regression of Y on D does not in general identify the ATE: selection bias is the norm rather than the exception in social science. One possible solution, considered in [chapter 3](#) is to make the *selection on observables* assumption. Under this assumption, conditioning on observed characteristics \mathbf{X} suffices to break any dependence between D and (Y_0, Y_1) . Selection on observables, however, is a very strong assumption. How likely is it that we truly observe *all* the factors that create dependence between D and (Y_0, Y_1) ? As an alternative to selection on observables, this chapter considers the use of an *instrumental variable* Z to identify causal effects. For simplicity we focus on the case where Z , like D , is binary. We first review the “textbook” homogeneous effects IV model before addressing our key question for this chapter: what does an instrumental variable identify in a world of heterogeneous effects?

4.1 Instrumental Variables with Homogeneous Effects

Suppose that $Y = \alpha + \beta D + U$, where (Y, D) are observed random variables, U is an unobserved random variable, and (α, β) are unknown constants. Under standard conditions, the OLS estimator for β converges in probability to

$$\beta_{OLS} = \frac{\text{Cov}(D, Y)}{\text{Var}(D)} = \frac{\beta \text{Cov}(D, D) + \text{Cov}(D, U)}{\text{Var}(D)} = \beta + \frac{\text{Cov}(D, U)}{\text{Var}(D)}$$

which does not equal β unless $\text{Cov}(D, U) = 0$. Suppose we have an instrumental variable Z such that $\text{Cov}(Z, U) = 0$ (exogeneity) and $\text{Cov}(Z, D) \neq 0$ (relevance). Then, under standard conditions, the instrumental variables (IV) estimator of β converges to

$$\beta_{IV} = \frac{\text{Cov}(Z, Y)}{\text{Cov}(Z, D)} = \frac{\beta \text{Cov}(Z, D) + \text{Cov}(Z, U)}{\text{Cov}(Z, D)} = \beta + \frac{\text{Cov}(Z, U)}{\text{Cov}(Z, D)} = \beta.$$

What do we make of $Y = \alpha + \beta D + U$ in light of our discussion of potential outcomes from above? This is a **homogeneous treatment effects** model. In other words the model (implicitly) assumes that the treatment effect is the same for everyone. We can express this model in the potential outcomes notation from above as follows. To find Y_0 , set $D = 0$; to find Y_1 , set $D = 1$. This gives

$$Y_0 \equiv \alpha + U, \quad Y_1 \equiv \alpha + \beta + U \implies Y_1 - Y_0 = \beta$$

so the ATE equals the *constant* β . But what if the assumption of homogeneous treatment effects is incorrect? Does IV still identify a meaningful causal quantity? To answer this question, we will drop the assumption that $Y = \alpha + \beta D + U$ where (α, β) are constants and study the behavior of the **IV estimand** $\beta_{IV} \equiv \text{Cov}(Z, Y) / \text{Cov}(Z, D)$ under heterogeneous treatment effects.

When D and Z are both binary, as we will assume they are in these notes, the IV estimand can be written in a simpler form. Applying [Lemma 1.1](#) to the numerator and denominator of β_{IV}

$$\beta_{IV} = \frac{p(1-p) [\mathbb{E}(Y|Z=1) - \mathbb{E}(Y|Z=0)]}{p(1-p) [\mathbb{E}(D|Z=1) - \mathbb{E}(D|Z=0)]} = \frac{\mathbb{E}(Y|Z=1) - \mathbb{E}(Y|Z=0)}{\mathbb{E}(D|Z=1) - \mathbb{E}(D|Z=0)} \quad (4.1)$$

where $p \equiv \mathbb{P}(Z=1)$. The rightmost fraction in [\(4.1\)](#) is often called the **Wald estimand**. Substituting sample means for population expectations gives the **Wald estimator**, a convenient shorthand for $\hat{\beta}_{IV}$ in the binary-treatment/binary-instrument case.

4.2 Non-compliance and the Intent-to-Treat Effect

Loosely speaking, an instrument is a variable Z that only affects Y through its affect on D . In the preceding section we assumed that Z was **relevant**, $\text{Cov}(Z, D) \neq 0$, and **exogenous**, $\text{Cov}(Z, U) = 0$. These two assumptions sufficed to identify the ATE under homogeneous treatment effects. Because it only involves D and Z , the relevance assumption is unchanged in a heterogeneous effects model, as we will see below. In contrast, the exogeneity assumption $\text{Cov}(Z, U) = 0$ explicitly involves the additive error term U from the homogeneous effects model. We will need to find alternative assumptions to take its place if we wish to allow different people to have different treatment effects. Before we can state these assumptions, however, we need to develop some terminology and definitions that are specific to the heterogeneous effects case.

While IV methods are routinely applied to observational datasets, the definitions we need to introduce are easiest understood by considering an experimental example. The influential “Moving to Opportunity” (MTO) intervention from the mid 1990s offered vouchers to families living in high-poverty neighborhoods that would allow them

to relocate to low-poverty areas. A number of influential recent papers in economics have studied the causal effect of the MTO intervention, e.g. on labor market outcomes later in life. Although vouchers were assigned at random, for obvious reasons families could neither be compelled to move if they received one nor prevented from moving if they did not. Nearly 50% of the families offered vouchers through the MOT intervention chose to remain in their original neighborhoods while 20% of those not offered a voucher nevertheless moved to a low-poverty neighborhood. This phenomenon is called **non-compliance**: subjects in social experiments can only be offered treatment rather than compelled to take it up.

Let $Z = 1$ for families who were offered a voucher and $D = 1$ for those that moved to a low-poverty neighborhood. Non-compliance means that Z may not equal D . Crucially, while Z was randomly assigned, D was not: families *chose* whether or not to move, and those who did likely differed in many ways from those who did not. For this reason, a naïve comparison of $\mathbb{E}[Y|D = 1]$ against $\mathbb{E}[Y|D = 0]$ will be polluted by selection bias, as detailed in [section 1.8](#). But what about conditioning on Z rather than D ? The so-called **intent-to-treat** estimand does exactly this, regressing Y on Z rather than D :

$$\text{ITT} \equiv \mathbb{E}[Y|Z = 1] - \mathbb{E}[Y|Z = 0].$$

Notice that the ITT is *precisely* the same thing as the numerator of the Wald estimand. Because Z was randomly assigned, the ITT identifies the ATE of Z on Y : the average causal effect of being *offered* a voucher on labor market outcomes.¹

There is nothing wrong with the ITT. It answers a perfectly well-defined causal question and is immune to selection bias. At the same time, the intervention was called *moving* to opportunity for a reason: we don't expect that being offered a voucher, on its own, could affect labor market outcomes for families that did not, in fact, relocate. In other words, it is the causal effect of D that truly interests us, not the causal effect of Z . Under **perfect compliance**, $Z = D$ so the ITT equals the ATE. If everyone who is offered a voucher moves, and everyone who is not offered a voucher stays put, then the effect of being offered a voucher is the same as the effect of actually moving. By driving a wedge between Z and D , however, non-compliance causes the ITT and ATE to differ. Recall that 50% of families offered a voucher ($Z = 1$) chose not to move ($D = 0$). Since $Y = (1 - D)Y_0 + DY_1$, we have

$$\begin{aligned} \mathbb{E}[Y|Z = 1] &= \mathbb{E}_{D|Z=1} [\mathbb{E}(Y|Z = 1, D)] \\ &= \mathbb{E}[Y_0|Z = 1, D = 0] \mathbb{P}(D = 0|Z = 1) + \mathbb{E}[Y_1|Z = 1, D = 1] \mathbb{P}(D = 1|Z = 1) \\ &= \frac{1}{2} \times \mathbb{E}(Y_0|Z = 1, D = 0) + \frac{1}{2} \times \mathbb{E}(Y_1|Z = 1, D = 1). \end{aligned}$$

¹See [section 2.3](#), changing the notation so that Z replaces D .

This calculation shows that $\mathbb{E}[Y|Z = 1]$ contains a mixture of Y_0 and Y_1 with mixing weights that depend on the extent of non-compliance. In effect, families that choose not to move despite being offered a voucher dilute $\mathbb{E}[Y|Z = 1]$ so that it averages over the outcomes of both treated and untreated families. Non-compliance causes similar challenges for interpreting $\mathbb{E}[Y|Z = 0]$. Since 20% of families who were not offered a voucher chose to relocate,

$$\begin{aligned}\mathbb{E}[Y|Z = 0] &= \mathbb{E}_{D|Z=0} [\mathbb{E}(Y|Z = 0, D)] \\ &= \mathbb{E}[Y_0|Z = 0, D = 0] \mathbb{P}(D = 0|Z = 0) + \mathbb{E}[Y_1|Z = 0, D = 1] \mathbb{P}(D = 1|Z = 0) \\ &= \frac{4}{5} \times \mathbb{E}(Y_0|Z = 0, D = 0) + \frac{1}{5} \times \mathbb{E}(Y_1|Z = 0, D = 1).\end{aligned}$$

Again we obtain a mixture of Y_0 and Y_1 that depends on the extent of non-compliance. None of these calculations in any way contradicts our earlier claim that the ITT identifies the average causal effect of Z on Y . They merely show the challenges involved in trying to relate this quantity to the causal effect of interest, that of D on Y .

4.3 Compliers, Defiers, and Friends

In the previous section we showed that $\mathbb{E}[Y|Z = 0]$ and $\mathbb{E}[Y|Z = 1]$ each contain a mixture of Y_0 and Y_1 , making it difficult to relate the causal effect of being offered treatment, the ITT, to that of actually receiving it. But there is a second and more subtle challenge lurking here. Notice that $\mathbb{E}(Y|Z = 1)$ involves $\mathbb{E}(Y_0|Z = 1, D = 0)$ while $\mathbb{E}(Y|Z = 0)$ involves $\mathbb{E}(Y_0|Z = 0, D = 0)$. While these are both averages of Y_0 , they average over *different kinds of families*. Families who did not choose to move despite being offered a voucher ($Z = 1, D = 0$) are probably very different from those that didn't move but weren't offered a voucher ($Z = 0, D = 0$).² Perhaps the group with $Z = 1$ and $D = 0$ is much poorer, making it harder for them to move even with a voucher. Or perhaps they are less ambitious, and less willing to take advantage of the opportunity presented by moving to a new neighborhood. Whatever the reasons behind their choice not to move, we would likely expect their values of Y_0 to differ systematically from those of other families. Similarly, $\mathbb{E}[Y|Z = 0]$ involves $\mathbb{E}[Y_1|Z = 0, D = 1]$ while $\mathbb{E}[Y|Z = 1]$ involves $\mathbb{E}[Y_1|Z = 1, D = 1]$. Both are averages of Y_1 but, again, for different groups of families. Those that chose to move despite not being offered a voucher ($Z = 0, D = 1$) may be richer, making it easier for them to move. They might also be more ambitious given their willingness to “move to opportunity” without being offered a voucher.

