Course Lecture Notes

Introduction to Causal Inference

from a Machine Learning Perspective

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Preface

Prerequisites There is one main prerequisite: **basic probability**. This course assumes you've taken an introduction to probability course or have had equivalent experience. Topics from statistics and machine learning will pop up in the course from time to time, so some familiarity with those will be helpful but is not necessary. For example, if cross-validation is a new concept to you, you can learn it relatively quickly at the point in the book that it pops up. And we give a primer on some statistics terminology that we'll use in Section 2.4.

Active Reading Exercises Research shows that one of the best techniques to remember material is to actively try to recall information that you recently learned. You will see "active reading exercises" throughout the book to help you do this. They'll be marked by the Active reading exercise: heading.

Many Figures in This Book As you will see, there are a ridiculous amount of figures in this book. This is on purpose. This is to help give you as much visual intuition as possible. We will sometimes copy the same figures, equations, etc. that you might have seen in preceding chapters so that we can make sure the figures are always right next to the text that references them.

Sending Me Feedback This is a book *draft*, so I greatly appreciate any feedback you're willing to send my way. If you're unsure whether I'll be receptive to it or not, don't be. Please send any feedback to me at bradyneal11@gmail.com with "[Causal Book]" in the beginning of your email subject. Feedback can be at the word level, sentence level, section level, chapter level, etc. Here's a non-exhaustive list of useful kinds of feedback:

- Typoz.
- Some part is confusing.
- You notice your mind starts to wander, or you don't feel motivated to read some part.
- Some part seems like it can be cut.
- You feel strongly that some part absolutely should not be cut.
- Some parts are not connected well. Moving from one part to the next, you notice that there isn't a natural flow.
- A new active reading exercise you thought of.

Bibliographic Notes Although we do our best to cite relevant results, we don't want to disrupt the flow of the material by digging into exactly where each concept came from. There will be complete sections of bibliographic notes in the final version of this book, but they won't come until after the course has finished.

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Motivation: Why You Might Care

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1.1 Simpson's Paradox

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Consider a purely hypothetical future where there is a new disease known as COVID-27 that is prevalent in the human population. In this purely hypothetical future, there are two treatments that have been developed: treatment A and treatment B. Treatment B is more scarce than treatment A, so the split of those currently receiving treatment A vs. treatment B is roughly 73%/27%. You are in charge of choosing which treatment your country will exclusively use, in a country that only cares about minimizing loss of life.

You have data on the percentage of people who die from COVID-27, given the treatment they were assigned and given their condition at the time treatment was decided. Their condition is a binary variable: either mild or severe. In this data, 16% of those who receive A die, whereas 19% of those who receive B die. However, when we examine the people with mild condition separately from the people with severe condition, the numbers reverse order. In the mild subpopulation, 15% of those who receive A die, whereas 10% of those who receive B die. In the severe subpopulation, 30% of those who receive A die, whereas 20% of those who receive B die. We depict these percentages and the corresponding counts in Table 1.1.

		Condition: Mild	Condition: Severe	Condition: Total
Treatment	A	15% (210/1400)	30% (30/100)	**16%** (240/1500)
	B	**10%** (5/50)	**20%** (100/500)	19% (105/550)

Table 1.1: Simpson's paradox in COVID-27 data. The percentages denote the mortality rates in each of the groups. Lower is better. The numbers in parentheses are the corresponding counts. This apparent paradox stems from the interpretation that treatment A looks better when examining the whole population, but treatment B looks better in all subpopulations.

The apparent paradox stems from the fact that, in Table 1.1, the "Total" column could be interpreted to mean that we should prefer treatment A, whereas the "Mild" and "Severe" columns could both be interpreted to mean that we should prefer treatment B.[1] In fact, the answer is that if we know someone's condition, we should give them treatment B, and if we do *not* know their condition, we should give them treatment A. Just kidding... that doesn't make any sense. So really, what treatment should you choose for your country?

Either treatment A or treatment B could be the right answer, depending on the causal structure of the data. In other words, causality is essential to solve Simpson's paradox. For now, we will just give the intuition for when you should prefer treatment A vs. when you should prefer treatment B, but it will be made more formal in Chapter 4.

[1] A key ingredient necessary to find Simpson's paradox is the **non-uniformity of allocation of people to the groups**. 1400 of the 1500 people who received treatment A had mild condition, whereas 500 of the 550 people who received treatment B had severe condition. Because people with mild condition are less likely to die, this means that the total mortality rate for those with treatment A is lower than what it would have been if mild and severe conditions were equally split among them. The opposite bias is true for treatment B.

Scenario 1 If the condition C is a cause of the treatment T (Figure 1.1), treatment B is more effective at reducing mortality Y. An example scenario is where doctors decide to give treatment A to most people who have mild conditions. And they save the more expensive and more limited treatment B for people with severe conditions. Because having severe condition causes one to be more likely to die ($C \rightarrow Y$ in Figure 1.1) and causes one to be more likely to receive treatment B ($C \rightarrow T$ in Figure 1.1), treatment B will be associated with higher mortality in the total population. In other words, treatment B is associated with a higher mortality rate simply because condition is a common cause of both treatment and mortality. Here, condition confounds the effect of treatment on mortality. To correct for this confounding, we must examine the relationship of T and Y among patients with the same conditions. This means that the better treatment is the one that yields lower mortality in each of the subpopulations (the "Mild" and "Severe" columns in Table 1.1): treatment B.

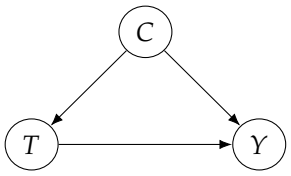

Figure 1.1: Causal structure of scenario 1, where condition C is a common cause of treatment T and mortality Y. Given this causal structure, treatment B is preferable.

Scenario 2 If the prescription[2] of treatment T is a cause of the condition C (Figure 1.2), treatment A is more effective. An example scenario is where treatment B is so scarce that it requires patients to wait a long time after they were prescribed the treatment before they can receive the treatment. Treatment A does not have this problem. Because the condition of a patient with COVID-27 worsens over time, the prescription of treatment B actually causes patients with mild conditions to develop severe conditions, causing a higher mortality rate. Therefore, even if treatment B is more effective than treatment A once *administered* (positive effect along $T \rightarrow Y$ in Figure 1.2), because *prescription* of treatment B causes worse conditions (negative effect along $T \rightarrow C \rightarrow Y$ in Figure 1.2), treatment B is less effective in total. Note: Because treatment B is more expensive, treatment B is prescribed with 0.27 probability, while treatment A is prescribed with 0.73 probability; importantly, treatment prescription is independent of condition in this scenario.

[2] T refers to the prescription of the treatment, rather than the subsequent reception of the treatment.

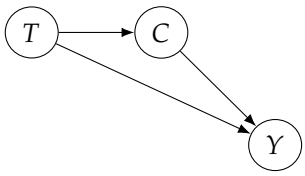

Figure 1.2: Causal structure of scenario 2, where treatment T is a cause of condition C. Given this causal structure, treatment A is preferable.

In sum, the more effective treatment is completely dependent on the causal structure of the problem. In Scenario 1, where C was a cause of T (Figure 1.1), treatment B was more effective. In Scenario 2, where T was a cause of C (Figure 1.2), treatment A was more effective. Without causality, Simpson's paradox cannot be resolved. With causality, it is not a paradox at all.

1.2 Applications of Causal Inference

Causal inference is essential to science, as we often want to make causal claims, rather than merely associational claims. For example, if we are choosing between treatments for a disease, we want to choose the treatment that causes the most people to be cured, without causing too many bad side effects. If we want a reinforcement learning algorithm to maximize reward, we want it to take actions that cause it to achieve the maximum reward. If we are studying the effect of social media on mental health, we are trying to understand what the main causes of a given mental health outcome are and order these causes by the percentage of the outcome that can be attributed to each cause.

Causal inference is essential for rigorous decision-making. For example, say we are considering several different policies to implement to reduce greenhouse gas emissions, and we must choose just one due to budget constraints. If we want to be maximally effective, we should carry out causal analysis to determine which policy will cause the largest reduction in emissions. As another example, say we are considering several interventions to reduce global poverty. We want to know which policies will cause the largest reductions in poverty.

Now that we've gone through the general example of Simpson's paradox and a few specific examples in science and decision-making, we'll move to how causal inference is so different from prediction.

1.3 Correlation Does Not Imply Causation

Many of you will have heard the mantra "correlation does not imply causation." In this section, we will quickly review that and provide you with a bit more intuition about why this is the case.

1.3.1 Nicolas Cage and Pool Drownings

It turns out that the yearly number of people who drown by falling into swimming pools has a high degree of correlation with the yearly number of films that Nicolas Cage appears in [1]. See Figure 1.3 for a graph of this data. Does this mean that Nicolas Cage encourages bad swimmers to hop in the pool in his films? Or does Nicolas Cage feel more motivated to act in more films when he sees how many drownings are happening that year, perhaps to try to prevent more drownings? Or is there some other explanation? For example, maybe Nicolas Cage is interested in increasing his popularity among causal inference practitioners, so he travels back in time to convince his past self to do just the right number of movies for us to see this correlation, but not too close of a match as that would arouse suspicion and potentially cause someone to prevent him from rigging the data this way. We may never know for sure.

[1]: Vigen (2015), *Spurious correlations*

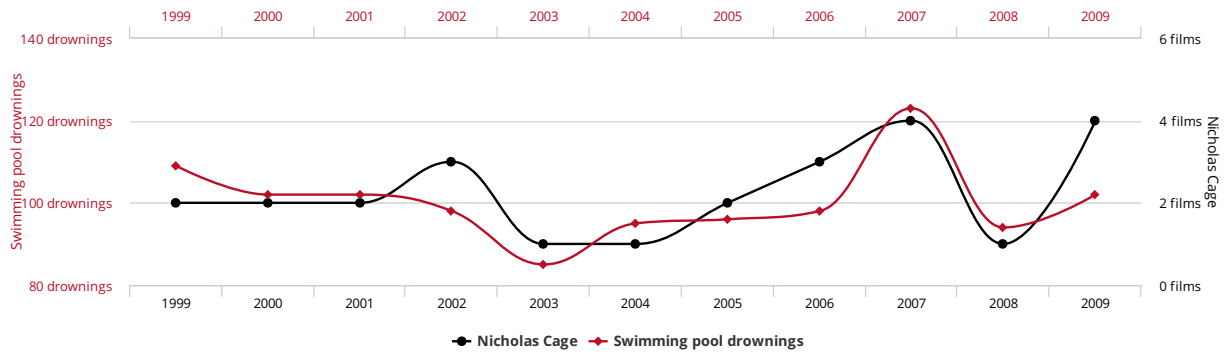

Figure 1.3: The yearly number of movies Nicolas Cage appears in correlates with the yearly number of pool drownings [1].

Of course, all of the possible explanations in the preceding paragraph seem quite unlikely. Rather, it is likely that this is a *spurious correlation*, where there is no causal relationship. We'll soon move on to a more

illustrative example that will help clarify how spurious correlations can arise.

1.3.2 Why is Association Not Causation?

Before moving to the next example, let's be a bit more precise about terminology. "Correlation" is often colloquially used as a synonym for statistical dependence. However, "correlation" is technically only a measure of *linear* statistical dependence. We will largely be using the term *association* to refer to statistical dependence from now on.

Causation is not all or none. For any given amount of association, it does not need to be "all of the association is causal" or "none of the association is causal." Rather, it is possible to have a large amount of association with only *some* of it being causal. The phrase "association is not causation" simply means that the amount of association and the amount of causation can be different. Some amount of association and zero causation is a special case of "association is not causation."

Say you happen upon some data that relates wearing shoes to bed and waking up with a headache, as one does. It turns out that most times that someone wears shoes to bed, that person wakes up with a headache. And most times someone doesn't wear shoes to bed, that person doesn't wake up with a headache. It is not uncommon for people to interpret data like this (with associations) as meaning that wearing shoes to bed causes people to wake up with headaches, especially if they are looking for a reason to justify not wearing shoes to bed. A careful journalist might make claims like "wearing shoes to bed is associated with headaches" or "people who wear shoes to bed are at higher risk of waking up with headaches." However, the main reason to make claims like that is that most people will internalize claims like that as "if I wear shoes to bed, I'll probably wake up with a headache."

We can explain how wearing shoes to bed and headaches are associated without either being a cause of the other. It turns out that they are both caused by a *common cause*: drinking the night before. We depict this in Figure 1.4. You might also hear this kind of variable referred to as a "confounder" or a "lurking variable." We will call this kind of association *confounding association* since the association is facilitated by a confounder.

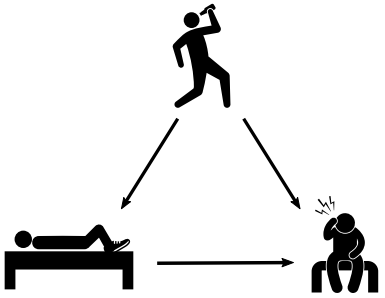

Figure 1.4: Causal structure, where drinking the night before is a common cause of sleeping with shoes on and of waking up with a headaches.

The total association observed can be made up of both confounding association and causal association. It could be the case that wearing shoes to bed does have some small causal effect on waking up with a headache. Then, the total association would not be solely confounding association nor solely causal association. It would be a mixture of both. For example, in Figure 1.4, causal association flows along the arrow from shoe-sleeping to waking up with a headache. And confounding association flows along the path from shoe-sleeping to drinking to headachening (waking up with a headache). We will make the graphical interpretation of these different kinds of association clear in Chapter 3.

The Main Problem The main problem motivating causal inference is that association is not causation.[3] If the two were the same, then causal inference would be easy. Traditional statistics and machine learning would already have causal inference solved, as measuring causation would be as simple as just looking at measures such as correlation and predictive performance in data. A large portion of this book will be about better understanding and solving this problem.

[3] As we'll see in Chapter 5, if we randomly assign the treatment in a controlled experiment, association actually *is* causation.

1.4 Main Themes

There are several overarching themes that will keep coming up throughout this book. These themes will largely be comparisons of two different categories. As you are reading, it is important that you understand which categories different sections of the book fit into and which categories they do not fit into.

Statistical vs. Causal Even with an infinite amount of data, we sometimes cannot compute some causal quantities. In contrast, much of statistics is about addressing uncertainty in finite samples. When given infinite data, there is no uncertainty. However, association, a statistical concept, is not causation. There is more work to be done in causal inference, even after starting with infinite data. This is the main distinction motivating causal inference. We have already made this distinction in this chapter and will continue to make this distinction throughout the book.

Identification vs. Estimation Identification of causal effects is unique to causal inference. It is the problem that remains to solve, even when we have infinite data. However, causal inference also shares estimation with traditional statistics and machine learning. We will largely begin with identification of causal effects (in Chapters 2, 4 and 6) before moving to estimation of causal effects (in Chapter 7). The exceptions are Section 2.5 and Section 4.6.2, where we carry out complete examples with estimation to give you an idea of what the whole process looks like early on.

Interventional vs. Observational If we can intervene/experiment, identification of causal effects is relatively easy. This is simply because we can actually take the action that we want to measure the causal effect of and simply measure the effect after we take that action. Observational data is where it gets more complicated because confounding is almost always introduced into the data.

Assumptions There will be a large focus on what assumptions we are using to get the results that we get. Each assumption will have its own box to help make it difficult to not notice. Clear assumptions should make it easy to see where critiques of a given causal analysis or causal model will be. The hope is that presenting assumptions clearly will lead to more lucid discussions about causality.

2 Potential Outcomes

In this chapter, we will ease into the world of causality. We will see that new concepts and corresponding notations need to be introduced to clearly describe causal concepts. These concepts are "new" in the sense that they may not exist in traditional statistics or math, but they should be familiar in that we use them in our thinking and describe them with natural language all the time.

Familiar statistical notation We will use T to denote the random variable for treatment, Y to denote the random variable for the outcome of interest and X to denote covariates. In general, we will use uppercase letters to denote random variables (except in maybe one case) and lowercase letters to denote values that random variables take on. Much of what we consider will be settings where T is binary. Know that, in general, we can extend things to work in settings where T can take on more than two values or where T is continuous.

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2.1 Potential Outcomes and Individual Treatment Effects

We will now introduce the first causal concept to appear in this book. These concepts are sometimes characterized as being unique to the Neyman-Rubin [2–4] causal model (or potential outcomes framework), but they are not. For example, these same concepts are still present (just under different notation) in the framework that uses causal graphs (Chapters 3 and 4). It is important that you spend some time ensuring that you understand these initial causal concepts. If you have not studied causal inference before, they will be unfamiliar to see in mathematical contexts, though they may be quite familiar intuitively because we commonly think and communicate in causal language.

Scenario 1 Consider the scenario where you are unhappy. And you are considering whether or not to get a dog to help make you happy. If you become happy after you get the dog, does this mean the dog caused you to be happy? Well, what if you would have also become happy had you *not* gotten the dog? In that case, the dog was not necessary to make you happy, so its claim to a causal effect on your happiness is weak.

Scenario 2 Let's switch things up a bit. Consider that you will still be happy if you get a dog, but now, if you don't get a dog, you will remain unhappy. In this scenario, the dog has a pretty strong claim to a causal effect on your happiness.

In both the above scenarios, we have used the causal concept known as potential outcomes. Your outcome Y is happiness: $Y = 1$ corresponds to happy while $Y = 0$ corresponds to unhappy. Your treatment T is whether or not you get a dog: $T = 1$ corresponds to you getting a dog while $T = 0$

[2]: Splawa-Neyman (1923 [1990]), 'On the Application of Probability Theory to Agricultural Experiments. Essay on Principles. Section 9.'
[3]: Rubin (1974), 'Estimating causal effects of treatments in randomized and nonrandomized studies.'
[4]: Sekhon (2008), 'The Neyman-Rubin Model of Causal Inference and Estimation via Matching Methods'

corresponds to you not getting a dog. We denote by $Y(1)$ the *potential* outcome of happiness you would observe if you were to get a dog ($T = 1$). Similarly, we denote by $Y(0)$ the potential outcome of happiness you would observe if you were to not get a dog ($T = 0$). In scenario 1, $Y(1) = 1$ and $Y(0) = 1$. In contrast, in scenario 2, $Y(1) = 1$ and $Y(0) = 0$.

More generally, the *potential outcome* $Y(t)$ denotes what your outcome would be, if you were to take treatment t. A potential outcome $Y(t)$ is distinct from the observed outcome Y in that not all potential outcomes are observed. Rather all potential outcomes can *potentially* be observed. The one that is actually observed depends on the value that the treatment T takes on.

In the previous scenarios, there was only a single individual in the whole population: you. However, generally, there are many individuals [1] in the population of interest. We will denote the treatment, covariates, and outcome of the i_{th} individual using T_i, X_i, and Y_i. Then, we can define the *individual treatment effect* (ITE) [2] for individual i:

$$\tau_i \triangleq Y_i(1) - Y_i(0) \tag{2.1}$$

Whenever there is more than one individual in a population, $Y(t)$ is a random variable because different individuals will have different potential outcomes. In contrast, $Y_i(t)$ is usually treated as non-random[3] because the subscript i means that we are conditioning on so much individualized (and context-specific) information, that we restrict our focus to a single individual (in a specific context) whose potential outcomes are deterministic.

ITEs are some of the main quantities that we care about in causal inference. For example, in scenario 2 above, you would choose to get a dog because the causal effect of getting a dog on your happiness is positive: $Y(1) - Y(0) = 1 - 0 = 1$. In contrast, in scenario 1, you might choose to not get a dog because there is no causal effect of getting a dog on your happiness: $Y(1) - Y(0) = 1 - 1 = 0$.

Now that we've introduced potential outcomes and ITEs, we can introduce the main problems that pop up in causal inference that are not present in fields where the main focus is on association or prediction.

[1] "Unit" is often used in the place of "individual" as the units of the population are not always people.

[2] The ITE is also known as the *individual causal effect*, *unit-level causal effect*, or *unit-level treatment effect*.

[3] Though, $Y_i(t)$ can be treated as random.

2.2 The Fundamental Problem of Causal Inference

It is impossible to observe all potential outcomes for a given individual [3] . Consider the dog example. You could observe $Y(1)$ by getting a dog and observing your happiness after getting a dog. Alternatively, you could observe $Y(0)$ by not getting a dog and observing your happiness. However, you cannot observe both $Y(1)$ and $Y(0)$, unless you have a time machine that would allow you to go back in time and choose the version of treatment that you didn't take the first time. You cannot simply get a dog, observe $Y(1)$, give the dog away, and then observe $Y(0)$ because the second observation will be influenced by all the actions you took between the two observations and anything else that changed since the first observation.

[3]: Rubin (1974), 'Estimating causal effects of treatments in randomized and nonrandomized studies.'

This is known as the *fundamental problem of causal inference* [5]. It is fundamental because if we cannot observe both $Y_i(1)$ and $Y_i(0)$, then we cannot observe the causal effect $Y_i(1) - Y_i(0)$. This problem is unique to causal inference because, in causal inference, we care about making causal claims, which are defined in terms of potential outcomes. For contrast, consider machine learning. In machine learning, we often only care about predicting the observed outcome Y, so there is no need for potential outcomes, which means machine learning does not have to deal with this fundamental problem that we must deal with in causal inference.

[5]: Holland (1986), 'Statistics and Causal Inference'

The potential outcomes that you do not (and cannot) observe are known as *counterfactuals* because they are counter to fact (reality). "Potential outcomes" are sometimes referred to as "counterfactual outcomes," but we will never do that in this book because a potential outcome $Y(t)$ does not become counter to fact until another potential outcome $Y(t')$ is observed. The potential outcome that is observed is sometimes referred to as a *factual*. Note that there are no counterfactuals or factuals until the outcome is observed. Before that, there are only *potential* outcomes.

2.3 Getting Around the Fundamental Problem

I suspect this section is where this chapter might start to get a bit unclear. If that is the case for you, don't worry too much, and just continue to the next chapter, as it will build up parallel concepts in a hopefully more intuitive way.

2.3.1 Average Treatment Effects and Missing Data Interpretation

We know that we can't access individual treatment effects, but what about *average* treatment effects? We get the *average treatment effect* (ATE)[4] by taking an average over the ITEs:

$$\tau \triangleq \mathbb{E}[Y_i(1) - Y_i(0)] = \mathbb{E}[Y(1) - Y(0)]\,, \tag{2.2}$$

[4] The ATE is also known as the "average *causal* effect (ACE)."

where the average is over the individuals i if $Y_i(t)$ is deterministic. If $Y_i(t)$ is random, the average is also over any other randomness.

Okay, but how would we actually compute the ATE? Let's look at some made-up data in Table 2.1 for this. If you like examples, feel free to substitute in the COVID-27 example from Section 1.1 or the dog-happiness example from Section 2.1. We will take this table as the whole population of interest. Because of the fundamental problem of causal inference, this is fundamentally a missing data problem. All of the question marks in the table indicate that we do not observe that cell.

A natural quantity that comes to mind is the *associational difference*: $\mathbb{E}[Y|T = 1] - \mathbb{E}[Y|T = 0]$. By linearity of expectation, we have that the ATE $\mathbb{E}[Y(1) - Y(0)] = \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)]$. Then, maybe $\mathbb{E}[Y(1)] - \mathbb{E}[Y(0)]$ equals $\mathbb{E}[Y|T = 1] - \mathbb{E}[Y|T = 0]$. Unfortunately, this is not true in general. If it were, that would mean that causation is simply association. $\mathbb{E}[Y|T = 1] - \mathbb{E}[Y|T = 0]$ is an associational quantity, whereas $\mathbb{E}[Y(1)] - \mathbb{E}[Y(0)]$

i	T	Y	$Y(1)$	$Y(0)$	$Y(1) - Y(0)$
1	0	0	?	0	?
2	1	1	1	?	?
3	1	0	0	?	?
4	0	0	?	0	?
5	0	1	?	1	?
6	1	1	1	?	?

Table 2.1: Example data to illustrate that the fundamental problem of causal inference can be interpreted as a missing data problem.

is a causal quantity. They are not equal due to confounding, which we discussed in Section 1.3. The graphical interpretation of this, depicted in Figure 2.1, is that X confounds the effect of T on Y because there is this $T \leftarrow X \rightarrow Y$ path that non-causal association flows along.[5]

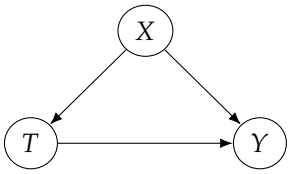

Figure 2.1: Causal structure of X confounding the effect of T on Y.

[5] Keep reading to Chapter 3, where we will flesh out and formalize this graphical interpretation.

2.3.2 Ignorability and Exchangeability

Well, what assumption(s) would make it so that the ATE is simply the associational difference? This is equivalent to saying "what makes it valid to calculate the ATE by taking the average of the $Y(0)$ column, ignoring the question marks, and subtracting that from the average of the $Y(1)$ column, ignoring the question marks?"[6] This ignoring of the question marks (missing data) is known as *ignorability*. Assuming ignorability is like ignoring how people ended up selecting the treatment they selected and just assuming they were randomly assigned their treatment; we depict this graphically in Figure 2.2 by the lack of a causal arrow from X to T. We will now state this assumption formally.

[6] Active reading exercise: verify that this procedure is equivalent to $\mathbb{E}[Y|T = 1] - \mathbb{E}[Y|T = 0]$ in the data in Table 2.1.