To make it easier to discuss these different types of families, and as a lead-in to our derivations for IV under heterogeneous effects, we introduce the notion of **compliance**

²Remember that Z was randomly assigned, but D is something that families could *choose*.

types.³ The idea is to consider every possible rule that a family could use to decide whether or not to move, depending on whether they were offered a voucher. Mathematically, this involves listing every function that maps Z to D . Because both are binary, there are four possibilities. We give each a name corresponding to the “type” (T) of family that would adopt it:

$$\begin{aligned}\text{Never-taker: } T = n &\iff D(Z) = 0 \\ \text{Always-taker: } T = a &\iff D(Z) = 1 \\ \text{Complier: } T = c &\iff D(Z) = Z \\ \text{Defier: } T = d &\iff D(Z) = (1 - Z).\end{aligned}$$

In the MTO example, never-takers are the families that would *never* choose leave their current neighborhood, regardless of whether they are offered a voucher. Similarly, always-takers are families that would *always* choose to move to a lower-poverty area, regardless of whether they are offered a voucher. As their name suggests, compliers are the families that *comply* with their treatment offer: they move when given a voucher and stay put otherwise. Defiers are the most exotic of the four compliance types. These are families with a decidedly contrarian streak: they will *only* move to a low-poverty neighborhood when they are *not* offered a voucher.⁴

There is a crucial point about compliance types that is easy to miss: they are defined relative to a *particular* instrumental variable Z . Consider three different instruments intended to encourage families to move to low-poverty neighborhoods: Z_1 is an offer to provide help looking for a new apartment, Z_2 is a voucher worth \$500 per month towards rent for families that move, and Z_3 is title to a house, completely free of charge, in a low-poverty area. Someone who is a never-taker for Z_1 could easily be a complier for Z_2 , and someone who is a never-taker for Z_2 could easily be a complier for Z_3 . Our intuition is that Z_1 provides the weakest inducement to move while Z_3 provides the strongest. This point will be especially important below, where we will show that IV identifies the ATE for *compliers* when treatment effects can vary across individuals (or families).

4.4 The Local Average Treatment Effects Model

Having defined the four compliance types—always-takers, never-takers, compliers, and defiers—we can now state the assumptions that take the place of instrument exogeneity when treatment effects may be heterogeneous. The first is **unconfounded type**.

³Much of the literature refers to these as the “LATE Principal Strata.” The meaning of LATE will be clarified below, but *principal stratum* is exceedingly vague, so I prefer to avoid the term.

⁴Our definition of “defier” assumes that $Z = 1$ should make families *more* likely to move. If the reverse is true, simply replace Z with $\tilde{Z} = 1 - Z$.

Assumption 4.1 (Unconfounded Type). *For all compliance types $t \in \{a, c, n, d\}$*

$$\mathbb{P}(T = t) = \mathbb{P}(T = t|Z = 0) = \mathbb{P}(T = t|Z = 1).$$

Assumption 4.1 says that knowing the value of a person's instrumental variable tells us nothing about her type. If Z is randomly assigned, as in an experiment like MTO example, this assumption holds automatically: a person's type can be viewed as a pre-existing characteristic just like age, sex, or income. In examples where Z arises from observational data, **Assumption 4.1** is a substantive assumption that could in principle fail to hold. The second assumption is that there are **no defiers**.

Assumption 4.2 (No Defiers). $\mathbb{P}(T = d) = 0$.

Assumption 4.2 says that there are no defiers in the population: everyone is a never-taker, a complier, or an always-taker. If we view Z as an inducement that lowers the cost of taking up the treatment, this assumption entails that the instrument shifts everyone in the *same direction* or not at all. In the MTO example, this requires that offering a family a voucher cannot make them less likely to move. For this reason the no defiers assumption is sometimes called **monotonicity**. **Assumption 4.2** is natural in a model of rational choice, particularly if Z is a straightforward cost shifter such as the rent voucher from the MTO example. Nevertheless, it is still a restriction. Under the unconfounded type and no-defiers assumptions we can calculate the fraction of each compliance type in the population using the results of the following lemma.

Lemma 4.1. *Under Assumptions 4.1 and 4.2,*

$$\mathbb{P}(D = 1|Z = 1) = \mathbb{P}(T = a) + \mathbb{P}(T = c)$$

$$\mathbb{P}(D = 0|Z = 1) = \mathbb{P}(T = n)$$

$$\mathbb{P}(D = 1|Z = 0) = \mathbb{P}(T = a)$$

$$\mathbb{P}(D = 0|Z = 0) = \mathbb{P}(T = c) + \mathbb{P}(T = n)$$

Notice that **Lemma 4.1** directly identifies the share of never-takers and always-takers in the population: $\mathbb{P}(D = 0|Z = 1)$ equals $\mathbb{P}(T = n)$ while $\mathbb{P}(D = 1|Z = 0)$ equals $\mathbb{P}(T = a)$. To obtain the share of compliers, we take differences:

$$\mathbb{P}(T = c) = \mathbb{P}(D = 1|Z = 1) - \mathbb{P}(D = 1|Z = 0). \quad (4.2)$$

Notice that the right-hand side of (4.2) is *precisely* the denominator of the Wald estimand from (4.1): since D is binary, $\mathbb{E}(D|Z) = \mathbb{P}(D = 1|Z)$. To keep us from getting bogged down in book-keeping, I defer the proof of **Lemma 4.1** to **section 4.6**. The intuition behind the proof, however, is fairly straightforward. Consider $\mathbb{P}(D = 0|Z = 1)$. Because

we have assumed that there are no defiers ([Assumption 4.2](#)), a family that is offered a voucher ($Z = 1$) but doesn't move ($D = 0$) is a never-taker. Since we have assumed that the fraction of each type is the same among those who are offered a voucher and those who are not ([Assumption 4.1](#)), $\mathbb{P}(D = 0|Z = 1)$ tells us the overall fraction of never-takers in the population. The remaining equations follow by similar reasoning: see [section 4.6](#). The third and final assumption that replaces the more familiar instrument invalidity assumption from [section 4.1](#) is called **mean exclusion**.

Assumption 4.3 (Mean Exclusion). $\mathbb{E}[Y_j|Z = z, T = t] = \mathbb{E}[Y_j|T = t]$ for all (j, z, t) .

In words, [Assumption 4.3](#) says that the average values of (Y_0, Y_1) for any type t of family does not depend on whether they were offered a voucher. Mean exclusion does *not* require that Y is unaffected by Z . Because $Y = (1 - D)Y_0 + DY_1$, the instrument will necessarily affect Y if it affects D . What [Assumption 4.3](#) entails is that Z has no *direct* effect on Y : it may shift D but leaves Y_0 and Y_1 unchanged. In the MTO example, this requires that merely being offered a voucher has no effect on labor market outcomes for a family that does not choose to move. Similarly, it requires that not being offered a voucher has no effect on labor market outcomes for a family that does choose to move. Combining [Assumptions 4.1](#), [4.2](#), and [4.3](#) we can derive a lemma that will ultimately allow us to relate the ITT to the causal effect of D on Y .

Lemma 4.2. *Under Assumptions [4.1](#), [4.2](#), and [4.3](#).*

$$\begin{aligned}\mathbb{E}[Y|D = 1, Z = 1] &= \frac{\mathbb{P}(T = a)\mathbb{E}[Y_1|T = a] + \mathbb{P}(T = c)\mathbb{E}[Y_1|T = c]}{\mathbb{P}(T = a) + \mathbb{P}(T = c)} \\ \mathbb{E}[Y|D = 0, Z = 1] &= \mathbb{E}[Y_0|T = n] \\ \mathbb{E}[Y|D = 1, Z = 0] &= \mathbb{E}[Y_1|T = a] \\ \mathbb{E}[Y|D = 0, Z = 0] &= \frac{\mathbb{P}(T = n)\mathbb{E}[Y_0|T = n] + \mathbb{P}(T = c)\mathbb{E}[Y_0|T = c]}{\mathbb{P}(T = n) + \mathbb{P}(T = c)}\end{aligned}$$

[Lemma 4.2](#) relates the means of the observed outcome Y given D and Z to the means of the potential outcomes (Y_0, Y_1) for different types of families. In the MTO intervention, for example, the average value of Y for families that were not offered a voucher but moved to a low-poverty neighborhood nonetheless, $\mathbb{E}[Y|D = 1, Z = 0]$, identifies the average value of Y_1 for *always-takers*. Similarly, the average value of Y for families that were offered a voucher but chose not to move, $\mathbb{E}[Y|D = 0, Z = 1]$, identifies the average value of Y_0 for *never-takers*. So far we have used [Assumptions 4.1](#), [4.2](#), and [4.3](#) to derive two lemmas. [Lemma 4.1](#) tells us something about the denominator of the Wald estimand. And since $\mathbb{E}[Y|Z] = \mathbb{E}_{D|Z}[\mathbb{E}(Y|D, Z)]$ by the law of iterated expectations, [Lemma 4.2](#) tells us something about the numerator of the Wald estimator, i.e. the ITT. Before combining them, we require one further assumption.

Assumption 4.4 (Existence of Compliers). $\mathbb{P}(T = c) > 0$

By (4.2), Assumption 4.4 is equivalent to $\mathbb{P}(D = 1|Z = 1) \neq \mathbb{P}(D = 1|Z = 0)$ under Assumptions 4.1 and 4.2. To put it another way, the *lack* of a first-stage relationship between Z and D would indicate that there are *no compliers* in the population. We cannot carry out IV without compliers: notice that the Wald estimator is undefined if $\mathbb{E}(D|Z = 1) = \mathbb{E}(D|Z = 0)$. Because D is binary, $\text{Cov}(Z, D) = p(1 - p) [\mathbb{E}(D|Z = 1) - \mathbb{E}(D|Z = 0)]$. Thus, Assumption 4.4 is equivalent to the instrument relevance condition in the textbook homogeneous effects IV case from section 4.1. We are finally ready to answer our main question for the chapter: what does IV identify in a world of heterogeneous effects?