Assumption 2.1 (Ignorability / Exchangeability)

$$(Y(1), Y(0)) \perp\!\!\!\perp T$$

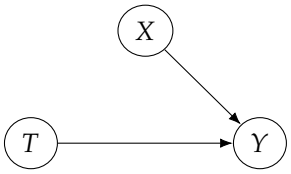

Figure 2.2: Causal structure when the treatment assignment mechanism is ignorable. Notably, this means there's no arrow from X to T, which means there is no confounding.

This assumption is key to causal inference because it allows us to reduce the ATE to the associational difference:

$$\begin{aligned} \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] &= \mathbb{E}[Y(1) \mid T = 1] - \mathbb{E}[Y(0) \mid T = 0] && (2.3) \\ &= \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0] && (2.4) \end{aligned}$$

The ignorability assumption is used in Equation 2.3. We will talk more about Equation 2.4 when we get to Section 2.3.5.

Another perspective on this assumption is that of *exchangeability*. Exchangeability means that the treatment groups are exchangeable in the sense that if they were swapped, the new treatment group would observe the same outcomes as the old treatment group, and the new control group would observe the same outcomes as the old control group. Formally, this assumption means $\mathbb{E}[Y(1)|T = 0] = \mathbb{E}[Y(1)|T = 1]$ and $\mathbb{E}[Y(0)|T = 1] = \mathbb{E}[Y(0)|T = 0]$, respectively. Then, this implies $\mathbb{E}[Y(1)|T = t] = \mathbb{E}[Y(1)]$ and $\mathbb{E}[Y(0)|T = t] = \mathbb{E}[Y(0)]$, for all t, which is nearly equivalent[7] to Assumption 2.1.

An important intuition to have about exchangeability is that it guarantees that the treatment groups are comparable. In other words, the treatment groups are the same in all relevant aspects other than the treatment. This intuition is what underlies the concept of "controlling for" or "adjusting

[7] Technically, this is *mean* exchangeability, which is a weaker assumption than the full exchangeability that we describe in Assumption 2.1 because it only constrains the first moment of the distribution. Generally, we only need mean ignorability/exchangeability for average treatment effects, but it is common to assume complete independence, as in Assumption 2.1.

for" variables, which we will discuss shortly when we get to conditional exchangeability.

We have leveraged Assumption 2.1 to identify causal effects. To *identify* a causal effect is to reduce a causal expression to a purely statistical expression. In this chapter, that means to reduce an expression from one that uses potential outcome notation to one that uses only statistical notation such as T, X, Y, expectations, and conditioning. This means that we can calculate the causal effect from just the observational distribution $P(X, T, Y)$.

Definition 2.1 (Identifiability) *A causal quantity (e.g. $\mathbb{E}[Y(t)]$) is identifiable if we can compute it from a purely statistical quantity (e.g. $\mathbb{E}[Y \mid t]$).*

We have seen that ignorability is extremely important (Equation 2.3), but how realistic of an assumption is it? In general, it is completely unrealistic because there is likely to be confounding in most data we observe (causal structure shown in Figure 2.1). However, we can make this assumption realistic by running randomized experiments, which force the treatment to not be caused by anything but a coin toss, so then we have the causal structure shown in Figure 2.2. We cover randomized experiments in greater depth in Chapter 5.

We have covered two prominent perspectives on this main assumption (2.1): ignorability and exchangeability. Mathematically, these mean the same thing, but their names correspond to different ways of thinking about the same assumption. Exchangeability and ignorability are only two names for this assumption. We will see more aliases after we cover the more realistic, conditional version of this assumption.

2.3.3 Conditional Exchangeability and Unconfoundedness

In observational data, it is unrealistic to assume that the treatment groups are exchangeable. In other words, there is no reason to expect that the groups are the same in all relevant variables other than the treatment. However, if we control for relevant variables by conditioning, then maybe the subgroups will be exchangeable. We will clarify what the "relevant variables" are in Chapter 3, but for now, let's just say they are all of the covariates X. Then, we can state conditional exchangeability formally.

Assumption 2.2 (Conditional Exchangeability / Unconfoundedness)

$$(Y(1), Y(0)) \perp\!\!\!\perp T \mid X$$

The idea is that although the treatment and potential outcomes may be unconditionally associated (due to confounding), within levels of X, they are not associated. In other words, there is no confounding within levels of X because controlling for X has made the treatment groups comparable. We'll now give a bit of graphical intuition for the above. We will not draw the rigorous connection between the graphical intuition and Assumption 2.2 until Chapter 3; for now, it is just meant to aid intuition.

We do not have exchangeability in the data because X is a common cause of T and Y. We illustrate this in Figure 2.3. Because X is a common cause of T and Y, there is non-causal association between T and Y. This non-causal association flows along the $T \leftarrow X \rightarrow Y$ path; we depict this with a red dashed arc.

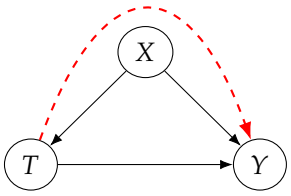

Figure 2.3: Causal structure of X confounding the effect of T on Y. We depict the confounding with a red dashed line.

However, we *do* have *conditional* exchangeability in the data. This is because, when we condition on X, there is no longer any non-causal association between T and Y. The non-causal association is now "blocked" at X by conditioning on X. We illustrate this blocking in Figure 2.4 by shading X to indicate it is conditioned on and by showing the red dashed arc being blocked there.

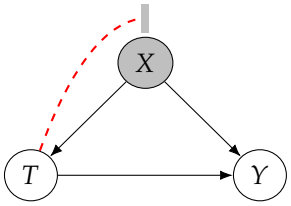

Figure 2.4: Illustration of conditioning on X leading to no confounding.

Conditional exchangeability is the main assumption necessary for causal inference. Armed with this assumption, we can identify the causal effect within levels of X, just like we did with (unconditional) exchangeability:

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0) \mid X] &= \mathbb{E}[Y(1) \mid X] - \mathbb{E}[Y(0) \mid X] && (2.5)\\
&= \mathbb{E}[Y(1) \mid T = 1,\, X] - \mathbb{E}[Y(0) \mid T = 0,\, X] && (2.6)\\
&= \mathbb{E}[Y \mid T = 1,\, X] - \mathbb{E}[Y \mid T = 0,\, X] && (2.7)
\end{aligned}$$

In parallel to before, we get Equation 2.5 by linearity of expectation. And we now get Equation 2.6 by conditional exchangeability. If we want the marginal effect that we had before when assuming (unconditional) exchangeability, we can get that by simply marginalizing out X:

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0)] &= \mathbb{E}_X \mathbb{E}[Y(1) - Y(0) \mid X] && (2.8)\\
&= \mathbb{E}_X \left[\mathbb{E}[Y \mid T = 1,\, X] - \mathbb{E}[Y \mid T = 0,\, X]\right] && (2.9)
\end{aligned}$$

This marks an important result for causal inference, so we'll give it its own proposition box. The proof we give above leaves out some details. Read through to Section 2.3.6 (where we redo the proof with all details specified) to get the rest of the details. We will call this result the *adjustment formula*.

Theorem 2.1 (Adjustment Formula) *Given the assumptions of unconfoundedness, positivity, consistency, and no interference, we can identify the average treatment effect:*

$$\mathbb{E}[Y(1) - Y(0)] = \mathbb{E}_X \left[\mathbb{E}[Y \mid T = 1,\, X] - \mathbb{E}[Y \mid T = 0,\, X]\right]$$

Conditional exchangeability (Assumption 2.2) is a core assumption for causal inference and goes by many names. For example, the following are reasonably commonly used to refer to the same assumption: unconfoundedness, conditional ignorability, no unobserved confounding, selection on observables, no omitted variable bias, etc. We will use the name "unconfoundedness" a fair amount throughout this book.

The main reason for moving from exchangeability (Assumption 2.1) to conditional exchangeability (Assumption 2.2) was that it seemed like a more realistic assumption. However, we often cannot know for certain if conditional exchangeability holds. There may be some unobserved confounders that are not part of X, meaning conditional exchangeability is violated. Fortunately, that is not a problem in randomized experiments

(Chapter 5). Unfortunately, it is something that we must always be conscious of in observational data. Intuitively, the best thing we can do is to observe and fit as many covariates into X as possible to try to ensure unconfoundedness.[8]

[8] As we will see in Chapters 3 and 4, it is not necessarily true that conditioning on more covariates always helps our causal estimates be less biased.

2.3.4 Positivity/Overlap and Extrapolation

While conditioning on many covariates is attractive for achieving unconfoundedness, it can actually be detrimental for another reason that has to do with another important assumption that we have yet to discuss: *positivity*. We will get to why at the end of this section. Positivity is the condition that all subgroups of the data with different covariates have some probability of receiving any value of treatment. Formally, we define positivity for binary treatment as follows.

Assumption 2.3 (Positivity / Overlap / Common Support) *For all values of covariates x present in the population of interest (i.e. x such that $P(X = x) > 0$),*

$$0 < P(T = 1 \mid X = x) < 1$$

To see why positivity is important, let's take a closer look at Equation 2.9:

$$\mathbb{E}[Y(1) - Y(0)] = \mathbb{E}_X \left[\mathbb{E}[Y \mid T = 1, X] - \mathbb{E}[Y \mid T = 0, X]\right] \tag{2.9 revisited}$$

In short, if we have a positivity violation, then we will be conditioning on a zero probability event. This is because there will be some value of x with non-zero probability for which $P(T = 1 \mid X = x) = 0$ or $P(T = 0 \mid X = x) = 0$. This means that for some value of x that we are marginalizing out in the above equation, $P(T = 1, X = x) = 0$ or $P(T = 0, X = x) = 0$, and these are the two events that we condition on in Equation 2.9.

To clearly see how a positivity violation translates to division by zero, let's rewrite the right-hand side of Equation 2.9. For discrete covariates and outcome, it can be rewritten as follows:

$$\sum_x P(X = x) \left(\sum_y y \, P(Y = y \mid T = 1, X = x) - \sum_y y \, P(Y = y \mid T = 0, X = x) \right) \tag{2.10}$$

Then, applying Bayes' rule, this can be further rewritten:

$$\sum_x P(X = x) \left(\sum_y y \, \frac{P(Y = y, T = 1, X = x)}{P(T = 1 \mid X = x) P(X = x)} - \sum_y y \, \frac{P(Y = y, T = 0, X = x)}{P(T = 0 \mid X = x) P(X = x)} \right) \tag{2.11}$$

In Equation 2.11, we can clearly see why positivity is essential. If $P(T = 1 \mid X = x) = 0$ for *any* level of covariates x with non-zero probability, then there is division by zero in the first term in the equation, so $\mathbb{E}_X \mathbb{E}[Y \mid T = 1, X]$ is undefined. Similarly, if $P(T = 1 \mid X = x) = 1$ for any level of x, then $P(T = 0 \mid X = x) = 0$, so there is division by zero in the second term and $\mathbb{E}_X \mathbb{E}[Y \mid T = 0, X]$ is undefined. With either of these violations of the positivity assumption, the causal effect is undefined.

Intuition That's the math for why we need the positivity assumption, but what's the intuition? Well, if we have a positivity violation, that means that within some subgroup of the data, everyone always receives treatment or everyone always receives the control. It wouldn't make sense to be able to estimate a causal effect of treatment vs. control in that subgroup since we see only treatment or only control. We never see the alternative in that subgroup.

Another name for positivity is *overlap*. The intuition for this name is that we want the covariate distribution of the treatment group to overlap with the covariate distribution of the control group. More specifically, we want $P(X \mid T = 1)$[9] to have the same support as $P(X \mid T = 0)$.[10] This is why another common alias for positivity is *common support*.

[9] Whenever we use a random variable (denoted by a capital letter) as the argument for P, we are referring to the whole distribution, rather than just the scalar that something like $P(x \mid T = 1)$ refers to.

[10] Active reading exercise: convince yourself that this formulation of overlap/positivity is equivalent to the formulation in Assumption 2.3.

The Positivity-Unconfoundedness Tradeoff Although conditioning on more covariates could lead to a higher chance of satisfying unconfoundedness, it can lead to a higher chance of violating positivity. As we increase the dimension of the covariates, we make the subgroups for any level x of the covariates smaller.[11] As each subgroup gets smaller, there is a higher and higher chance that either the whole subgroup will have treatment or the whole subgroup will have control. For example, once the size of any subgroup has decreased to one, positivity is guaranteed to not hold. See [6] for a rigorous argument of high-dimensional covariates leading to positivity violations.

[11] This is related to the *curse of dimensionality*.

[6]: D'Amour et al. (2017), *Overlap in Observational Studies with High-Dimensional Covariates*

Extrapolation Violations of the positivity assumption can actually lead to demanding too much from models and getting very bad behavior in return. Many causal effect estimators[12] fit a model to $\mathbb{E}[Y|t, x]$ using the (t, x, y) tuples as data. The inputs to these models are (t, x) pairs and the outputs are the corresponding outcomes. These models will be forced to extrapolate in regions (using their parametric assumptions) where $P(T = 1, X = x) = 0$ and regions where $P(T = 0, X = x) = 0$ when they are used in the adjustment formula (Theorem 2.1) in place of the corresponding conditional expectations.

[12] An "estimator" is a function that takes a dataset as input and outputs an estimate. We discuss this statistics terminology more in Section 2.4.

2.3.5 No interference, Consistency, and SUTVA

There are a few additional assumptions we've been smuggling in throughout this chapter. We will specify all the rest of these assumptions in this section. The first assumption in this section is that of *no interference*. No interference means that my outcome is unaffected by anyone else's treatment. Rather, my outcome is only a function of my own treatment. We've been using this assumption implicitly throughout this chapter. We'll now formalize it.

Assumption 2.4 (No Interference)

$$Y_i(t_1, \ldots, t_{i-1}, t_i, t_{i+1}, \ldots, t_n) = Y_i(t_i)$$

Of course, this assumption could be violated. For example, if the treatment is "get a dog" and the outcome is my happiness, it could easily be that my happiness is influenced by whether or not my friends get dogs because we could end up hanging out more to have our dogs play together. As you

might expect, violations of the no interference assumption are rampant in network data.

The last assumption is *consistency*. Consistency is the assumption that the outcome we observe Y is actually the potential outcome under the observed treatment T.

Assumption 2.5 (Consistency) *If the treatment is T, then the observed outcome Y is the potential outcome under treatment T. Formally,*

$$T = t \implies Y = Y(t) \tag{2.12}$$

We could write this equivalently as follow:

$$Y = Y(T) \tag{2.13}$$

Note that T is different from t, and $Y(T)$ is different from $Y(t)$. T is a random variable that corresponds to the observed treatment, whereas t is a specific value of treatment. Similarly, $Y(t)$ is the potential outcome for some specific value of treatment, whereas $Y(T)$ is the potential outcome for the actual value of treatment that we observe.

When we were using exchangeability to prove identifiability, we actually assumed consistency in Equation 2.4 to get the follow equality:

$$\mathbb{E}[Y(1) \mid T = 1] - \mathbb{E}[Y(0) \mid T = 0] = \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0]$$

Similarly, when we were using conditional exchangeability to prove identifiability, we assumed consistency in Equation 2.7.

It might seem like consistency is obviously true, but that is not always the case. For example, if the treatment specification is simply "get a dog" or "don't get a dog," this can be too coarse to yield consistency. It might be that if I were to get a puppy, I would observe $Y = 1$ (happiness) because I needed an energetic friend, but if I were to get an old, low-energy dog, I would observe $Y = 0$ (unhappiness). However, both of these treatments fall under the category of "get a dog," so both correspond to $T = 1$. This means that $Y(1)$ is not well defined, since it will be 1 or 0, depending on something that is not captured by the treatment specification. In this sense, consistency encompasses the assumption that is sometimes referred to as "no multiple versions of treatment." See Sections 3.4 and 3.5 of Hernán and Robins [7] and references therein for more discussion on this topic.

[7]: Hernán and Robins (2020), *Causal Inference: What If*

SUTVA You will also commonly see the *stable unit-treatment value assumption* (SUTVA) in the literature. SUTVA is satisfied if unit (individual) i's outcome is simply a function of unit i's treatment. Therefore, SUTVA is a combination of consistency and no interference (and also deterministic potential outcomes).[13]

[13] Active reading exercise: convince yourself that SUTVA is a combination of consistency and no inference

2.3.6 Tying It All Together

We introduced unconfoundedness (conditional exchangeability) first because it is the main causal assumption. However, all of the assumptions are necessary:

1. Unconfoundedness (Assumption 2.2)
2. Positivity (Assumption 2.3)
3. No interference (Assumption 2.4)
4. Consistency (Assumption 2.5)

We'll now review the proof of the adjustment formula (Theorem 2.1) that was done in Equation 2.5 through Equation 2.9 and list which assumptions are used for each step. Even before we get to these equations, we use the no interference assumption to justify that the quantity we should be looking at for causal inference is $\mathbb{E}[Y(1) - Y(0)]$, rather than something more complex like the left-hand side of Assumption 2.4. In the proof below, the first two equalities follow from mathematical facts, whereas the last two follow from these key assumptions.

Proof of Theorem 2.1.

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0)] &= \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] && \text{(linearity of expectation)} \\
&= \mathbb{E}_X\left[\mathbb{E}[Y(1) \mid X] - \mathbb{E}[Y(0) \mid X]\right] \\
& && \text{(law of iterated expectations)} \\
&= \mathbb{E}_X\left[\mathbb{E}[Y(1) \mid T = 1,\, X] - \mathbb{E}[Y(0) \mid T = 0,\, X]\right] \\
& && \text{(unconfoundedness and positivity)} \\
&= \mathbb{E}_X\left[\mathbb{E}[Y \mid T = 1,\, X] - \mathbb{E}[Y \mid T = 0,\, X]\right] \\
& && \text{(consistency)}
\end{aligned}$$

□

That's how all of these assumptions tie together to give us identifiability of the ATE. We'll soon see how to use this result to get an actual estimated number for the ATE.

2.4 Fancy Statistics Terminology Defancified

Before we start computing concrete numbers for the ATE, we must quickly introduce some terminology from statistics that will help clarify the discussion. An *estimand* is the quantity that we want to estimate. For example, $\mathbb{E}_X\left[\mathbb{E}[Y \mid T = 1,\, X] - \mathbb{E}[Y \mid T = 0,\, X]\right]$ is the estimand we care about for estimating the ATE. An *estimate* (noun) is an approximation of some estimand, which we get using data. We will see concrete numbers in the next section; these are estimates. Given some estimand α, we write an estimate of that estimand by simply putting a hat on it: $\hat{\alpha}$. And an *estimator* is a function that maps a dataset to an estimate of the estimand. The process that we will use to go from data + estimand to a concrete number is known as *estimation*. To *estimate* (verb) is to feed data into an estimator to get an estimate.

In this book, we will use even more specific language that allows us to make the distinction between causal quantities and statistical quantities. We will use the phrase *causal estimand* to refer to any estimand that contains a potential outcome in it. We will use the phrase *statistical estimand* to denote the complement: any estimand that does not contain a potential outcome.[14] For an example, recall the adjustment formula

[14] As we will see in Chapter 4, we will equivalently refer to a causal estimand as any estimand that contains a *do*-operator, and we will refer to a statistical estimand as any estimand that does not contain a *do*-operator.

(Theorem 2.1):

$$\mathbb{E}[Y(1) - Y(0)] = \mathbb{E}_X\left[\mathbb{E}[Y \mid T = 1,\ X] - \mathbb{E}[Y \mid T = 0,\ X]\right] \tag{2.14}$$

$\mathbb{E}[Y(1) - Y(0)]$ is the causal estimand that we are interested in. In order to actually estimate this causal estimand, we must translate it into a statistical estimand: $\mathbb{E}_X\left[\mathbb{E}[Y \mid T = 1,\ X] - \mathbb{E}[Y \mid T = 0,\ X]\right]$.[15]

[15] Active reading exercise: Why can't we directly estimate a causal estimand without first translating it to a statistical estimand?

When we say "identification" in this book, we are referring to the process of moving from a *causal* estimand to an equivalent *statistical* estimand. When we say "estimation," we are referring to the process of moving from a statistical estimand to an estimate. We illustrate this in the flowchart in Figure 2.5.

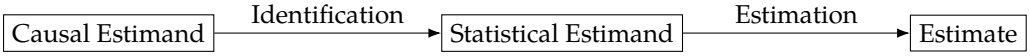

Figure 2.5: The Identification-Estimation Flowchart – a flowchart that illustrates the process of moving from a target causal estimand to a corresponding estimate, through identification and estimation.

What do we do when we go to actually estimate quantities such as $\mathbb{E}_X\left[\mathbb{E}[Y \mid T = 1,\ X] - \mathbb{E}[Y \mid T = 0,\ X]\right]$? We will often use a model (e.g. linear regression or some more fancy predictor from machine learning) in place of the conditional expectations $\mathbb{E}[Y \mid T = t,\ X = x]$. We will refer to estimators that use models like this as *model-assisted estimators*. Now that we've gotten some of this terminology out of the way, we can proceed to an example of estimating the ATE.

2.5 A Complete Example with Estimation

Theorem 2.1 and the corresponding recent copy in Equation 2.14 give us identification. However, we haven't discussed estimation at all. In this section, we will give a short example complete with estimation. We will cover the topic of estimation of causal effects more completely in Chapter 7.

We use Luque-Fernandez et al. [8]'s example from epidemiology. The outcome Y of interest is (systolic) blood pressure. This is an important outcome because roughly 46% of Americans have high blood pressure, and high blood pressure is associated with increased risk of mortality [9]. The "treatment" T of interest is sodium intake. Sodium intake is a continuous variable; in order to easily apply Equation 2.14, which is specified for binary treatment, we will binarize T by letting $T = 1$ denote daily sodium intake above 3.5 grams and letting $T = 0$ denote daily sodium intake below 3.5 grams.[16] We will be estimating the causal effect of sodium intake on blood pressure. In our data, we also have the age of the individuals and amount of protein in their urine as covariates X. Luque-Fernandez et al. [8] run a simulation, taking care to be sure that the range of values is "biologically plausible and as close to reality as possible."

[8]: Luque-Fernandez et al. (2018), 'Educational Note: Paradoxical collider effect in the analysis of non-communicable disease epidemiological data: a reproducible illustration and web application'

[9]: Virani et al. (2020), 'Heart Disease and Stroke Statistics—2020 Update: A Report From the American Heart Association'

[16] As we will see, this binarization is purely pedagogical and does not reflect any limitations of adjusting for confounders.

Because we are using data from a simulation, we know that the true ATE of sodium on blood pressure is 1.05. More concretely, the line of code that generates blood pressure Y looks as follows:

```
blood_pressure = 1.05 * sodium + ...
```

Now, how do we actually estimate the ATE? First, we assume consistency, positivity, and unconfoundedness given X. As we recently recalled in Equation 2.14, this means that we've identified the ATE as

$$\mathbb{E}_X \left[\mathbb{E}[Y \mid T = 1, X] - \mathbb{E}[Y \mid T = 0, X] \right] .$$

We then take that outer expectation over X and replace it with an empirical mean over the data, giving us the following:

$$\frac{1}{n} \sum_i \left[\mathbb{E}[Y \mid T = 1, X = x_i] - \mathbb{E}[Y \mid T = 0, X = x_i] \right] \tag{2.15}$$

To complete our estimator, we then fit some machine learning model to the conditional expectation $\mathbb{E}[Y \mid t, x]$. Minimizing the mean-squared error (MSE) of predicting Y from (T, X) pairs is equivalent to modeling this conditional expectation [see, e.g., 10, Section 2.4]. Therefore, we can plug in any machine learning model for $\mathbb{E}[Y \mid t, x]$, which gives us a model-assisted estimator. We'll use linear regression here, which works out nicely since blood pressure is generated as a linear combination of other variables, in this simulation. We give Python code for this below, where our data are in a Pandas DataFrame called `df`. We fit the model for $\mathbb{E}[Y \mid t, x]$ in line 8, and we take the empirical mean over X in lines 10-14.

[10]: Hastie et al. (2001), *The Elements of Statistical Learning*

```
1  import numpy as np
2  import pandas as pd
3  from sklearn.linear_model import LinearRegression
4
5  Xt = df[['sodium', 'age', 'proteinuria']]
6  y = df['blood_pressure']
7  model = LinearRegression()
8  model.fit(Xt, y)
9
10 Xt1 = pd.DataFrame.copy(Xt)
11 Xt1['sodium'] = 1
12 Xt0 = pd.DataFrame.copy(Xt)
13 Xt0['sodium'] = 0
14 ate_est = np.mean(model.predict(Xt1) - model.predict(Xt0))
15 print('ATE estimate:', ate_est)
```

Listing 2.1: Python code for estimating the ATE

Full code, complete with simulation, is available at https://github.com/bradyneal/causal-book-code/blob/master/sodium_example.py.

This yields an ATE estimate of 0.85. If we were to naively regress Y on only T, which corresponds to replacing line 5 in Listing 2.1 with `Xt = df[['sodium']]`,[17] we would get an ATE estimate of 5.33. That's a $\frac{|5.33-1.05|}{1.05} \times 100\% = \mathbf{407}\%$ error! In contrast, when we control for X (as in Listing 2.1), our percent error is only $\frac{|.85-1.05|}{1.05} \times 100\% = \mathbf{19}\%$.

[17] Active reading exercise: This naive version is equivalent to just taking the associational difference: $\mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0]$. Why?