Theorem 4.1. *Under Assumptions 4.1–4.4,*

$$\frac{\mathbb{E}(Y|Z = 1) - \mathbb{E}(Y|Z = 0)}{\mathbb{E}(D|Z = 1) - \mathbb{E}(D|Z = 0)} = \mathbb{E}[Y_1 - Y_0|T = c]$$

Proof of Theorem 4.1. To begin, consider the numerator of the Wald Estimand. By the Law of Iterated Expectations,

$$\begin{aligned} \mathbb{E}(Y|Z = z) &= \mathbb{E}_{D|Z=z} [\mathbb{E}(Y|D, Z = z)] = \mathbb{E}(Y|D = 0, Z = z)\mathbb{P}(D = 0|Z = z) \\ &\quad + \mathbb{E}(Y|D = 1, Z = z)\mathbb{P}(D = 1|Z = z). \end{aligned} \quad (4.3)$$

Now that we have expressed $\mathbb{E}(Y|Z)$ in terms of $\mathbb{E}(Y|D, Z)$ and $\mathbb{P}(D|Z)$, we can substitute our results from Lemma 4.1 and Lemma 4.2 into (4.3) to relate the numerator of the Wald Estimand to the average potential outcomes $\mathbb{E}(Y_j|T = t)$ of the different types and their prevalence $\mathbb{P}(T = t)$ in the population. In particular:

$$\begin{aligned} \mathbb{E}(Y|Z = 1) &= \mathbb{E}(Y|D = 0, Z = 1)\mathbb{P}(D = 0|Z = 1) + \mathbb{E}(Y|D = 1, Z = 1)\mathbb{P}(D = 1|Z = 1) \\ &= \mathbb{P}(T = n)\mathbb{E}(Y_0|T = n) + [\mathbb{P}(T = a)\mathbb{E}(Y_1|T = a) + \mathbb{P}(T = c)\mathbb{E}(Y_1|T = c)] \end{aligned}$$

and similarly for $Z = 0$,

$$\begin{aligned} \mathbb{E}(Y|Z = 0) &= \mathbb{E}(Y|D = 0, Z = 0)\mathbb{P}(D = 0|Z = 0) + \mathbb{E}(Y|D = 1, Z = 0)\mathbb{P}(D = 1|Z = 0) \\ &= [\mathbb{P}(T = n)\mathbb{E}(Y_0|T = n) + \mathbb{P}(T = c)\mathbb{E}(Y_0|T = c)] + \mathbb{P}(T = a)\mathbb{E}(Y_1|T = a). \end{aligned}$$

Taking the differences of these two expressions, we find that

$$\mathbb{E}(Y|Z = 1) - \mathbb{E}(Y|Z = 0) = \mathbb{P}(T = c)\mathbb{E}(Y_1 - Y_0|T = c) \quad (4.4)$$

since $\mathbb{E}(Y_1|T = c) - \mathbb{E}(Y_0|T = c) = \mathbb{E}(Y_1 - Y_0|T = c)$ by the Linearity of Conditional Expectation. Now consider the denominator of the Wald Estimand. Since D is binary,

$\mathbb{E}(D|Z = z) = \mathbb{P}(D = 1|Z = z)$. Thus, by [Lemma 4.1](#) we obtain

$$\mathbb{E}(D|Z = 1) - \mathbb{E}(D|Z = 0) = \mathbb{P}(D = 1|Z = 1) - \mathbb{P}(D = 1|Z = 0) = \mathbb{P}(T = c). \quad (4.5)$$

Since $\mathbb{P}(T = c) \neq 0$ we can divide [\(4.4\)](#) by [\(4.5\)](#), completing the proof. \square

[Theorem 4.1](#) shows that IV does not in general identify the ATE in a heterogeneous treatment effects setting. Instead, it identifies the average treatment effect *for compliers*. In the MTO intervention, for example, IV would identify the average causal effect of moving to a low-poverty neighborhood for the kind of family that could be *induced* to move by offering them a voucher. This average effect for compliers is typically called the **local average treatment effect** or **LATE** for short. Accordingly, [Theorem 4.1](#) is sometimes called the LATE Theorem and Assumptions [4.1–4.4](#) the LATE assumptions. We discuss the interpretation of LATE in [section 4.5](#) below.

4.5 Who are the compliers? Why should we care?

As shown in [section 4.4](#), IV does *not* identify the ATE in a world of heterogeneous treatment effects. Instead it identifies the LATE: an average treatment effect for a particular *subset* of individuals, namely the compliers. So who are these compliers, and why should we care about them? The short answer is: we don’t know, and it is unclear whether we should. The key point to recognize is this: IV came first, and LATE came second. No one sat down and asked “how can I recover the average treatment effect for the compliers?” Instead the question was “suppose I run IV in a world of heterogeneous effects. Is there any way to give an interpretation to the result?” The answer, as we have seen, is *yes* but the interpretation is somewhat strained for several reasons. First, we cannot point to any individual in the sample and say “this is a complier.” A person with $(Z = 1, D = 1)$ could be a complier or an always-taker; a person with $(Z = 0, D = 0)$ could be a complier or a never-taker. Second, as discussed in [section 4.5](#) above, compliance is only defined relative to a *particular* instrumental variable. In the MTO intervention, families were offered a rent voucher that could be used to move to a low-poverty area. The LATE is specific not only to the fact that the IV was a rent voucher—as opposed, say, to an offer of assistance searching for an apartment—but also to the precise *amount* of the voucher. A \$500 per month rent voucher, for example, identifies a different LATE from a \$600 per month rent voucher if there are any families that would choose to move when offered the larger amount but not the smaller one.

While we cannot identify individual compliers, it is possible to say something about the so-called **compliant sub-population**, i.e. the population of compliers. First, as seen from [\(4.2\)](#), the denominator of the Wald estimator tells us the fraction of compliers in the population. If this fraction is large, the fact that IV identifies a LATE rather than

the ATE is less worrying: when nearly everyone is a complier, the two causal effects will likely be quite similar. Second, we can estimate the *average* characteristics of compliers. Suppose, for example, that we wanted to determine whether compliers are more likely to be women. Let W be a dummy variable that equals one if a person is female, zero otherwise. Then $\mathbb{P}(W = 1|T = c)$ is the share of women among compliers. By Bayes' Theorem, we obtain

$$\mathbb{P}(W = 1|T = c) = \frac{\mathbb{P}(T = c|W = 1)\mathbb{P}(W = 1)}{\mathbb{P}(T = c)} = \frac{\mathbb{P}(T = c|W = 1)\mathbb{P}(W = 1)}{\mathbb{E}(D|Z = 1) - \mathbb{E}(D|Z = 0)} \quad (4.6)$$

using $\mathbb{P}(T = c) = \mathbb{E}(D|Z = 1) - \mathbb{E}(D|Z = 0)$ from [Lemma 4.1](#). Now, $\mathbb{P}(W = 1)$ is simply the share of women in the population but what about $\mathbb{P}(T = c|W = 1)$? By an argument nearly identical to the proof of [Lemma 4.1](#), only with additional conditioning on $W = 1$, we can show that

$$\mathbb{P}(T = c|W = 1) = \mathbb{E}(D|Z = 1, W = 1) - \mathbb{E}(D|Z = 0, W = 1). \quad (4.7)$$

Combining [\(4.6\)](#) and [\(4.7\)](#)

$$\mathbb{P}(W = 1|T = c) = \mathbb{P}(W = 1) \left[\frac{\mathbb{E}(D|Z = 1, W = 1) - \mathbb{E}(D|Z = 0, W = 1)}{\mathbb{E}(D|Z = 1) - \mathbb{E}(D|Z = 0)} \right].$$

Since all of the quantities on the right-hand-side of this equation are observable, even though we cannot tell *which* women are compliers, we can nevertheless identify the share of women among compliers.

4.6 LATE Appendix: Proofs

Proof of [Lemma 4.1](#). By the Law of Total Probability

$$\begin{aligned} \mathbb{P}(D = d|Z = z) &= \sum_{t \in \{a, c, d, n\}} \mathbb{P}(D = d|Z = z, T = t)\mathbb{P}(T = t|Z = z) \\ &= \sum_{t \in \{a, c, n\}} \mathbb{P}(D = d|Z = z, T = t)\mathbb{P}(T = t) \end{aligned}$$

since $\mathbb{P}(T = t|Z = z) = \mathbb{P}(T = t)$ by [Assumption 4.1](#) and $\mathbb{P}(T = d) = 0$ by [Assumption 4.2](#). The key to the rest of the argument is that D is *completely determined* by Z and T : if I know that your offer is $Z = z$ and your type is $T = t$, then I know with certainty what your take-up decision will be. It follows that $\mathbb{P}(D = d|Z = z, T = t)$ is either zero or one, depending on the values of (d, z, t) .

Suppose first that $Z = 1$ but $D = 0$. Because you did not take up treatment, you cannot be an always-taker. Moreover, because $D \neq Z$ you cannot be a complier. Hence

$\mathbb{P}(D = 0|Z = 1, T = c) = \mathbb{P}(D = 0|Z = 1, T = a) = 0$. If you were a never-taker, then given $Z = 1$ you would indeed have $D = 0$: $\mathbb{P}(D = 0|Z = 1, T = n) = 1$. Therefore, we see that:

$$\begin{aligned}\mathbb{P}(D = 0|Z = 1) &= 0 \times \mathbb{P}(T = a) + 0 \times \mathbb{P}(T = c) + 1 \times \mathbb{P}(T = n) \\ &= \mathbb{P}(T = n).\end{aligned}$$

Now suppose that $Z = 0$ but $D = 1$. Because you took up treatment, you cannot be a never-taker. Moreover, because $Z \neq D$ you cannot be a complier. As a result, we see that $\mathbb{P}(D = 1|Z = 0, T = c) = \mathbb{P}(D = 1|Z = 0, T = n) = 0$. If you were an always-taker, you would indeed have $D = 1$: $\mathbb{P}(D = 1|Z = 1, T = a) = 1$. Hence,

$$\begin{aligned}\mathbb{P}(D = 1|Z = 0) &= 1 \times \mathbb{P}(T = a) + 0 \times \mathbb{P}(T = c) + 0 \times \mathbb{P}(T = n) \\ &= \mathbb{P}(T = a).\end{aligned}$$

Next suppose that $Z = 1$ and $D = 1$. Because you took treatment, we know that you cannot be a never-taker: $\mathbb{P}(D = 1|Z = 1, T = n) = 0$. You could, however, be a complier or an always-taker: $\mathbb{P}(D = 1|Z = 1, T = c) = \mathbb{P}(D = 1|Z = 1, T = a) = 1$. Hence,

$$\begin{aligned}\mathbb{P}(D = 1|Z = 1) &= 1 \times \mathbb{P}(T = a) + 1 \times \mathbb{P}(T = c) + 0 \times \mathbb{P}(T = n) \\ &= \mathbb{P}(T = a) + \mathbb{P}(T = c).\end{aligned}$$

Finally, suppose that $Z = 0$ and $D = 0$. Because you did not take up treatment, you cannot be an always-taker: $\mathbb{P}(D = 0|Z = 0, T = a) = 0$. You could, however, be a never-taker or a complier: $\mathbb{P}(D = 0|Z = 0, T = n) = \mathbb{P}(D = 0|Z = 0, T = c) = 1$. Hence,

$$\begin{aligned}\mathbb{P}(D = 0|Z = 0) &= 0 \times \mathbb{P}(T = a) + 1 \times \mathbb{P}(T = c) + 1 \times \mathbb{P}(T = n) \\ &= \mathbb{P}(T = c) + \mathbb{P}(T = n)\end{aligned}$$

completing the proof. □

Proof of Lemma 4.2. By the Law of Iterated Expectations,

$$\begin{aligned}\mathbb{E}[Y|D = d, Z = z] &= \mathbb{E}_{T|(D=d, Z=z)} [\mathbb{E}(Y|D = d, Z = z, T)] \\ &= \sum_{t \in \{a, c, n\}} \mathbb{E}(Y|D = d, Z = z, T = t) \mathbb{P}(T = t|D = d, Z = z)\end{aligned}\quad (4.8)$$

since $\mathbb{P}(T = d) = 0$ by [Assumption 4.2](#). In the proof of [Lemma 4.1](#) above, we examined the probabilities $\mathbb{P}(D = d|Z = z, T = t)$ in detail, arguing that they must be either zero or one, depending on whether the take-up and offer combination $(D = d, Z = z)$

is compatible with the type $T = t$. In contrast, the present argument involves $\mathbb{P}(T = t|D = d, Z = z)$. Fortunately, the two probabilities are related by the conditional version of Bayes' Theorem. In particular, using the fact that $\mathbb{P}(T = t|Z = z) = \mathbb{P}(T = t)$ by [Assumption 4.1](#), we have

$$\mathbb{P}(T = t|D = d, Z = z) = \frac{\mathbb{P}(D = d|Z = z, T = t)\mathbb{P}(T = t)}{\mathbb{P}(D = d|Z = z)}. \quad (4.9)$$

While it may appear that we have made things more complicated rather than less, the preceding equality is actually very useful: in [Lemma 4.1](#) we have already shown that the denominator is a sum of type probabilities $\mathbb{P}(T = t)$. Moreover, as argued above, the first term in the numerator is either zero or one depending on the values of (d, t, z) .