All of the above is done using the adjustment formula with model-assisted estimation, where we first fit a model for the conditional expectation $\mathbb{E}[Y \mid t, x]$, and then we take an empirical mean over X, using that model. However, because we are using a linear model, this is equivalent to just taking the coefficient in front of T in the linear regression as the ATE estimate. This is what we do in the following code (which gives the exact same ATE estimate):

```
1  Xt = df[['sodium', 'age', 'proteinuria']]
2  y = df['blood_pressure']
3  model = LinearRegression()
```

Listing 2.2: Python code for estimating the ATE using the coefficient of linear regression

```
model.fit(Xt, y)
ate_est = model.coef_[0]
print('ATE estimate:', ate_est)
```

Continuous Treatment What if we allow the treatment, daily sodium intake, to remain continuous, instead of binarizing it? The cool thing about just taking the regression coefficient as the ATE estimate is that it doesn't require taking a difference between two values of treatment (e.g. $T = 1$ and $T = 0$), so it trivially generalizes to when T is continuous. When T is continous, we care about how $\mathbb{E}[Y(t)]$ changes with t. Since we are assuming $\mathbb{E}[Y(t)]$ is linear, this change is completely captured by $\frac{d}{dt}\mathbb{E}[Y(t)]$.[18] When $\mathbb{E}[Y(t)]$ is linear, it turns out that this quantity is exactly what taking the coefficient from linear regression estimates. Seemingly magically, we have compressed all of $\mathbb{E}[Y(t)] = \mathbb{E}[Y \mid t]$, which is a *function* of t, into a single value.

However, this effortless compression of all of $\mathbb{E}[Y \mid t]$ for continuous t comes as a cost: the linear parametric form we assumed. If this model were misspecified,[19] our ATE estimate would be biased. And because linear models are so simple, they will likely be misspecified. For example, the following assumption is implicit in assuming that a linear model is well-specified: the treatment effect is the same for all individuals. See Morgan and Winship [12, Sections 6.2 and 6.3] for a more complete critique of using the coefficient in front of treatment as the ATE estimate.

[18] Concisely summarizing *nonlinear* functions $\mathbb{E}[Y(t)]$ is an open problem. See, e.g., Janzing et al. [11].

[11]: Janzing et al. (2013), 'Quantifying causal influences'

[19] By "misspecified," we mean that the functional form of the model does not match the functional form of the data generating process.

[12]: Morgan and Winship (2014), *Counterfactuals and Causal Inference: Methods and Principles for Social Research*

The Flow of Association and Causation in Graphs

3

We've been using causal graphs in the previous chapters to aid intuition. In this chapter, we will introduce the formalisms that underlie this intuition. Hopefully, we have sufficiently motivated this chapter and made the utility of graphical models clear with all of the graphical interpretations of concepts in previous chapters.

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3.1 Graph Terminology

In this section, we will use the *terminology machine gun* (see Figure 3.1). To be able to use nice convenient graph language in the following sections, rapid-firing a lot of graph terminology is a necessary evil, unfortunately.

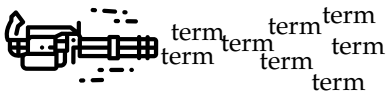


Figure 3.1: Terminology machine gun

The term "graph" is often used to describe a variety of visualizations. For example, "graph" might refer to a visualization of a single variable function $f(x)$, where x is plotted on the x-axis and $f(x)$ is plotted on the y-axis. Or "bar graph" might be used as a synonym for a bar chart. However, in graph theory, the term "graph" refers to a specific mathematical object.

A *graph* is a collection of *nodes* (also called "vertices") and *edges* that connect the nodes. For example, in Figure 3.2, A, B, and C are the nodes of the graph, and the lines connecting them are the edges. Figure 3.2 is called an *undirected graph* because the edges do not have any direction. In contrast, Figure 3.3 is a *directed graph*. A directed graph's edges go out of a *parent* node and into a *child* node, with the arrows signifying which direction the edges are going. We will denote the parents of a node X with $\text{pa}(X)$. We'll use an even simpler shorthand when the nodes are ordered so that we can denote the i^{th} node by X_i; in that case, we will also denote the parents of X_i by pa_i. Two nodes are said to be *adjacent* if they are connected by an edge. For example, in both Figure 3.2 and Figure 3.3, A and C are adjacent, but A and D are not.

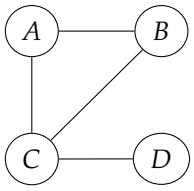


Figure 3.2: Undirected graph

A *path* in a graph is any sequence of adjacent nodes, regardless of the direction of the edges that join them. For example, $A - C - B$ is a path in Figure 3.2, and $A \to C \leftarrow B$ is a path in Figure 3.3. A *directed path* is a path that consists of directed edges that are all directed in the same direction (no two edges along the path both point into or both point out of the same node). For example, $A \to C \to D$ is a directed path in Figure 3.3, but $A \to C \leftarrow B$ and $C \leftarrow A \to B$ are not.

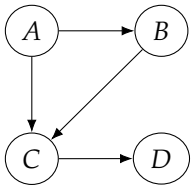


Figure 3.3: Directed graph

If there is a directed path that starts at node X and ends at node Y, then X is an *ancestor* of Y, and Y is a *descendant* of X. We will denote descendants of X by $\text{de}(X)$. For example, in Figure 3.3, A is an ancestor of B and D, and B and D are both descendants of A ($\text{de}(A)$). If X is an ancestor of itself, then some funky time travel has taken place. In seriousness, a directed path from some node X back to itself is known as a *cycle* (see Figure 3.4). If there are no cycles in a directed graph, the graph is known

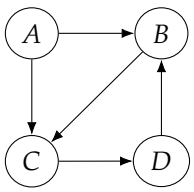


Figure 3.4: Directed graph with cycle

as a *directed acyclic graph* (DAG). The graphs we focus on in this book will mostly be DAGs.

If two parents X and Y share some child Z, but there is no edge connecting X and Y, then $X \to Z \leftarrow Y$ is known as an *immorality*. Seriously; that's a real term in graphical models. For example, if we remove the $A \to B$ from Figure 3.3 to get Figure 3.5, then $A \to C \leftarrow B$ is an immorality.

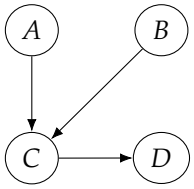


Figure 3.5: Directed graph with immorality

3.2 Bayesian Networks

It turns out that much of the work for causal graphical models was done in the field of probabilistic graphical models. Probabilistic graphical models are statistical models while causal graphical models are causal models. Bayesian networks are the main probabilistic graphical model that causal graphical models (causal Bayesian networks) inherit most of their properties from.

Imagine that we only cared about modeling association, without any causal modeling. We would want to model the data distribution $P(x_1, x_2, \ldots, x_n)$. In general, we can use the chain rule of probability to factorize any distribution:

$$P(x_1, x_2, \ldots, x_n) = P(x_1) \prod_i P(x_i \mid x_{i-1}, \ldots, x_1) \tag{3.1}$$

However, if we were to model these factors with tables, it would take an exponential number of parameters. To see this, take each x_i to be binary and consider how we would model the factor $P(x_n \mid x_{n-1}, \ldots, x_1)$. Since x_n is binary, we only need to model $P(X_n = 1 \mid x_{n-1}, \ldots, x_1)$ because $P(X_n = 0 \mid x_{n-1}, \ldots, x_1)$ is simply $1 - P(X_n = 1 \mid x_{n-1}, \ldots, x_1)$. Well, we would need 2^{n-1} parameters to model this. As a specific example, let $n = 4$. As we can see in Table 3.1, this would require $2^{4-1} = 8$ parameters: $\alpha_1, \ldots, \alpha_8$. This brute-force parametrization quickly becomes intractable as n increases.

Table 3.1: Table required to model the single factor $P(x_n \mid x_{n-1}, \ldots, x_1)$ where $n = 4$ and the variables are binary. The number of parameters to necessary is exponential in n.

x_1	x_2	x_3	$P(x_4 \mid x_3, x_2, x_1)$
0	0	0	α_1
0	0	1	α_2
0	1	0	α_3
0	1	1	α_4
1	0	0	α_5
1	0	1	α_6
1	1	0	α_7
1	1	1	α_8

An intuitive way to more efficiently model many variables together in a joint distribution is to only model local dependencies. For example, rather than modeling the X_4 factor as $P(x_4|x_3, x_2, x_1)$, we could model it as $P(x_4|x_3)$ if we have reason to believe that X_4 only locally depends on X_3. In fact, in the corresponding graph in Figure 3.6, the only node that feeds into X_4 is X_3. This is meant to signify that X_4 only locally depends on X_3. Whenever we use a graph G in relation to a probability distribution P, there will always be a one-to-one mapping between the nodes in G and the random variables in P, so when we talk about nodes being independent, we mean the corresponding random variables are independent.

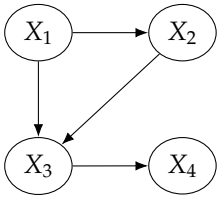


Figure 3.6: Four node DAG where X_4 locally depends on only X_3.

Given a probability distribution and a corresponding directed acyclic graph (DAG), we can formalize the specification of independencies with the *local Markov assumption*:

Assumption 3.1 (Local Markov Assumption) *Given its parents in the DAG, a node X is independent of all its non-descendants.*

This assumption (along with specific DAGs) gives us a lot. We will demonstrate this in the next few equations. In our four variable example, the chain rule of probability tells us that we can factorize any P such that

$$P(x_1, x_2, x_3, x_4) = P(x_1)\,P(x_2 \mid x_1)\,P(x_3 \mid x_2, x_1)\,P(x_4 \mid x_3, x_2, x_1)\,. \tag{3.2}$$

If P is Markov with respect to the graph[1] in Figure 3.6, then we can simplify the last factor:

$$P(x_1, x_2, x_3, x_4) = P(x_1)\,P(x_2 \mid x_1)\,P(x_3 \mid x_2, x_1)\,P(x_4 \mid x_3)\,. \tag{3.3}$$

If we further remove edges, removing $X_1 \to X_2$ and $X_2 \to X_3$ as in Figure 3.7, we can further simplify the factorization of P:

$$P(x_1, x_2, x_3, x_4) = P(x_1)\,P(x_2)\,P(x_3 \mid x_1)\,P(x_4 \mid x_3)\,. \tag{3.4}$$

With the understanding that we have hopefully built up from a few examples,[2] we will now state one of the main consequences of the local Markov assumption:

Definition 3.1 (Bayesian Network Factorization) *Given a probability distribution P and a DAG G, P factorizes according to G if*

$$P(x_1, \ldots, x_n) = \prod_i P(x_i \mid \text{pa}_i)$$

Hopefully you see the resemblance between the move from Equation 3.2 to Equation 3.3 or the move to Equation 3.4 and the generalization of this that is presented in Definition 3.1.

The Bayesian network factorization is also known as the *chain rule for Bayesian networks* or *Markov compatibility*. For example, if P factorizes according to G, then P and G are Markov compatible.

We have given the intuition of how the local Markov assumption implies the Bayesian network factorization, and it turns out that the two are actually equivalent. In other words, we could have started with the Bayesian network factorization as the main assumption (and labeled it as an assumption) and shown that it implies the local Markov assumption. See Koller and Friedman [13, Chapter 3] for these proofs and more information on this topic.

As important as the local Markov assumption is, it only gives us information about the *independencies* in P that a DAG implies. It does not even tell us that if X and Y are adjacent in the DAG, then X and Y are dependent. And this additional information is very commonly assumed in causal DAGs. To get this guaranteed dependence between adjacent nodes, we will generally assume a slightly stronger assumption than the local Markov assumption: *minimality*.

Assumption 3.2 (Minimality Assumption)
1. *Given its parents in the DAG, a node X is independent of all its non-descendants (Assumption 3.1).*
2. *Adjacent nodes in the DAG are dependent.*[3]

[1] A probability distribution is said to be (locally) Markov with respect to a DAG if they satisfy the local Markov assumption.

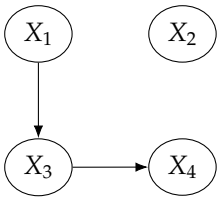


Figure 3.7: Four node DAG with even more independencies.

[2] Active reading exercise:: ensure that you know how we get from Equation 3.2 to Equation 3.3 and to Equation 3.4 using the local Markov assumption.

[13]: Koller and Friedman (2009), *Probabilistic Graphical Models: Principles and Techniques*

[3] This is often equivalently stated in the following way: if we were to remove any edges from the DAG, P would not be Markov with respect to the graph with the removed edges [see, e.g., 14, Section 6.5.3].

[14]: Peters et al. (2017), *Elements of Causal Inference: Foundations and Learning Algorithms*

To see why this assumption is named "minimality" consider, what we know when we know that P is Markov with respect to a DAG G. We know that P satisfies a set of independencies that are specific to the structure of G. If P and G also satisfy minimality, then this set of independencies is *minimal* in the sense the P does not satisfy any additional independencies. This is equivalent to saying that adjacent nodes are dependent.