Before we can combine all of these ingredients, however, we first need to take a closer look at the expectation $\mathbb{E}(Y|D = d, Z = z, T = t)$. Recall from [\(1.2\)](#) that $Y = DY_1 + (1 - D)Y_0$. Taking conditional expectations of this equality, we have

$$\begin{aligned} \mathbb{E}[Y|D = 0, Z = z, T = t] &= \mathbb{E}[Y_0|D = 0, Z = z, T = t] \\ \mathbb{E}[Y|D = 1, Z = z, T = t] &= \mathbb{E}[Y_1|D = 1, Z = z, T = t] \end{aligned}$$

A key idea in our proof of [Lemma 4.1](#) above was that, given the way we have defined T , knowledge of a person's type t and her treatment offer z immediately implies her take-up decision d . In other words, D is a *function* of Z and T . For this reason, conditioning on D *in addition* to Z and T is *redundant*: a person's take-up decision cannot provide us with any further information given that we already know her treatment offer and type. It follows that we can drop D from the conditioning set in the preceding pair of equalities, yielding

$$\begin{aligned} \mathbb{E}[Y|D = 0, Z = z, T = t] &= \mathbb{E}[Y_0|Z = z, T = t] \\ \mathbb{E}[Y|D = 1, Z = z, T = t] &= \mathbb{E}[Y_1|Z = z, T = t]. \end{aligned}$$

There is one more simplification that we can apply to the expressions for $\mathbb{E}[Y|D, Z, T]$. [Assumption 4.3](#) states that the conditional mean of Y_0 and Y_1 does not depend on Z after conditioning on T : in other words the average potential outcomes for any type of individual (a, c, n) are unaffected by her treatment offer. Imposing this restriction, the preceding equalities become

$$\mathbb{E}[Y|D = 0, Z = z, T = t] = \mathbb{E}[Y_0|T = t] \quad (4.10)$$

$$\mathbb{E}[Y|D = 1, Z = z, T = t] = \mathbb{E}[Y_1|T = t]. \quad (4.11)$$

The remainder of the argument, though admittedly somewhat tedious, is just algebra.

For each pair of values (d, z) we first substitute either (4.10) or (4.11) into (4.8), depending on whether $D = 0$ or $D = 1$. We then substitute Lemma 4.1 into (4.9), and the result into (4.8).

Consider first $\mathbb{E}[Y|D = 0, Z = 1]$. Recall from the proof of Lemma 4.1 above that the probability $\mathbb{P}(D = 0|Z = 1, T = t)$ equals one for $T = n$ and zero for all other types $T = t$. (If you do not take when offered, you must be either a never-taker or defier, but we have assumed that there are no defiers.) By Equation 4.9, this implies that $\mathbb{P}(T = t|D = 0, Z = 1)$ equals zero for any $T \neq n$. Hence, substituting Equation 4.9 and (4.10) into (4.8), we obtain

$$\mathbb{E}[Y|D = 0, Z = 1] = \mathbb{E}[Y_0|T = n] \frac{\mathbb{P}(T = n)}{\mathbb{P}(D = 0|Z = 1)}$$

but since $\mathbb{P}(D = 0|Z = 1) = \mathbb{P}(T = n)$ by Lemma 4.1, the numerator and denominator cancel, leaving us with $\mathbb{E}[Y|D = 0, Z = 1] = \mathbb{E}[Y_0|T = n]$. Nearly identical reasoning for the case in which $(D = 1, Z = 0)$ gives $\mathbb{E}[Y|D = 1, Z = 0] = \mathbb{E}[Y_1|T = a]$, using the fact that anyone who takes up treatment when *not* offered must be an always-taker, given that we have assumed that there are no defiers.

Now consider $\mathbb{E}[Y|D = 1, Z = 1]$. Recall from the proof of Lemma 4.1 above that the probability $\mathbb{P}(D = 1|Z = 1, T = t)$ equals one for $T = c$ and $T = a$ but zero for $T = n$. By Equation 4.9, this implies that $\mathbb{P}(T = n|D = 0, Z = 1)$ equals zero. Hence, substituting Equation 4.9 and (4.10) into (4.8), we obtain

$$\mathbb{E}[Y|D = 1, Z = 1] = \mathbb{E}[Y_1|T = a] \frac{\mathbb{P}(T = a)}{\mathbb{P}(D = 1|Z = 1)} + \mathbb{E}[Y_1|T = c] \frac{\mathbb{P}(T = c)}{\mathbb{P}(D = 1|Z = 1)}.$$

The desired result follows since $\mathbb{P}(D = 1|Z = 1) = \mathbb{P}(T = a) + \mathbb{P}(T = c)$ by Lemma 4.1. A nearly identical argument gives the required expression for $\mathbb{E}[Y|D = 0, Z = 0]$, with never-takers replacing always-takers. \square

Chapter 5

Regression Discontinuity

Selection bias arises when people are free to choose whether or not they are treated. An idealized randomized controlled trial eliminates this bias by removing the element of choice: subjects are *compelled* to take the treatment or to refrain from doing so. Yet experiments are not the only situations in which people’s freedom to choose their own treatment is restricted. “Naturally occurring” constraints on self-selection are common and, under the right conditions, can provide a powerful tool for causal inference using observational data. Regression discontinuity methods exploit the existence of an administrative or legal cutoff that either completely or partially determines whether a person is treated. In the **sharp** regression discontinuity design, everyone on one side of the cutoff is treated and everyone on the other side is not, so the treatment variable “jumps” from zero to one at the cutoff. In a **fuzzy** regression discontinuity design, a person’s *probability* of treatment jumps at the cutoff. Either way, the basic idea is to compare those who are slightly above the cutoff to those who are slightly below. In the sharp design we compare mean outcomes for data near the threshold; in the fuzzy design we calculate the Wald estimate in the same region. This chapter draws mainly on [Hahn et al. \(2001\)](#). For a detailed survey of regression discontinuity methods, see [Lee and Lemieux \(2010\)](#).

In the discussion below, we will use some notation that has not appeared in earlier chapters. First, $\mathbb{1}\{A\}$ denotes the **indicator function** of the event A . If A occurs, this function equals one; otherwise it equals zero. Second, $\lim_{x \downarrow c} f(x)$ and $\lim_{x \uparrow c} f(x)$ denote the **one-sided limits** of a function f “from above” and “from below,” respectively. Some references call these the limits “from the right” and “from the left.” We will tacitly assume that both one-sided limits exist, so that if f has a discontinuity at c , it is a **jump discontinuity**, also known as a discontinuity of the first kind. This means that $\lim_{x \downarrow c} f(x) \neq \lim_{x \uparrow c} f(x)$.

5.1 The Sharp Regression Discontinuity Design

The best way to understand the sharp regression discontinuity design, or sharp RD for short, is with an example. The following is based on [Sekhri \(2020\)](#). Quite unlike the situation in the US, the most prestigious colleges in India are public. Given the choice, effectively everyone would choose to attend a public rather than a private institution. Admissions to public colleges are based on a threshold rule: anyone whose senior secondary school exam score exceeds the appropriate cutoff is admitted and anyone whose score falls below this threshold is rejected. The cutoffs vary from year-to-year and subject-to-subject, but crucially applicants do not know the cutoff and all applications are evaluated blind. This situation is depicted in [Figure 5.1](#). If your senior secondary school exam score X exceeds the admissions cutoff c , then you attend a public college ($D = 1$); if it does not, then you attend a less-prestigious private college ($D = 0$). In RD parlance, the variable X that determines a person's treatment status is called the **running variable**.

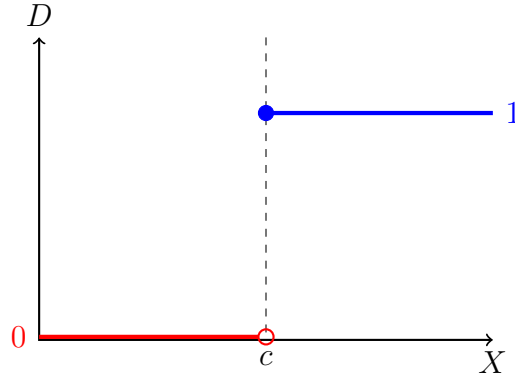


Figure 5.1: Treatments in the sharp regression discontinuity design. Anyone with $X \geq c$ is treated ($D = 1$) and no one with $X < c$ is treated ($D = 0$).

The key insight behind sharp RD is that students with exam scores just below the admissions cutoff are effectively identical to those with scores just above the cutoff. Whether you score 59.9% versus 60% on an exam is basically down to luck, but moving from just below the cutoff to just above it causes your treatment to jump discontinuously from zero to one. Hence, if we restrict our attention to students whose exam scores fall within a small window around the cutoff, it's *as if* some of them had been randomly assigned to attend an elite public college ($D = 1$) while the rest had been randomly assigned to attend a less-prestigious public college ($D = 0$). We formalize as follows.

Assumption 5.1 (Sharp RD Design). $D = \mathbb{1}\{X \geq c\}$ where X is an observed covariate, and c is a known threshold.

[Assumption 5.1](#) states that treatment is completely determined by the running variable X and a cutoff c that is known to the researcher. This is the situation depicted in

Figure 5.1. Note that this *implies* selection on observables (Assumption 3.1). We have:

$$\mathbb{E}[Y_j|D, X] = \mathbb{E}[Y_j|\mathbb{1}\{X \geq c\}, X] = \mathbb{E}[Y_j|X], \quad j = 0, 1$$

because $\mathbb{1}\{X \geq c\}$ is a measurable function of X . So why can't we just use the methods from chapter 3 to identify the ATE? The basic idea from chapter 3 was to compare people with the same value of X but different values of D . For this to be possible, we required not just selection on observables, but *overlap*: Assumption 3.2. Under Assumption 5.1, however, overlap fails completely: because anyone with $X \geq c$ is treated and no one with $X < c$ is treated, we cannot compare people with different values of D but the same value of X . This is why we need a different approach.

Assumption 5.2 (Continuity of Conditional Means). $\mathbb{E}[Y_0|X = x]$ and $\mathbb{E}[Y_1|X = x]$ are both continuous functions of x at the point $x = c$.

Assumption 5.2 formalizes the idea that students whose test scores fall in a sufficiently small neighborhood around the admissions cutoff are “effectively identical.” Say that Y is wage. We would expect that both Y_0 and Y_1 depend on X . Regardless of whether you attend a public or private college, your wage is likely related to your secondary school test score. Assumption 5.2 allows the potential outcomes to be related to X . What it rules out is a “jump” in Y_0 or Y_1 as X moves from just below to just above the cutoff c . How could such a jump occur? Suppose that it were possible for students to “precisely manipulate” their test scores. Perhaps I know both my own likely score and the cutoff. If I'm a highly-motivated person and my expected score falls just below what I know to be the cutoff, then perhaps I might choose to work extremely hard in the weeks before the exam to boost my score. Or if I'm a highly unscrupulous person, perhaps I might try to cheat in some way. Either of these possibilities would lead to a systematic difference between people with X just below c and those with X just above c . If being highly-motivated, or highly unscrupulous, affects wages later in life, this would lead to a violation of Assumption 5.2. In the context of Sekhri (2020), it is unlikely that students can “precisely manipulate” their secondary school test scores in this way because admissions thresholds vary both year-to-year and subject-to-subject. The fact that diligence causes higher test scores as well as later-life outcomes does *not* constitute a violation of Assumption 5.2 unless diligence jumps discontinuously at the admissions cutoff. In effect, we require that D is the *only thing* that jumps at c .