For example, if the DAG were simply two connected nodes X and Y as in Figure 3.8, the local Markov assumption would tell us that we can factorize $P(x, y)$ as $P(x)P(y|x)$, but it would also allow us to factorize $P(x, y)$ as $P(x)P(y)$, meaning it allows distributions where X and Y are independent. In contrast, the minimality assumption does not allow this additional independence. Minimality would tell us to factorize $P(x, y)$ as $P(x)P(y|x)$, *and* it would tell us that no additional independencies ($X \perp\!\!\!\perp Y$) exist in P that are minimal with respect to Figure 3.8.

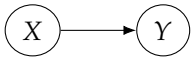


Figure 3.8: Two connected nodes

Because removing edges in a Bayesian network is equivalent to adding independencies,[4] the minimality assumption is equivalent to saying that we can't remove any more edges from the graph. In a sense, every edge is "active." More concretely, consider that P and G are Markov compatible and that G' is what we get when we remove some edge from G. If P is also Markov with respect to G', then P is not minimal with respect to G.

[4] Active reading exercise: why is *removing* edges in a Bayesian network equivalent to *adding* independencies?

Armed with the minimality assumption and what it implies about how distributions factorize when they are Markov with respect to some DAG (Definition 3.1), we are now ready to discuss the flow of association in DAGs. However, because everything in this section is purely statistical, we are not ready to discuss the flow of *causation* in DAGs. To do that, we must make causal assumptions. Pedagogically, this will also allow us to use intuitive causal language when we explain the flow of association.

3.3 Causal Graphs

The previous section was all about statistical models and modeling association. In this section, we will augment these models with causal assumptions, turning them into causal models and allowing us to study causation. In order to introduce causal assumptions, we must first have an understanding of what it means for X to be a cause of Y.

Definition 3.2 (What is a cause?) *A variable X is said to be a cause of a variable Y if Y can change in response to changes in X.*[5]

[5] See Section 4.5.1 for a definition using mathematical notation.

Another phrase commonly used to describe this primitive is that Y "listens" to X. With this, we can now specify the main causal assumption that we will use throughout this book.

Assumption 3.3 ((Strict) Causal Edges Assumption) *In a directed graph, every parent is a direct cause of all its children.*

Here, the set of *direct causes* of Y is everything that Y directly responds to; if we fix all of the direct causes of Y, then changing any other cause of Y won't induce any changes in Y. This assumption is "strict" in the sense

that every edge is "active," just like in DAGs that satisfy minimality. In other words, because the definition of a cause (Definition 3.2) implies that a cause and its effect are dependent and because we are assuming all parents are causes of their children, we are assuming that parents and their children are dependent. So the second part of minimality (Assumption 3.2) is baked into the strict causal edges assumption.

In contrast, the *non-strict* causal edges assumption would allow for some parents to not be causes of their children. It would just assume that children are not causes of their parents. This allows us to draw graphs with extra edges to make fewer assumptions, just like we would in Bayesian networks, where more edges means fewer independence assumptions. Causal graphs are sometimes drawn with this kind of non-minimal meaning, but the vast majority of the time, when someone draws a causal graph, they mean that parents are causes of their children. Therefore, unless we specify otherwise, throughout this book, we will use "causal graph" to refer to a DAG that satisfies the strict causal edges assumption. And we will often omit the word "strict" when we refer to this assumption.

When we add the causal edges assumption, directed paths in the DAG take on a very special meaning; they correspond to causation. This is in contrast to other paths in the graph, which association may flow along, but causation certainly may not. This will become more clear when we go into detail on these other kinds of paths in Sections 3.5 and 3.6.

Moving forward, we will now think of the edges of graphs as causal, in order to describe concepts intuitively with causal language. However, all of the associational claims about statistical independence will still hold, even when the edges do not have causal meaning like in the vanilla Bayesian networks of Section 3.2.

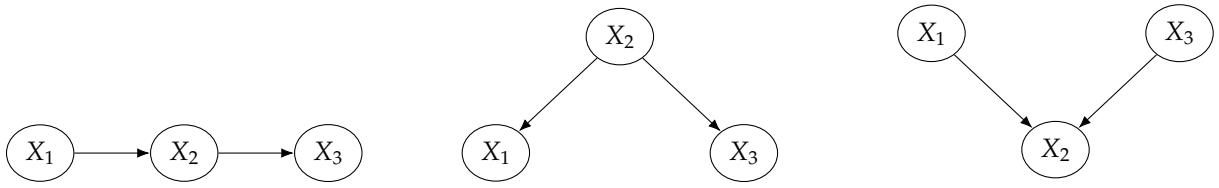
As we will see in the next few sections, the main assumptions that we need for our causal graphical models to tell us how association and causation flow between variables are the following two:

1. Local Markov Assumption (Assumption 3.1)
2. Causal Edges Assumption (Assumption 3.3)

We will discuss these assumptions throughout the next few sections and come back to discuss them more fully again in Section 3.8 after we've established the necessary preliminaries.

3.4 Two-Node Graphs and Graphical Building Blocks

Now that we've gotten the basic assumptions and definitions out of the way, we can get to the core of this chapter: the flow of association and causation in DAGs. We can understand this flow in general DAGs by understanding the flow in the minimal building blocks of graphs. These minimal building blocks consist of chains (Figure 3.9a), forks (Figure 3.9b), immoralities (Figure 3.9c), two unconnected nodes (Figure 3.10), and two connected nodes (Figure 3.11).



(a) Chain **(b)** Fork **(c)** Immorality

Figure 3.9: Basic graph building blocks

By "flow of association," we mean whether any two nodes in a graph are associated or not associated. Another way of saying this is whether two nodes are (statistically) dependent or (statistically) independent. Additionally, we will study whether two nodes are *conditionally* independent or not.

For each building block, we will give the intuition for why two nodes are (conditionally) independent or not, and we will give a proof as well. We can prove that two nodes A and B are conditionally independent given some set of nodes C by simply showing that $P(a, b|c)$ factorizes as $P(a|c)\,P(b|c)$. We will now do this in the case of the simplest basic building block: two unconnected nodes.

Given a graph that is just two unconnected nodes, as depicted in Figure 3.10, these nodes are not associated simply because there is no edge between them. To show this, consider the factorization of $P(x_1, x_2)$ that the Bayesian network factorization (Definition 3.1) gives us:

$$P(x_1, x_2) = P(x_1)\,P(x_2) \tag{3.5}$$

Figure 3.10: Two unconnected nodes

That's it; applying the Bayesian network factorization immediately gives us a proof that the two nodes X_1 and X_2 are unassociated (independent) in this building block. And what is the assumption that allows us to prove this? That P is Markov with respect to the graph in Figure 3.10.

In contrast, if there is an edge between the two nodes (as in Figure 3.11), then the two nodes are associated. The assumption we leverage here is the causal edges assumption (Assumption 3.3), which means that X_1 is a cause of X_2. Since X_1 is a cause of X_2, X_2 must be able to change in response to changes in X_1, so X_2 and X_1 are associated. In general, any time two nodes are adjacent in a causal graph, they are associated.[6] We will see this same concept several more times in Section 3.5 and Section 3.6.

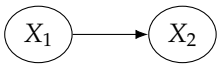


Figure 3.11: Two connected nodes

[6] Two adjacent nodes in a *non-strict* causal graph can be unassociated.

Now that we've covered the relevant two-node graphs, we'll cover the flow of association in the remaining graphical building blocks (three-node graphs in Figure 3.9), starting with chain graphs.

3.5 Chains and Forks

Chains (Figure 3.12) and forks (Figure 3.13) share the same set of dependencies. In both structures, X_1 and X_2 are dependent, and X_2 and X_3 are dependent for the same reason that we discussed toward the end of Section 3.4. Adjacent nodes are always dependent when we make the causal edges assumption (Assumption 3.3). What about X_1 and X_3,

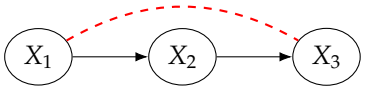


Figure 3.12: Chain with flow of association drawn as a dashed red arc.

though? Does association flow from X_1 to X_3 through X_2 in chains and forks?

Usually, yes, X_1 and X_3 are associated in both chains and forks. In chain graphs, X_1 and X_3 are usually dependent simply because X_1 causes changes in X_2 which then causes changes in X_3. In a fork graph, X_1 and X_3 are also usually dependent. This is because the same value that X_2 takes on is used to determine both the value that X_1 takes on and the value that X_3 takes on. In other words, X_1 and X_3 are associated through their (shared) common cause. We use the word "usually" throughout this paragraph because there exist pathological cases where the conditional distributions $P(x_2|x_1)$ and $P(x_3|x_2)$ are misaligned in such a specific way that makes X_1 and X_3 not actually associated [see, e.g., 15, Section 2.2].

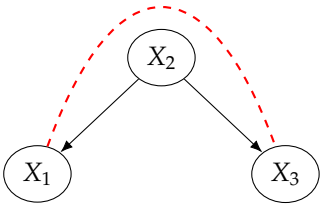


Figure 3.13: Fork with flow of association drawn as a dashed red arc.

[15]: Pearl et al. (2016), *Causal inference in statistics: A primer*

An intuitive graphical way of thinking about X_1 and X_3 being associated in chains and forks is to visualize the flow of association. We visualize this with a dashed red line in Figure 3.12 and Figure 3.13. In the chain graph (Figure 3.12), association flows from X_1 to X_3 along the path $X_1 \rightarrow X_2 \rightarrow X_3$. Symmetrically, association flows from X_3 to X_1 along that same path, just running opposite the arrows. In the fork graph (Figure 3.13), association flows from X_1 to X_3 along the path $X_1 \leftarrow X_2 \rightarrow X_3$. And similarly, we can think of association flowing from X_3 to X_1 along that same path, just as was the case with chains. In general, the flow of association is symmetric.

Chains and forks also share the same set of *independencies*. When we condition on X_2 in both graphs, it blocks the flow of association from X_1 to X_3. This is because of the local Markov assumption; each variable can locally depend on only its parents. So when we condition on X_2 (X_3's parent in both graphs), X_3 becomes independent of X_1 (and vice versa).

We will refer to this independence as an instance of a *blocked path*. We illustrate these blocked paths in Figure 3.14 and Figure 3.15. Conditioning blocks the flow of association in chains and forks. Without conditioning, association is free to flow in chains and forks; we will refer to this as an *unblocked path*. However, the situation is completely different with immoralities, as we will see in the next section.

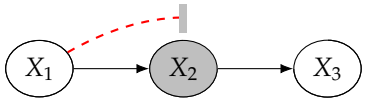


Figure 3.14: Chain with association blocked by conditioning on X_2.

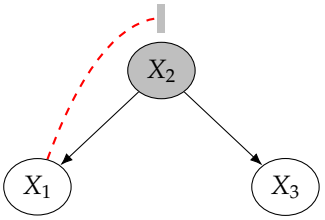


Figure 3.15: Fork with association blocked by conditioning on X_2.

That's all nice intuition, but what about the proof? We can prove that $X_1 \perp\!\!\!\perp X_3 \mid X_2$ using just the local Markov assumption. We will do this by showing that $P(x_1, x_3 \mid x_2) = P(x_1 \mid x_2)\, P(x_3 \mid x_2)$. We'll show the proof for chain graphs. It is usually useful to start with the Bayesian network factorization. For chains, we can factorize $P(x_1, x_2, x_3)$ as follows:

$$P(x_1, x_2, x_3) = P(x_1)\, P(x_2|x_1)\, P(x_3|x_2) \tag{3.6}$$

Bayes' rule tells us that $P(x_1, x_3 \mid x_2) = \frac{P(x_1, x_2, x_3)}{P(x_2)}$, so we have

$$P(x_1, x_3 \mid x_2) = \frac{P(x_1)\, P(x_2|x_1)\, P(x_3|x_2)}{P(x_2)} \tag{3.7}$$

Since we're looking to end up with $P(x_1 \mid x_2)\, P(x_3 \mid x_2)$ and we already have $P(x_3|x_2)$, we must turn the rest into $P(x_1 \mid x_2)$. We can do this by

another application of Bayes rule:

$$P(x_1, x_3 \mid x_2) = \frac{P(x_1, x_2)}{P(x_2)} P(x_3|x_2) \tag{3.8}$$

$$= P(x_1|x_2)\, P(x_3|x_2) \tag{3.9}$$

With that, we've shown that $X_1 \perp\!\!\!\perp X_3 \mid X_2$. Try it yourself; prove the analog in forks.[7]

[7] Active reading exercise: prove that $X_1 \perp\!\!\!\perp X_3 \mid X_2$ for forks (Figure 3.15).

Flow of Causation The flow of association is symmetric, whereas the flow of causation is not. Under the causal edges assumption (Assumption 3.3), causation only flows in a single direction. Causation only flows along *directed* paths. Association flows along any path that does not contain an immorality.

3.6 Colliders and their Descendants

Recall from Section 3.1 that we have an immorality when we have a child whose two parents do not have an edge connecting them (Figure 3.16). And in this graph structure, the child is known as a bastard. No, just kidding; it's called a *collider*.

In contrast to chains and forks, in an immorality, $X_1 \perp\!\!\!\perp X_3$. Look at the graph structure and think about it a bit. Why would X_1 and X_3 be associated? One isn't the descendant of the other like in chains, and they don't share a common cause like in forks. Rather, we can think of X_1 and X_3 simply as unrelated events that happen, which happen to both contribute to some common effect (X_2). To show this, we apply the Bayesian network factorization and marginalize out x_2:

$$P(x_1, x_3) = \sum_{x_2} P(x_1, x_2, x_3) \tag{3.10}$$

$$= \sum_{x_2} P(x_1)\, P(x_3)\, P(x_2 \mid x_1, x_3) \tag{3.11}$$

$$= P(x_1)\, P(x_3) \sum_{x_2} P(x_2 \mid x_1, x_3) \tag{3.12}$$

$$= P(x_1)\, P(x_3) \tag{3.13}$$

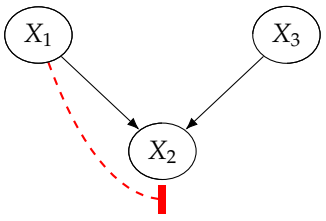


Figure 3.16: Immorality with association blocked by a collider.

We illustrate the independence of X_1 and X_3 in Figure 3.16 by showing that the association that we could have imagined as flowing along the path $X_1 \rightarrow X_2 \leftarrow X_3$ is actually blocked at X_2. Because we have a collider on the path connecting X_1 and X_3, association does not flow through that path. This is another example of a *blocked path*, but this time the path is not blocked by conditioning; the path is blocked by a collider.

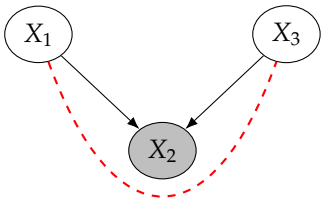


Figure 3.17: Immorality with association unblocked by conditioning on the collider.

Good-Looking Men are Jerks Oddly enough, when we condition on the collider X_2, its parents X_1 and X_3 become dependent (depicted in Figure 3.17). An example is the easiest way to see why this is the case. Imagine that you're out dating men, and you notice that most of the nice men you meet are not very good-looking, and most of the good-looking men you meet are jerks. It seems that you have to choose between looks and kindness. In other words, it seems like kindness and looks are negatively associated. However, what if I also told you that there is an important third variable here: availability (whether men are

already in a relationship or not)? And what if I told you that a man's availability is largely determined by their looks and kindness; if they are both good-looking and kind, then they are in a relationship. The available men are the remaining ones, the ones who are either not good-looking or not kind. You see an association between looks and kindness because you've conditioned on a collider (availability). You're only looking at men who are not in a relationship. You can see the causal structure of this example by taking Figure 3.17 and replacing X_1 with "looks," X_3 with "kindness," and X_2 with "availability."

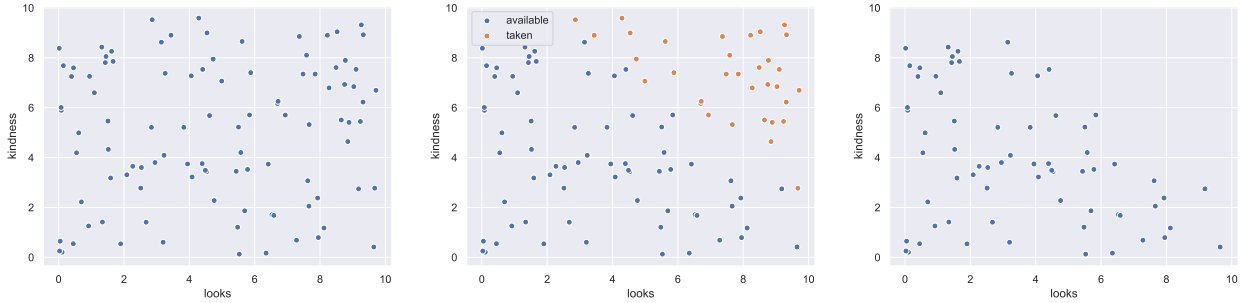
The above example naturally suggests that, when dating men, maybe you should consider not conditioning on X_2 = "not in a relationship" and, instead, condition on X_2 = "in a relationship." However, you could run into other variables X_4 that introduce new immoralities there. Such moral questions are outside the scope of this book.

Active reading exercise: Come up with your own example of an immorality and how conditioning on the collider induces association between its parents. Hint: think of rare events for X_1 and X_3 where, if either of them happens, some outcome X_2 will happen.

Returning to inside the scope of this book, we have that conditioning on a collider can turn a blocked path into an *unblocked path*. The parents X_1 and X_3 are not associated in the general population, but when we condition on their shared child X_2 taking on a specific value, they become associated. Conditioning on the collider X_2 allows associated to flow along the path $X_1 \rightarrow X_2 \leftarrow X_3$, despite the fact that it does not when we don't condition on X_2. We illustrate this in the move from Figure 3.16 to Figure 3.17.

We also illustrate this with a scatter plot in Figure 3.18. In Figure 3.18a, we plot the whole population, with kindness on the x-axis and looks on the y-axis. As you can see, the variables are not associated in the general population. However, if we remove the ones who are already in a relationship (the orange ones in Figure 3.18b), we are left with the clear negative association that we see in Figure 3.18c. This phenomenon is known as *Berkson's paradox*. The fact that we see this negative association simply because we are selecting a biased subset of the general population to look at is why this is sometimes referred to as *selection bias* [see, e.g., 7, Chapter 8].

[7]: Hernán and Robins (2020), *Causal Inference: What If*



(a) Looks and kindness data for the whole population. Looks and kindness are independent.

(b) Looks and kindness data grouped by whether the person is available or not. Within each group, there is a negative correlation.

(c) Looks and kindness data for only the available people. Now, there is a negative correlation.

Figure 3.18: Example data for the "good-looking men are jerks" example. Both looks and kindness are continuous values on a scale from 0 to 10.

Numerical Example All of the above has been to give you intuition about why conditioning on a collider induces association between its parents, but we have yet to give a concrete numerical example of this. We will give a simple one here. Consider the following *data generating*

process (DGP), where X_1 and X_3 are drawn independently from standard normal distributions and then used to compute X_2:

$$X_1 \sim N(0,1)\,, \quad X_3 \sim N(0,1) \tag{3.14}$$
$$X_2 = X_1 + X_3 \tag{3.15}$$

We've already stated that X_1 and X_3 are independent, but to juxtapose the two calculations, let's compute their covariance:

$$\begin{aligned}\text{Cov}(X_1, X_3) &= \mathbb{E}[(X_1 - \mathbb{E}[X_1])(X_3 - \mathbb{E}[X_3])] \\ &= \mathbb{E}[X_1 X_3] && \text{(zero mean)} \\ &= \mathbb{E}[X_1]\mathbb{E}[X_3] && \text{(independent)} \\ &= 0\end{aligned}$$

Now, let's compute their covariance, conditional on X_2:

$$\text{Cov}(X_1, X_3 \mid X_2 = x) = \mathbb{E}[X_1 X_3 \mid X_2 = x] \tag{3.16}$$
$$= \mathbb{E}[X_1(x - X_1)] \tag{3.17}$$
$$= x\mathbb{E}[X_1] - \mathbb{E}[X_1^2] \tag{3.18}$$
$$= -1 \tag{3.19}$$

Crucially, in Equation 3.17, we used Equation 3.15 to plug in for X_3 in terms of X_1 and X_2 (conditioned to x). This led to a second-order term, which led to the calculation giving a nonzero number, which means X_1 and X_3 are associated, conditional on X_2.

Descendants of Colliders Conditioning on descendants of a collider also induces association in between the parents of the collider. The intuition is that if we learn something about a collider's descendant, we usually also learn something about the collider itself because there is a direct causal path from the collider to its descendants, and we know that nodes in a chain are usually associated (see Section 3.5), assuming minimality (Assumption 3.2). In other words, a descendant of a collider can be thought of as a proxy for that collider, so conditioning on the descendant is similar to conditioning on the collider itself.

Active reading exercise: We have provided several techniques for how to think about colliders: high-level examples, numerical examples, and abstract reasoning. Use at least one of them to convince yourself that conditioning on a descendant of a collider can induce association between the collider's parents.

3.7 d-separation

Before we define d-separation, we'll codify what we mean by the concept of a "blocked path," which we've been discussing in the previous sections:

Definition 3.3 (blocked path) *A path between nodes X and Y is blocked by a (potentially empty) conditioning set Z if either of the following is true:*

1. *Along the path, there is a chain $\cdots \to W \to \cdots$ or a fork $\cdots \leftarrow W \to \cdots$, where W is conditioned on ($W \in Z$).*
2. *There is a collider W on the path that is not conditioned on ($W \notin Z$) and none of its descendants are conditioned on* ($\text{de}(W) \nsubseteq Z$).

Then, an *unblocked path* is simply the complement; an unblocked path is a

path that is not blocked. The graphical intuition to have in mind is that association flows along unblocked paths, and association does not flow along blocked paths. If you don't have this intuition in mind, then it is probably worth it to reread the previous two sections, with the goal of gaining this intuition. Now, we are ready to introduce a very important concept: *d-separation*.

Definition 3.4 (d-separation) *Two (sets of) nodes X and Y are d-separated by a set of nodes Z if all of the paths between (any node in) X and (any node in) Y are blocked by Z [16].*

[16]: Pearl (1988), *Probabilistic Reasoning in Intelligent Systems: Networks of Plausible Inference*

If all the paths between two nodes X and Y are blocked, then we say that X and Y are *d-separated*. Similarly, if there exists at least one path between X and Y that is unblocked, then we say that X and Y are *d-connected*.

As we will see in Theorem 3.1, d-separation is such an important concept because it implies conditional independence. We will use the notation $X \perp\!\!\!\perp_G Y \mid Z$ to denote that X and Y are d-separated in the graph G when conditioning on Z. Similarly, we will use the notation $X \perp\!\!\!\perp_P Y \mid Z$ to denote that X and Y are independent in the distribution P when conditioning on Z.

Theorem 3.1 *Given that P is Markov with respect to G (satisfies the local Markov assumption, Assumption 3.1), if X and Y are d-separated in G conditioned on Z, then X and Y are independent in P conditioned on Z. We can write this succinctly as follows:*

$$X \perp\!\!\!\perp_G Y \mid Z \implies X \perp\!\!\!\perp_P Y \mid Z \tag{3.20}$$

Because this is so important, we will give Equation 3.20 a name: the *global Markov assumption*. Theorem 3.1 tells us that the local Markov assumption implies the global Markov assumption.

Just as we built up the intuition that suggested that the local Markov assumption (Assumption 3.1) implies the Bayesian network factorization (Definition 3.1) and alerted you to the fact that the Bayesian network factorization also implies the local Markov assumption (the two are equivalent), it turns out that the global Markov assumption also implies the local Markov assumption. In other words, the local Markov assumption, global Markov assumption, and the Bayesian network factorization are all equivalent [see, e.g., 13, Chapter 3]. Therefore, we will use the slightly shortened phrase **Markov assumption** to refer to these concepts as a group, or we will simply write "P is Markov with respect to G" to convey the same meaning.

[13]: Koller and Friedman (2009), *Probabilistic Graphical Models: Principles and Techniques*

Active reading exercise: To get some practice with d-separation, here are some questions about d-separation in Figure 3.19.

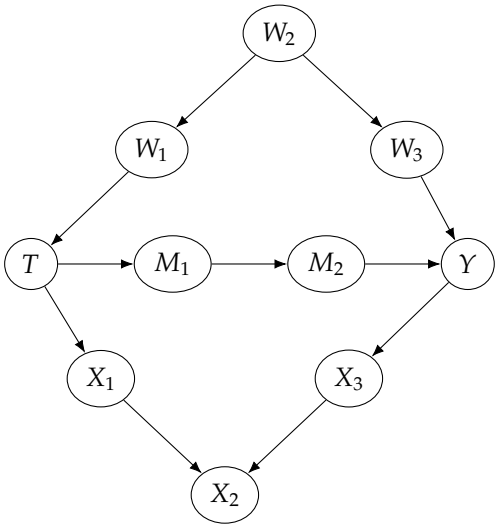
Questions about Figure 3.19a:

1. Are T and Y d-separated by the empty set?
2. Are T and Y d-separated by W_2?
3. Are T and Y d-separated by $\{W_2, M_1\}$?
4. Are T and Y d-separated by $\{W_1, M_2\}$?
5. Are T and Y d-separated by $\{W_1, M_2, X_2\}$?