Under Assumption 5.2, we can identify the conditional ATE at $X = c$ by studying the observed conditional mean function $\mathbb{E}[Y|X = x]$ close to the cutoff. Figure 5.2 gives the visual intuition. Since anyone with $X < c$ is untreated, $\mathbb{E}[Y|X = x]$ equals $\mathbb{E}[Y_0|X = x]$ for $x < c$. Similarly, since anyone with $X \geq c$ is treated, $\mathbb{E}[Y|X = x]$ equals $\mathbb{E}[Y_1|X = x]$ when $x \geq c$. By assumption, neither $\mathbb{E}[Y_0|X = x]$ nor $\mathbb{E}[Y_1|X = c]$ has a discontinuity at $X = c$. Hence, if $\mathbb{E}[Y|X = x]$ *does* have a discontinuity at this point, there must be a

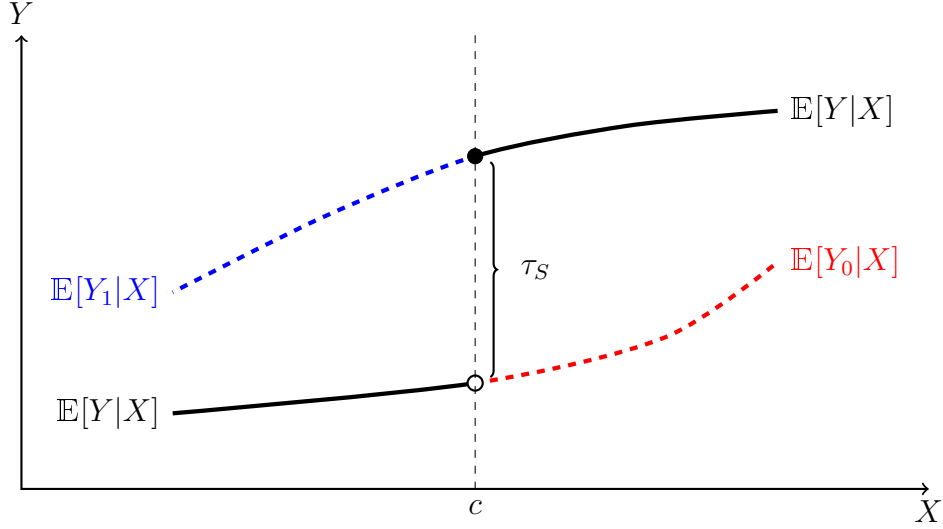


Figure 5.2: Outcomes in the sharp regression discontinuity design. For $X < c$ we observe $\mathbb{E}[Y|X] = \mathbb{E}[Y_0|X]$; for $X \geq c$ we observe $\mathbb{E}[Y|X] = \mathbb{E}[Y_1|X]$. The “jump” in $\mathbb{E}[Y|X]$ at c is the conditional ATE when $X = c$, namely $\tau_S \equiv \mathbb{E}[Y_1 - Y_0|X = c]$.

difference between $\mathbb{E}[Y_1|X = c]$ and $\mathbb{E}[Y_0|X = c]$. This difference, τ_S , is the conditional ATE at $X = c$. In the example from [Sekhri \(2020\)](#) discussed above, τ_S is the causal effect of attending an elite public institution on later-life wages for a certain kind of person: someone whose secondary school exam results are *close to the admissions cutoff*. This is a well-defined causal effect, but it may differ from the overall average treatment effect.

Theorem 5.1 (Sharp Regression Discontinuity). *Under Assumptions 5.1 and 5.2,*

$$\tau_S \equiv \mathbb{E}[Y_1 - Y_0|X = c] = \lim_{x \downarrow c} \mathbb{E}[Y|X = x] - \lim_{x \uparrow c} \mathbb{E}[Y|X = x].$$

Proof of Theorem 5.1. By [Assumption 5.1](#), we can re-write (1.2) as

$$Y = (1 - D)Y_0 + DY_1 = \mathbb{1}\{X < c\} Y_0 + \mathbb{1}\{X \geq c\} Y_1.$$

Taking conditional expectations of both sides, it follows that

$$\begin{aligned} \mathbb{E}[Y|X = x] &= \mathbb{E}[\mathbb{1}\{X < c\} Y_0 + \mathbb{1}\{X \geq c\} Y_1 | X = x] \\ &= \mathbb{1}\{x < c\} \mathbb{E}[Y_0|X = x] + \mathbb{1}\{x \geq c\} \mathbb{E}[Y_1|X = x]. \end{aligned}$$

Under [Assumption 5.2](#), both $\mathbb{E}[Y_0|X = x]$ and $\mathbb{E}[Y_1|X = x]$ are continuous at c . Hence,

$$\begin{aligned} \lim_{x \uparrow c} \mathbb{E}[Y_0|X = x] &= \lim_{x \downarrow c} \mathbb{E}[Y_0|X = x] = \mathbb{E}[Y_0|X = c] \\ \lim_{x \uparrow c} \mathbb{E}[Y_1|X = x] &= \lim_{x \downarrow c} \mathbb{E}[Y_1|X = x] = \mathbb{E}[Y_1|X = c]. \end{aligned}$$

Now, since $\lim_{x \uparrow c} \mathbb{1}\{x < c\} = 1$ while $\lim_{x \uparrow c} \mathbb{1}\{x \geq c\} = 0$,

$$\begin{aligned} \lim_{x \uparrow c} \mathbb{E}[Y|X = x] &= \lim_{x \uparrow c} \mathbb{1}\{x < c\} \lim_{x \uparrow c} \mathbb{E}[Y_0|X = x] + \lim_{x \uparrow c} \mathbb{1}\{x \geq c\} \lim_{x \uparrow c} \mathbb{E}[Y_1|X = x] \\ &= \mathbb{E}[Y_0|X = c]. \end{aligned}$$

Similarly, since $\lim_{x \downarrow c} \mathbb{1}\{x \geq c\} = 1$ while $\lim_{x \downarrow c} \mathbb{1}\{x < c\} = 0$,

$$\begin{aligned} \lim_{x \downarrow c} \mathbb{E}[Y|X = x] &= \lim_{x \downarrow c} \mathbb{1}\{x < c\} \lim_{x \downarrow c} \mathbb{E}[Y_0|X = x] + \lim_{x \downarrow c} \mathbb{1}\{x \geq c\} \lim_{x \downarrow c} \mathbb{E}[Y_1|X = x] \\ &= \mathbb{E}[Y_1|X = c]. \end{aligned}$$

The result follows by subtracting the equality for $\lim_{x \uparrow c} \mathbb{E}[Y|X = x]$ from that for $\lim_{x \downarrow c} \mathbb{E}[Y|X = x]$. \square

5.2 The Fuzzy Regression Discontinuity Design

Sometimes an administrative or legal cutoff strongly influences who is treated without completely determining D . [Jacob and Lefgren \(2004\)](#) provide an interesting example studying the causal effect of two remedial education policies in the Chicago Public Schools: *summer school*, requiring students to extend their school year through August, and *grade retention*, requiring students to repeat a year of school. Between 1996 and 1997, a policy was introduced to end the practice of “social promotion,” in which students are automatically advanced to the next school grade with no regard to their academic performance. The policy set achievement thresholds for third-grade, sixth-grade, and eighth-grade students. For these students to advance to the next grade, they were required to score sufficiently well on standardized tests of reading and mathematics. As written, the policy stated that students who exceeded the threshold in June would automatically advance to the next grade. Those who did not would be made to attend summer school and retested in August. Any student who fell below the threshold in the August retest would be required to repeat the previous grade.

So much for the intent of the policy. How was it actually administered in practice? In fact, test scores did *not* perfectly determine which students were assigned to remedial programs. Around 3% of students who fell below the June test threshold were given waivers that exempted them from summer school while roughly 14% of students who completed summer school but failed to exceed the required threshold in the August retest were exempted from repeating a grade. Moreover, some students were held back a grade despite exceeding the June admissions threshold. To accommodate examples like this one in which [Assumption 5.1](#) fails, the fuzzy RD design replaces it with the following.

Assumption 5.3 (Fuzzy RD Design).

$$\lim_{x \downarrow c} \mathbb{P}(D = 1|X = x) \neq \lim_{x \uparrow c} \mathbb{P}(D = 1|X = x)$$

where X is an observed covariate, and c is a known threshold.

Assumption 5.3 requires a jump in the *probability of treatment* at the cutoff c . Since D is binary, an equivalent and slightly more compact way of writing this assumption is

$$\lim_{x \downarrow c} \mathbb{E}[D|X = x] \neq \lim_{x \uparrow c} \mathbb{E}[D|X = x].$$

In the example of [Jacob and Lefgren \(2004\)](#), barely passing the August retest means that you will almost certainly be advanced to the next grade while barely failing means that you will very likely be held back. Thus **Assumption 5.3** is satisfied.

In [Jacob and Lefgren \(2004\)](#), the probability of treatment *falls* when test scores X exceed the cutoff c . For consistency with our treatment of sharp RD from above, however, the discussion below tacitly assumes that $\mathbb{P}(D = 1|X)$ jumps *upwards* at c . To accommodate the remedial education example in this context, we could simply re-define X and c . If S is a student’s test score, 100 is the maximum possible score, and 40 is the cutoff, for example, we would set $X = 100 - S$ and $c = 60$.

With this in mind, the rough intuition behind fuzzy RD is as follows. Because X does not completely determine D , some people with $X \geq c$ have $D = 0$ and some people with $X < c$ have $D = 1$. For this reason, both $\lim_{x \downarrow c} \mathbb{E}[Y|X = x]$ and $\lim_{x \uparrow c} \mathbb{E}[Y|X = x]$ contain a *mixture* of Y_0 and Y_1 , much like $E[Y|Z = 1]$ and $E[Y|Z = 0]$ in the ITT from (4.2). The Wald estimand from (4.1) “magnifies” the ITT by dividing it by the IV first-stage. Analogously, the fuzzy RD estimand τ_F divides the sharp RD estimand τ_S from [Theorem 5.1](#) by the equivalent fuzzy RD “first-stage,” in particular

$$\tau_F \equiv \frac{\lim_{x \downarrow c} \mathbb{E}[Y|X = x] - \lim_{x \uparrow c} \mathbb{E}[Y|X = x]}{\lim_{x \downarrow c} \mathbb{E}[D|X = x] - \lim_{x \uparrow c} \mathbb{E}[D|X = x]}. \quad (5.1)$$

Assumption 5.3 is effectively the “IV relevance” condition: it ensures that the denominator of (5.1) does not equal zero. The question remains: what if any *causal interpretation* can we give to τ_F ? The answer depends on exactly what we are willing to assume in addition to **Assumption 5.3**. We consider two possibilities. In the first, we make a relatively strong assumption under which τ_F equals the conditional ATE at $X = c$, namely $\mathbb{E}[Y_1 - Y_0|X = c]$, just as in the sharp RD case. In the second, we make a weaker but more plausible assumption under which τ_F is effectively a local average treatment effect.

Assumption 5.4. *There is some $\varepsilon > 0$ such that for $|x - c| < \varepsilon$,*

$$\mathbb{E}[Y_1 - Y_0|D, X = x] = \mathbb{E}[Y_1 - Y_0|X = x].$$

Assumption 5.4 states that the treatment effect $(Y_1 - Y_0)$ is mean-independent of D conditional on $X = x$ when x is sufficiently close to the threshold. This is very similar to selection on observables (**Assumption 3.1**). The main difference is that **Assumption 5.4** is only required for X in a small neighborhood of c while the selection on observables assumption was supposed to hold for all covariate values. Under this condition, a relatively straightforward argument shows that $\tau_F = \tau_S$, defined in **Theorem 5.1**.

Theorem 5.2. *Under Assumptions 5.2–5.4, the fuzzy RD estimand τ_F from (5.1) satisfies $\tau_F = \mathbb{E}[Y_1 - Y_0|X = c]$.*

Proof of Theorem 5.2. By (1.2), we have

$$\mathbb{E}[Y|X] = \mathbb{E}[Y_0 + D(Y_1 - Y_0)|X] = \mathbb{E}[Y_0|X] + \mathbb{E}[D(Y_1 - Y_0)|X].$$

Moreover, for $|x - c| < \varepsilon$, **Assumption 5.4** gives

$$\begin{aligned} \mathbb{E}[D(Y_1 - Y_0)|X = x] &= \mathbb{E}_{D|X=x} \{D\mathbb{E}[Y_1 - Y_0|X = x, D]\} = \mathbb{E}_{D|X=x} \{D\mathbb{E}[Y_1 - Y_0|X = x]\} \\ &= \mathbb{E}[D|X = x]\mathbb{E}[Y_1 - Y_0|X = x] \end{aligned}$$

by iterated expectations. Combining the two preceding equalities and taking the limit from above, it follows that

$$\begin{aligned} \lim_{x \downarrow c} \mathbb{E}[Y|X = x] &= \lim_{x \downarrow c} \mathbb{E}[Y_0|X = x] + \lim_{x \downarrow c} \mathbb{E}[D|X = x] \lim_{x \downarrow c} \{\mathbb{E}[Y_1|X = x] - \mathbb{E}[Y_0|X = x]\} \\ &= \mathbb{E}[Y_0|X = c] + \lim_{x \downarrow c} \mathbb{E}[D|X = x]\mathbb{E}[Y_1 - Y_0|X = c] \end{aligned}$$

by **Assumption 5.2**. Similarly, taking the limit from below,

$$\begin{aligned} \lim_{x \uparrow c} \mathbb{E}[Y|X = x] &= \lim_{x \uparrow c} \mathbb{E}[Y_0|X = x] + \lim_{x \uparrow c} \mathbb{E}[D|X = x] \lim_{x \uparrow c} \{\mathbb{E}[Y_1|X = x] - \mathbb{E}[Y_0|X = x]\} \\ &= \mathbb{E}[Y_0|X = c] + \lim_{x \uparrow c} \mathbb{E}[D|X = x]\mathbb{E}[Y_1 - Y_0|X = c]. \end{aligned}$$

The result follows by subtracting these expressions and solving for $\mathbb{E}[Y_1 - Y_0|X = c]$. \square

Assumption 5.4 rules out *selection on gains* near the threshold: it requires that individuals do not choose their treatment based on knowledge of their treatment effect $(Y_1 - Y_0)$. In the example from **Jacob and Lefgren (2004)** this would require there to be no relation between being granted an exemption from remedial education and the causal effect of remedial education, given a student's test score. This seems implausible. Indeed, **Jacob and Lefgren (2004)** found that students who received waivers and exemptions were systematically different in observable characteristics from those who did not. Giving τ_F a meaningful causal interpretation while allowing for selection on gains is more complicated. To do so, we view D as a *deterministic function* of x . For any possible value x that

X could take on, $D(x)$ tells us whether someone would take the treatment, $D(x) = 1$, or not, $D(x) = 0$. Although somewhat more complicated, this idea is very similar to the way that we viewed D as a deterministic function of a person's treatment offer z when defining the compliance types in [section 4.5](#) above. A person for whom $D(x)$ switches from 0 to 1 as x crosses the threshold c is a fuzzy RD *complier*. In the remedial education example, compliers are students who would receive remedial education if and only if their test scores warrant it, according to policy set down by the Chicago Public Schools. Under appropriate assumptions, τ_F turns out to identify the local average treatment effect of remedial education for *this type of student*. In particular, we impose the following.

Assumption 5.5 (No Defiers). *There is some $\varepsilon > 0$ such that $D(c - h) \leq D(c + h)$ for all $0 < h < \varepsilon$.*

Assumption 5.6 (Exclusion Restriction). *$(Y_1 - Y_0)$ and $D(x)$ are jointly independent of X near c .*

[Assumption 5.5](#) is analogous to [Assumption 4.2](#) in that it rules out *defiers*: people whose treatment status moves in the “wrong direction” as X crosses the threshold. In our running example, defiers would be students who only receive remedial education when their test scores *disqualify them* from doing so. [Assumption 5.6](#) is effectively [Assumption 4.1](#) and [Assumption 4.3](#) rolled into one. This condition is a bit tricky to interpret. In essence it states that, provided that we are sufficiently close to the cutoff, both a person's compliance type, as determined by $D(x)$, and her treatment effect $(Y_1 - Y_0)$ are independent of her test score X . Note that this assumption *allows* selection on gains: there is no requirement that $D(x)$ be independent of $(Y_0 - Y_1)$. Using these conditions, we obtain the following.

Theorem 5.3. *Suppose that $\mathbb{E}[Y_0|X = x]$ is a continuous function of x at $x = c$. Then, under Assumptions [5.5](#) and [5.6](#), the fuzzy RD estimand τ_F from [\(5.1\)](#) satisfies*

$$\tau_F = \lim_{h \downarrow 0} \mathbb{E}[Y_1 - Y_0 | D(c + h) - D(c - h) = 1].$$

Proof of Theorem 5.3. By [\(1.2\)](#),

$$\mathbb{E}[Y|X] = \mathbb{E}[Y_0 + D(Y_1 - Y_0)|X] = \mathbb{E}[Y_0|X] + \mathbb{E}[D(Y_1 - Y_0)|X]$$

and hence

$$\begin{aligned} \lim_{x \downarrow c} \mathbb{E}[Y|X = x] &= \lim_{x \downarrow c} \mathbb{E}[Y_0|X = x] + \lim_{x \downarrow c} \mathbb{E}[D(Y_1 - Y_0)|X = x] \\ \lim_{x \uparrow c} \mathbb{E}[Y|X = x] &= \lim_{x \uparrow c} \mathbb{E}[Y_0|X = x] + \lim_{x \uparrow c} \mathbb{E}[D(Y_1 - Y_0)|X = x]. \end{aligned}$$

Since $\mathbb{E}[Y_0|X = x]$ is continuous at c , its limit from the right at this point equals its limit from the left. Thus, subtracting the two preceding equalities

$$\begin{aligned} \lim_{x \downarrow c} \mathbb{E}[Y|X = x] - \lim_{x \uparrow c} \mathbb{E}[Y|X = x] &= \lim_{x \downarrow c} \mathbb{E}[D(Y_1 - Y_0)|X = x] - \lim_{x \uparrow c} \mathbb{E}[D(Y_1 - Y_0)|X = x] \\ &= \lim_{h \downarrow 0} \{ \mathbb{E}[D(Y_1 - Y_0)|X = c + h] - \mathbb{E}[D(Y_1 - Y_0)|X = c - h] \}. \end{aligned}$$

Now, for $0 < h < \varepsilon$ [Assumption 5.6](#) gives

$$\begin{aligned} &\mathbb{E}[D(Y_1 - Y_0)|X = c + h] - \mathbb{E}[D(Y_1 - Y_0)|X = c - h] \\ &= \mathbb{E}[D(c + h)(Y_1 - Y_0)|X = c + h] - \mathbb{E}[D(c - h)(Y_1 - Y_0)|X = c - h] \\ &= \mathbb{E}[D(c + h)(Y_1 - Y_0)] - \mathbb{E}[D(c - h)(Y_1 - Y_0)] \\ &= \mathbb{E}[\{D(c + h) - D(c - h)\}(Y_1 - Y_0)] \tag{5.2} \\ &= \mathbb{E}[Y_1 - Y_0|D(c + h) - D(c - h) = 1] \mathbb{P}[D(c + h) - D(c - h) = 1] \\ &= \mathbb{E}[Y_1 - Y_0|D(c + h) - D(c - h) = 1] \mathbb{E}[D(c + h) - D(c - h)] \end{aligned}$$

since $[D(c + h) - D(c - h)] \in \{0, 1\}$ by [Assumption 5.5](#). Similarly,

$$\begin{aligned} \mathbb{E}[D|X = c + h] - \mathbb{E}[D|X = c - h] &= \mathbb{E}[D(c + h)|X = c + h] - \mathbb{E}[D(c - h)|X = c - h] \\ &= \mathbb{E}[D(c + h) - D(c - h)]. \end{aligned}$$

The result follows by substituting the preceding into the final equality of [\(5.2\)](#), since

$$\lim_{x \downarrow c} \mathbb{E}[D|X = x] - \lim_{x \uparrow c} \mathbb{E}[D|X = x] = \lim_{h \downarrow 0} \mathbb{E}[D(c + h) - D(c - h)]. \quad \square$$

Like the assumptions under which it was proved, the result of [Theorem 5.3](#) is somewhat difficult to interpret at first glance. What does it mean to condition on the event $\{D(c + h) - D(c - h) = 1\}$ in the limit as h approaches zero from the right? Recall that the individuals for whom $D(x)$ changes from zero to one as x crosses the threshold are *precisely* the fuzzy RD compliers. It follows that [Theorem 5.3](#) identifies a causal effect for this sub-group of individuals. For the compliers, the fuzzy RD is really a *sharp* RD: these are precisely the individuals who take the treatment if and only if $X \geq c$. Accordingly, $\lim_{h \downarrow 0} \mathbb{E}[Y_1 - Y_0|D(c + h) - D(c - h) = 1]$ represents the average treatment effect for compliers at $X = c$.

Chapter 6

Difference-in-differences

Thus far we’ve focused exclusively on methods for causal inference that can be applied to *cross-section data*, a large number of individuals observed at one point in time. Panel data, repeated observations of the same individuals over time, open up a new range of possibilities for learning cause-and-effect from observational data. In this chapter we’ll examine one of them: the **difference-in-differences** (DiD) approach. When we only had a single time period to worry about, we used subscripts to represent potential outcomes: (Y_0, Y_1) . In this chapter we have the added complication of different time periods in addition to different potential outcomes, so we need to adopt a new convention. Below we use subscripts to denote *time periods* and parentheses to denote potential outcomes. Thus, $Y_t(d)$ is the potential outcome at time t when treatment status equals $d \in \{0, 1\}$. Remember it like this **P**arentheses = **P**otential Outcomes.

6.1 Adding a Time Dimension

Adding a time dimension to our problem doesn’t introduce any mathematical complications but it does present some new conceptual hurdles. Before discussing DiD and causal identification let’s begin by seeing how the problem has changed and making clear what we hope to achieve.

To keep life simple, we’ll focus on a *two period* model. The outcome Y_t is observed for two time periods: $t \in \{\text{Before}, \text{After}\}$.¹ Some individuals are *treated* between these two time periods: $D = 1$. Others are *untreated*: $D = 0$. “Before” refers to the time period before anyone has been treated, while “After” refers to the time period after some individuals have received the treatment. Each individual has a pair of potential outcome *time series*, namely $(Y_{\text{Before}}(0), Y_{\text{After}}(0))$ and $(Y_{\text{Before}}(1), Y_{\text{After}}(1))$. If Alice is treated, she experiences $Y_{\text{Before}}(1)$ followed by $Y_{\text{After}}(1)$; if she is untreated, she experiences $Y_{\text{Before}}(0)$

¹Many references write these as Y_0 and Y_1 . I use the “Before” and “After” convention to avoid confusing time periods with potential outcomes and to make it clear that treatment takes place *between* the time periods in which we observe outcomes.

followed by $Y_{\text{After}}(0)$. In other words,

$$Y_{\text{Before}} = (1 - D)Y_{\text{Before}}(0) + DY_{\text{Before}}(1) \quad (6.1)$$

$$Y_{\text{After}} = (1 - D)Y_{\text{After}}(0) + DY_{\text{After}}(1) \quad (6.2)$$

Notice that the treatment indicator D lacks a time subscript. This is because the treatment takes place *between* the periods “Before” and “After.” In our simple model no one is treated in the first period, but some people are treated before the second period. As such, $Y_{\text{Before}}(1)$ does *not* refer to the potential outcome if a person is treated in the first time period. Instead, it refers to the potential outcome in the first period if a person is *eventually treated*, i.e. treated between the two time periods.

That last sentence may strike you as needlessly complicated. Why on earth do we need to distinguish between $Y_{\text{Before}}(0)$ and $Y_{\text{Before}}(1)$? Or to put it another way: how can a person’s outcome in the first period depend on a treatment that she can only obtain after this period has ended? The answer to this question is **anticipation**: if I know that I will be treated tomorrow, this may lead me to change my behavior today in ways that affect today’s outcome. The fact that I am *eventually* treated could affect my potential outcome before I actually receive the treatment. This phenomenon is sometimes called an “Ashenfelter dip,” after [Ashenfelter \(1978\)](#) who found that “all of the trainee [treatment] groups suffered unpredicted earnings declines in the year prior to training,” in a study of the effects of a government training program.² [Figure 6.1](#) depicts this situation for a hypothetical individual, Alice. Let Y be wage. In the figure, Alice experiences an upward earnings trajectory when treated, the blue line from $Y_{\text{Before}}(1)$ to $Y_{\text{After}}(1)$, and a downward earnings trajectory when untreated, the red line from $Y_{\text{Before}}(0)$ to $Y_{\text{After}}(0)$. She also experiences an Ashenfelter dip, in that her pre-treatment earnings are *lower* if she receives treatment than if she does not: $Y_{\text{Before}}(1) < Y_{\text{Before}}(0)$.

Our goal is to learn the average effect of **treatment on the treated** (TOT) in the *second* time period, namely:

$$\text{TOT} \equiv \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{After}}(0) | D = 1]. \quad (6.3)$$

We focus on the second period because we’re not interested in learning the *anticipation effect* of the treatment but rather the *causal effect*: we want to know its effect on the future rather than its effect on the past! We focus on the *treated* sub-population, because these are the only people for whom we can carry out a before-and-after comparison: we observe them once before they have received the treatment and again afterwards. Returning to [Figure 6.1](#), the quantity $\Delta \equiv Y_{\text{After}}(1) - Y_{\text{Before}}(0)$ is Alice’s causal effect.

²Ashenfelter’s dip is great with tortilla chips. For more discussion of this phenomenon, but sadly no recipe, see [Heckman and Smith \(1999\)](#).

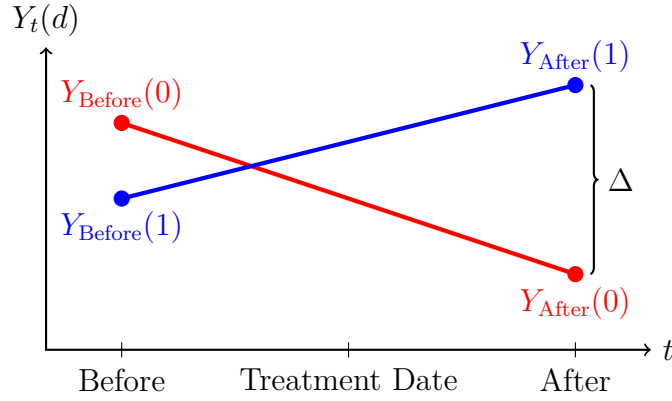


Figure 6.1: Potential time series for a particular individual in a two-period model, depicting an *Ashenfelter dip*. The treatment effect of interest is $\Delta \equiv Y_{\text{After}}(1) - Y_{\text{After}}(0)$.

The TOT is the average of Δ for individuals in the population who eventually receive the treatment.

While adding a time dimension is extremely helpful, the fundamental problem of causal inference remains: **we can never observe both $Y_{\text{After}}(1)$ and $Y_{\text{After}}(0)$ for the same person**. In earlier chapters, we addressed this problem by using *between-person* comparisons. The selection-on-observables approach, for example, constructs two groups of people who are “comparable” in terms of observed covariates X : one that was treated and one that was not. Similarly, the regression discontinuity approach compares people who are just above a threshold, hence treated, to people who are just below, hence untreated, in the hope that the arbitrariness of the cutoff itself makes the two groups comparable. The new idea in this chapter is to use the time dimension to make *within-person* comparisons. The following two sections discuss some simple assumptions under which we can use this approach to identify the TOT.

6.2 The Before-and-After Design

Let’s make two very strong assumptions. First we’ll rule out Ashenfelter dips, by assuming that there is *no anticipation* of the treatment. This means that Alice’s potential earnings in the first period are *the same* regardless of whether she eventually receives the treatment. Second we’ll assume that there’s no trend in untreated potential outcomes: the path from $Y_{\text{Before}}(0)$ to $Y_{\text{After}}(0)$ is *flat*. This means that Alice will have the *same* earnings in period two as she did in period one if she does not receive the treatment. **Figure 6.2** modifies our earlier diagram to incorporate these assumptions, producing a neat little right triangle with vertices $Y_{\text{Before}}(1)$, $Y_{\text{After}}(1)$, and $Y_{\text{After}}(0)$.

This little triangle turns out to solve all of our problems. While we can’t observe $Y_{\text{After}}(0)$ and $Y_{\text{After}}(1)$ for the same person, we can observe $Y_{\text{After}}(1)$ and $Y_{\text{Before}}(1)$ for someone who receives the treatment. These are the two endpoints of the blue line in the

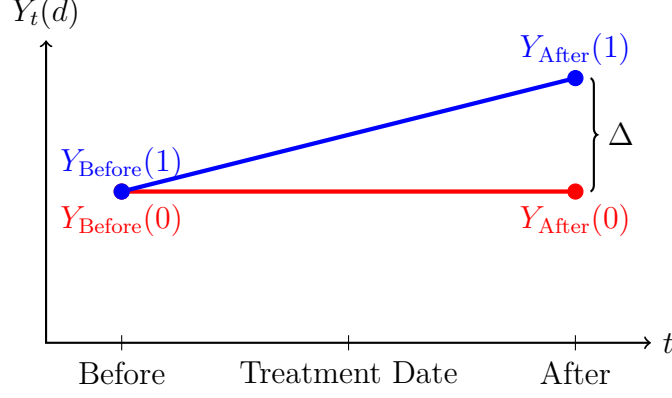


Figure 6.2: Potential time series for a particular individual in a two-period model in which the assumptions of the Before-and-after design are satisfied. The treatment effect of interest is $\Delta \equiv Y_{\text{After}}(1) - Y_{\text{After}}(0)$.

figure. But since $Y_{\text{Before}}(1)$ equals $Y_{\text{Before}}(0)$ which in turn equals $Y_{\text{After}}(0)$ we see that $\Delta = Y_{\text{After}}(1) - Y_{\text{Before}}(1)$. And since these are *precisely* the outcomes that we observe for someone with $D = 1$, it follows that $\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] = \text{TOT}$. This is the **before-and-after design**, a *within-person* comparison of observed outcomes for the treated sub-population: after minus before. And while it makes the pictures easier to read, we don't actually need $Y_{\text{After}}(0)$, $Y_{\text{Before}}(0)$, and $Y_{\text{Before}}(1)$ to be *equal*. We merely need them to be equal *on average*. Assumptions 6.1 and 6.2 make this precise; and **Theorem 6.1** shows what these assumptions buy us.

Assumption 6.1 (No Anticipation). $\mathbb{E}[Y_{\text{Before}}(1) - Y_{\text{Before}}(0) | D = 1] = 0$.

Assumption 6.2 (No Trend). $\mathbb{E}[Y_{\text{After}}(0) - Y_{\text{Before}}(0) | D = 1] = 0$.

Theorem 6.1. Under **Assumption 6.1** and **Assumption 6.2**, the before-and-after estimand identifies the TOT:

$$\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] = \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{After}}(0) | D = 1].$$

Proof. Taking the difference of average *observed* outcomes for the treated gives

$$\text{BA} \equiv \mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] = \mathbb{E}[Y_{\text{After}}(1) | D = 1] - \mathbb{E}[Y_{\text{Before}}(1) | D = 1].$$

But since

$$\mathbb{E}[Y_{\text{Before}}(1) | D = 1] = \mathbb{E}[Y_{\text{Before}}(0) | D = 1] = \mathbb{E}[Y_{\text{After}}(0) | D = 1]$$

by Assumptions 6.1 and 6.2, we obtain

$$\text{BA} = \mathbb{E}[Y_{\text{Before}}(1) | D = 1] - \mathbb{E}[Y_{\text{After}}(0) | D = 1] = \text{TOT}. \quad \square$$

The assumptions required for the before-and-after design to identify the TOT are strong, especially [Assumption 6.2](#). While there are some situations where the “no trends” assumption makes sense, e.g. over a very short time horizon and for a process that follows a random walk, there are many others where it doesn’t. Would we really expect Alice’s earnings to be the same on average in period two as in period one if she doesn’t received the treatment? A year from now the economy may be in a recession. And if Alice spent that year employed, in one year’s time she has an extra year of work experience. Here is a useful way to think about the before-and-after design: it provides sufficient conditions for a purely *within-person* comparison to identify the causal effect we hope to learn. If we want to weaken these conditions, we need something more than a within-person comparison.

6.3 The Difference-in-Differences Design

The difference-in-differences design builds on the idea of within-person comparisons introduced in the before-and-after design, but *combines this* with a between-person comparison similar to that used in earlier chapters. We continue to rule out anticipation effects, [Assumption 6.1](#). But rather than assuming that there is *no trend* in the untreated potential outcomes, [Assumption 6.2](#), we instead assume that trend that does exist is *the same* for the treated and untreated populations. In this way, the untreated population serves as a control group *for the trend*.

[Figure 6.3](#) gives the intuition. The treatment effect is $\Delta \equiv Y_{\text{After}}(1) - Y_{\text{After}}(0)$ but we can’t observe the two potential outcomes needed to compute this quantity for the same person. For the treated we observe $Y_{\text{After}}(1)$ and $Y_{\text{Before}}(1)$, allowing us to calculate $\Delta_1 \equiv Y_{\text{After}}(1) - Y_{\text{Before}}(1)$. This is precisely the *within-person* comparison that we used in the before-and-after design. But since $Y_{\text{After}}(0) \neq Y_{\text{Before}}(0)$, we see that $\Delta_1 \neq \Delta$. Here’s where the between person comparison comes in. For an *untreated* person, we observe $Y_{\text{After}}(0)$ and $Y_{\text{Before}}(0)$, allowing us to calculate $\Delta_0 \equiv Y_{\text{After}}(0) - Y_{\text{Before}}(0)$. This is the *trend* in untreated potential outcomes, the quantity that [Assumption 6.2](#) assumed was equal to zero. In [Figure 6.3](#) it clearly does not equal zero. But suppose that this trend were *the same* for treated and untreated people. Then we could use the value of Δ_0 computed from the untreated as a “stand-in” for the value of Δ_0 for the treated. And since $Y_{\text{Before}}(1) = Y_{\text{Before}}(0)$, [Assumption 6.1](#), $\Delta = \Delta_1 - \Delta_0$.

So as its name suggests, the DiD approach is based on taking *double differences*. First we compute the before-and-after average of Y for the treated and untreated individuals in our sample: $\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1]$ and $\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 0]$. The DiD estimand equals the difference of *these differences*, namely

$$\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] - \mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 0].$$

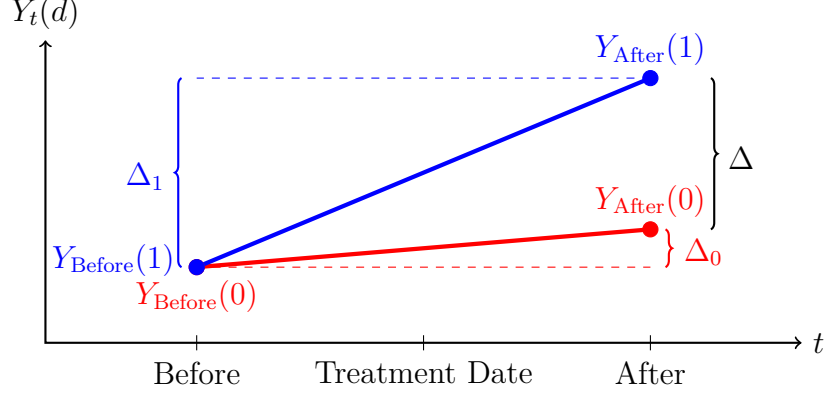


Figure 6.3: Schematic of the difference-in-differences design. The treatment effect of interest is $\Delta \equiv Y_{\text{After}}(1) - Y_{\text{After}}(0)$. We observe $\Delta_0 = Y_{\text{After}}(0) - Y_{\text{Before}}(0)$ for the untreated and $\Delta_1 = Y_{\text{After}}(1) - Y_{\text{Before}}(1)$ for the treated. Since $Y_{\text{Before}}(1) = Y_{\text{Before}}(0)$, no anticipation, $\Delta = \Delta_1 - \Delta_0$. The parallel trends assumption implies that Δ_0 is the same on average for the treated and untreated.

For this approach to identify the TOT, we need two assumptions. First is [Assumption 6.1](#), described above. Second is a new assumption called **parallel trends**.

Assumption 6.3 (Parallel Trends).

$$\mathbb{E}[Y_{\text{After}}(0) - Y_{\text{Before}}(0) | D = 1] = \mathbb{E}[Y_{\text{After}}(0) - Y_{\text{Before}}(0) | D = 0]$$

Notice that [Assumption 6.3](#) doesn't require that the trend in untreated potential outcomes is *identical* across treated and untreated individuals, merely that it's the same on average. This assumption both allows and restricts selection on unobservables. If *time-invariant* unobservables drive selection into treatment, [Assumption 6.3](#) holds. What the parallel trends assumption rules out is selection based on time-varying unobservables, aka “transitory shocks.”

Theorem 6.2. Under [Assumption 6.3](#) and [Assumption 6.1](#), the difference-in-differences estimand identifies the TOT:

$$\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] - \mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 0] = \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{After}}(0) | D = 1]$$

In [Figure 6.1](#), $\Delta \equiv Y_{\text{After}}(1) - Y_{\text{After}}(0)$ so DiD identifies the average value of Δ over the subpopulation of individuals who choose to take the treatment.

Proof of Theorem 6.2. Let θ denote the DiD estimand. By definition,

$$\theta \equiv \mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] - \mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 0] \quad (6.4)$$

and by (6.2),

$$\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] = \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{Before}}(1) | D = 1] \quad (6.5)$$

$$\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 0] = \mathbb{E}[Y_{\text{After}}(0) - Y_{\text{Before}}(0) | D = 0]. \quad (6.6)$$

Now, by [Assumption 6.1](#)

$$\mathbb{E}[Y_{\text{Before}}(1) | D = 1] = \mathbb{E}[Y_{\text{Before}}(0) | D = 1].$$

Substituting this equality into (6.5), it follows that

$$\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] = \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{Before}}(0) | D = 1]. \quad (6.7)$$

Therefore, substituting (6.6) and (6.7) into (6.4), we obtain

$$\begin{aligned} \theta &= \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{Before}}(0) | D = 1] - \mathbb{E}[Y_{\text{After}}(0) - Y_{\text{Before}}(0) | D = 0] \\ &= \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{Before}}(0) | D = 1] - \mathbb{E}[Y_{\text{After}}(0) - Y_{\text{Before}}(0) | D = 1] \\ &= \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{After}}(0) | D = 1] \end{aligned}$$

where the second equality follows from [Assumption 6.3](#). □

6.4 A Regression Interpretation of DiD

In our simple two-period model, computing the DiD estimator is easy. Simply replace population expectations with sample means and take differences:

$$\widehat{\text{DiD}} = (\bar{Y}_{\text{After, Treated}} - \bar{Y}_{\text{Before, Treated}}) - (\bar{Y}_{\text{After, Untreated}} - \bar{Y}_{\text{Before, Untreated}}).$$

An equivalent way of obtaining the same result is by running a linear regression of the observed outcome on a treatment dummy D_i a time dummy $\text{After}_t = \mathbb{1}(t = \text{After})$ and their interaction, namely

$$Y_{it} = \alpha + \beta D_i + \gamma \text{After}_t + \delta (D_i \times \text{After}_t) + U_{it}, \quad t \in \{\text{Before}, \text{After}\}.$$

The coefficient on the interaction, δ , is the DiD estimand. This approach has several advantages. First it simplifies the process of computing standard errors: we simply obtain them from our usual regression output, possibly adjusting for heteroskedasticity or clustering. Second, it allows us to introduce *control regressors*. Suppose an observed, time-varying covariate X_{it} is responsible for a violation of the parallel trends assumption:

X_{it} affects Y_{it} and is correlated with treatment. By including X_{it} in our regression, e.g.

$$Y_{it} = \alpha + \beta D_i + \gamma \text{After}_t + \delta (D_i \times \text{After}_t) + X'_{it}\theta + U_{it}.$$

we may be able to salvage the DiD approach in a situation where the parallel trends assumption does *not* hold unconditionally.

6.5 What if the treatment is anticipated?

The *crucial* assumption for DiD is parallel trends: [Assumption 6.3](#). But, as in the before-and-after design, we also assumed that there is no anticipation of the treatment, [Assumption 6.1](#). What happens if we relax this assumption? To find out, we'll first define an additional causal effect: the **anticipation effect for the treated** (AET)

$$\text{AET} \equiv \mathbb{E}[Y_{\text{Before}}(1) - Y_{\text{Before}}(0) | D = 1].$$

The AET quantifies the effect of *future* treatment on *current* outcomes. For example, if a criminal learns that harsher sentencing regimes will be put in place next year and decides to carry out a robbery *now* in response to this policy change, the AET would pick this up. As shown in the following two results, relaxing the no anticipation assumption changes the causal effect identified by the DiD and BA designs. Rather than identifying the TOT, they identify the *difference* TOT – AET. Depending on the application, this may or may not be an interesting quantity but it is at least a meaningful one: the “future effect” of treatment net of any anticipated effect in the present period.

Theorem 6.3. Under [Assumption 6.2](#) $\mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] = \text{TOT} - \text{AET}$.

Proof. By the definition of the before-and-after estimand and (6.2),

$$\text{BA} \equiv \mathbb{E}[Y_{\text{After}} - Y_{\text{Before}} | D = 1] = \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{Before}}(1) | D = 1].$$

But by [Assumption 6.2](#), $\mathbb{E}[Y_{\text{After}}(0) - Y_{\text{Before}}(0) | D = 1] = 0$. Therefore,

$$\begin{aligned} \text{BA} &= \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{Before}}(1) | D = 1] - 0 \\ &= \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{Before}}(1) | D = 1] - \mathbb{E}[Y_{\text{After}}(0) - Y_{\text{Before}}(0) | D = 1] \\ &= \mathbb{E}[Y_{\text{After}}(1) - Y_{\text{After}}(0) | D = 1] - \mathbb{E}[Y_{\text{Before}}(1) - Y_{\text{Before}}(0) | D = 1] \\ &= \text{TOT} - \text{AET}. \end{aligned} \quad \square$$

Theorem 6.4. Under [Assumption 6.3](#), the difference-in-differences estimand identifies

the difference of the TOT and AET:

$$\mathbb{E}[Y_{After} - Y_{Before}|D = 1] - \mathbb{E}[Y_{After} - Y_{Before}|D = 0] = TOT - AET.$$

Proof. By the definition of the DiD estimand and (6.2),

$$\begin{aligned} \text{DiD} &\equiv \mathbb{E}[Y_{After} - Y_{Before}|D = 1] - \mathbb{E}[Y_{After} - Y_{Before}|D = 0] \\ &= \mathbb{E}[Y_{After}(1) - Y_{Before}(1)|D = 1] - \mathbb{E}[Y_{After}(0) - Y_{Before}(0)|D = 0]. \end{aligned}$$

But by Assumption 6.3,

$$\mathbb{E}[Y_{After}(0) - Y_{Before}(0)|D = 0] = \mathbb{E}[Y_{After}(0) - Y_{Before}(0)|D = 1].$$

Substituting this in the expression for DiD, we obtain

$$\begin{aligned} \text{DiD} &= \mathbb{E}[Y_{After}(1) - Y_{Before}(1)|D = 1] - \mathbb{E}[Y_{After}(0) - Y_{Before}(0)|D = 1] \\ &= \mathbb{E}[Y_{After}(1) - Y_{After}(0)|D = 1] - \mathbb{E}[Y_{Before}(1) - Y_{Before}(0)|D = 1] \\ &= \text{TOT} - \text{AE}. \end{aligned}$$

□

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