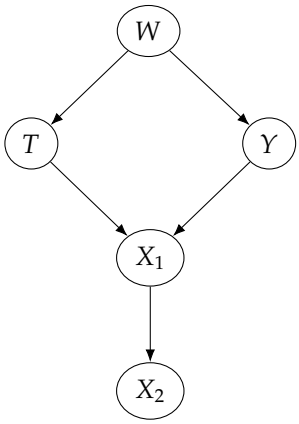
6. Are T and Y d-separated by $\{W_1, M_2, X_2, X_3\}$?

Questions about Figure 3.19b:

1. Are T and Y d-separated by the empty set?
2. Are T and Y d-separated by W?
3. Are T and Y d-separated by $\{W, X_2\}$?



(a)



(b)

Figure 3.19: Graphs for d-separation exercise

3.8 Flow of Association and Causation

Now that we have covered the necessary preliminaries (chains, forks, colliders, and d-separation), it is worth emphasizing how association and causation flow in directed graphs. Association flows along all unblocked paths. In causal graphs, causation flows along directed paths. Recall from Section 1.3.2 that not only is association not causation, but causation is a sub-category of association. That's why association and causation both flow along directed paths.

We refer to the flow of association along directed paths as *causal association*. A common type of non-causal association that makes total association not causation is *confounding association*. In the graph in Figure 3.20, we depict the confounding association in red and the causal association in blue.

Regular Bayesian networks are purely statistical models, so we can only talk about the flow of association in Bayesian networks. Association still flows in exactly the same way in Bayesian networks as it does in causal graphs, though. In both, association flows along chains and forks, unless a node is conditioned on. And in both, a collider blocks the flow of association, unless it is conditioned on. Combining these building blocks, we get how association flows in general DAGs. We can tell if two nodes are not associated (no association flows between them) by whether or not they are d-separated.

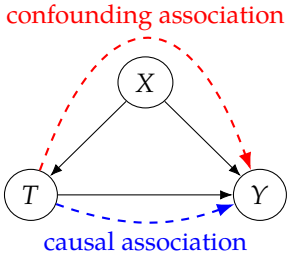


Figure 3.20: Causal graph depicting an example of how confounding association and causal association flow.

Causal graphs are special in that we additionally assume that the edges have causal meaning (causal edges assumption, Assumption 3.3). This assumption is what introduces causality into our models, and it makes one type of path take on a whole new meaning: directed paths. This assumption endows directed paths with the unique role of carrying causation along them. Additionally, this assumption is asymmetric; "X is a cause of Y" is not the same as saying "Y is a cause of X." This means that there is an important difference between association and causation: association is symmetric, whereas causation is asymmetric.

d-separation Implies Association is Causation Given that we have tools to measure association, how can we isolate causation? In other words, how can we ensure that the association we measure is causation, say, for measuring the causal effect of X on Y? Well, we can do that by ensuring that there is no non-causal association flowing between X and Y. This is true if X and Y are d-separated in the augmented graph where we remove outgoing edges from X. This is because all of X's causal effect on Y would flow through it's outgoing edges, so once those are removed, the only association that remains is purely non-causal association.

In Figure 3.21, we illustrate what each of the important assumptions gives us in terms of interpreting this flow of association. First, we have the (local/global) Markov assumption (Assumption 3.1). As we saw in Section 3.7, this assumption allows us to know which nodes are unassociated. In other words, the Markov assumption tells along which paths the association does *not* flow. When we slightly strengthen the Markov assumption to the minimality assumption (Assumption 3.2), we get which paths association *does* flow along (except in intransitive edges cases). When we further add in the causal edges assumption (Assumption 3.3), we get that causation flows along directed paths. Therefore, the following two[8] assumptions are essential for graphical causal models:

1. Markov Assumption (Assumption 3.1)
2. Causal Edges Assumption (Assumption 3.3)

[8] Recall that the first part of the minimality assumption is just the local Markov assumption and that the second part is contained in the causal edges assumption.

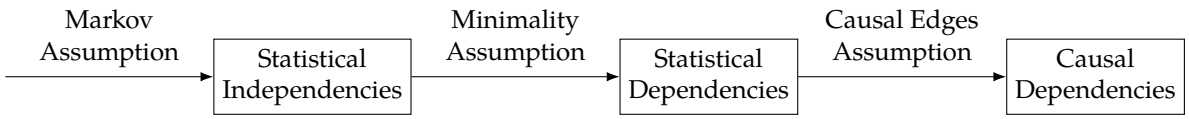


Figure 3.21: A flowchart that illustrates what kind of claims we can make about our data as we add each additional important assumption.

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Causal models are essential for identification of causal quantities. When we presented the Identification-Estimation Flowchart (Figure 2.5) back in Section 2.4, we described identification as the process of moving from a causal estimand to a statistical estimand. However, to do that, we must have a causal model. We depict this more full version of the Identification-Estimation Flowchart in Figure 4.1.

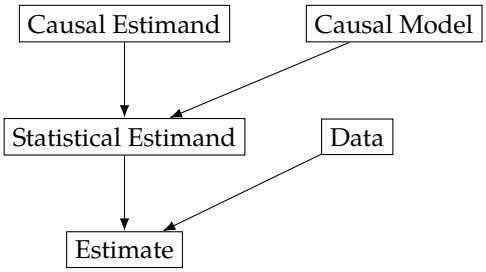


Figure 4.1: The Identification-Estimation Flowchart – a flowchart that illustrates the process of moving from a target causal estimand to a corresponding estimate, through identification and estimation. In contrast to Figure 2.5, this version is augmented with a causal model and data.

The previous chapter gives graphical intuition for causal models, but it doesn't explain how to identify causal quantities and formalize causal models. We will do that in this chapter.

4.1 The *do*-operator and Interventional Distributions

The first thing that we will introduce is a mathematical operator for intervention. In the regular notation for probability, we have conditioning, but that isn't the same as intervening. Conditioning on $T = t$ just means that we are restricting our focus to the subset of the population to those who received treatment t. In contrast, an intervention would be to take the whole population and give everyone treatment t. We illustrate this in Figure 4.2. We will denote intervention with the *do*-operator: $do(T = t)$. This is the notation commonly used in graphical causal models, and it has equivalents in potential outcomes notation. For example, we can write the distribution of the potential outcome $Y(t)$ that we saw in Chapter 2 as follows:

$$P(Y(t) = y) \triangleq P(Y = y \mid do(T = t)) \triangleq P(y \mid do(t)) \tag{4.1}$$

Note that we shorten $do(T = t)$ to just $do(t)$ in the last option in Equation 4.1. We will use this shorthand throughout the book. We can similarly write the ATE (average treatment effect) when the treatment is binary as follows:

$$\mathbb{E}[Y \mid do(T = 1)] - \mathbb{E}[Y \mid do(T = 0)] \tag{4.2}$$

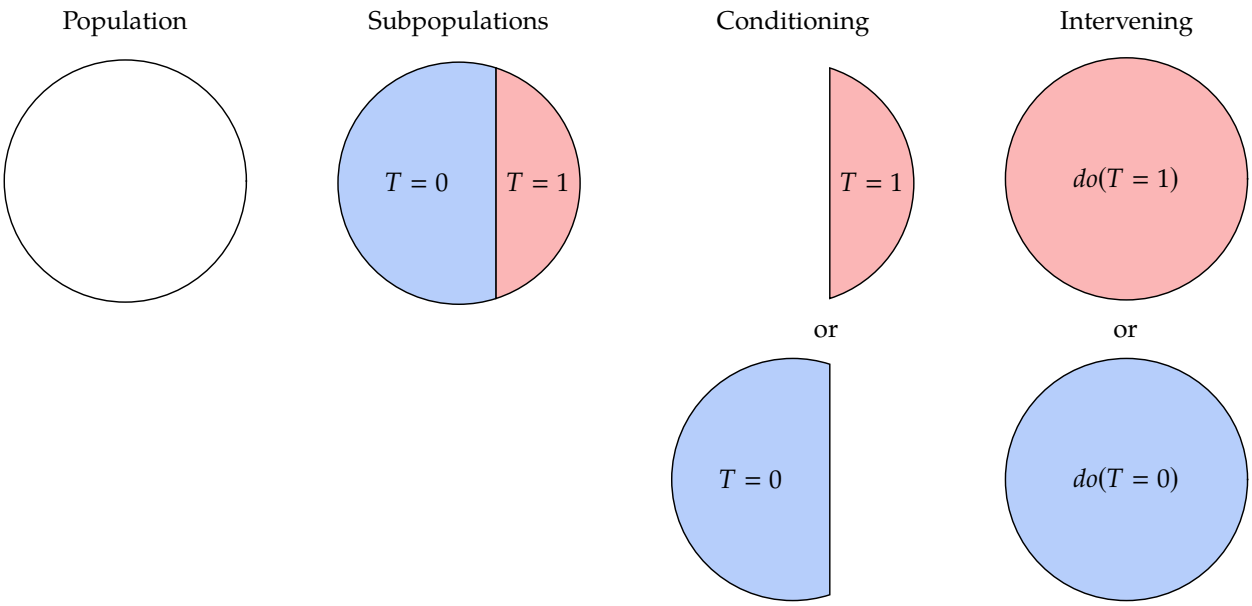


Figure 4.2: Illustration of the difference between conditioning and intervening

We will often work with full distributions like $P(Y \mid do(t))$, rather than their means, as this is more general; if we characterize $P(Y \mid do(t))$, then we've characterized $\mathbb{E}[Y \mid do(t)]$. We will commonly refer to $P(Y \mid do(T = t))$ and other expressions with the *do*-operator in them as *interventional distributions*.

Interventional distributions such as $P(Y \mid do(T = t))$ are conceptually quite different from the *observational distribution* $P(Y)$. Observational distributions such as $P(Y)$ or $P(Y, T, X)$ do not have the *do*-operator in them. Because they don't have the *do*-operator, we can observe data from them without needing to carry out any experiment. This is why we call data from $P(Y, T, X)$ *observational data*. If we can reduce an expression Q with *do* in it (an interventional expression) to one without *do* in it (an observational expression), then Q is said to be *identifiable*. An expression with a *do* in it is fundamentally different from an expression without a *do* in it, despite the fact that in *do*-notation, *do* appears after a regular conditioning bar. As we discussed in Section 2.4, we will refer to an estimand as a *causal estimand* when it contains a *do*-operator, and we refer to an estimand as a *statistical estimand* when it doesn't contain a *do*-operator.

Whenever, $do(t)$ appears after the conditioning bar, it means that everything in that expression is in the *post-intervention* world where the intervention $do(t)$ occurs. For example, $\mathbb{E}[Y \mid do(t), Z = z]$ refers to the expected outcome in the subpopulation where $Z = z$ after the whole subpopulation has taken treatment t. In contrast, $\mathbb{E}[Y \mid Z = z]$ simply refers to the expected value in the (*pre-intervention*) population where individuals take whatever treatment they would normally take (T). This distinction will become important when we get to counterfactuals in Chapter 11.

4.2 The Main Assumption: Modularity

Before we can describe a very important assumption, we must specify what a *causal mechanism* is. There are a few different ways to think about causal mechanisms. In this section, we will refer to the causal mechanism that generates X_i as the conditional distribution of X_i given all of its causes: $P(x_i \mid \text{pa}_i)$. As we show graphically in Figure 4.3, the causal mechanism that generates X_i is all of X_i's parents and their edges that go into X_i. We will give a slightly more specific description of what a causal mechanism is in Section 4.5.1, but these suffice for now.

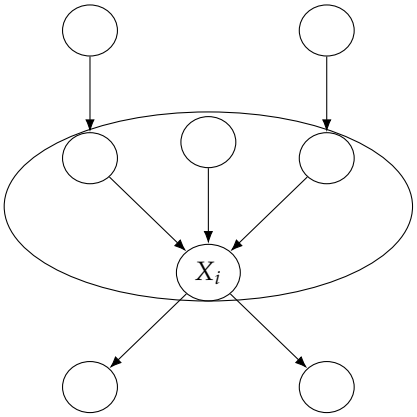


Figure 4.3: A causal graph with the causal mechanism that generates X_i depicted inside an ellipse.

In order to get many causal identification results, the main assumption we will make is that interventions are local. More specifically, we will assume that intervening on a variable X_i only changes the causal mechanism for X_i; it does not change the causal mechanisms that generate any other variables. In this sense, the causal mechanisms are *modular*. Other names that are used for the modularity property are *independent mechanisms*, *autonomy*, and *invariance*. We will now state this assumption more formally.

Assumption 4.1 (Modularity / Independent Mechanisms / Invariance) *If we intervene on a set of nodes $S \subseteq [n]$,[1] setting them to constants, then for all i, we have the following:*

1. *If $i \notin S$, then $P(x_i \mid \text{pa}_i)$ remains unchanged.*
2. *If $i \in S$, then $P(x_i \mid \text{pa}_i) = 1$ if x_i is the value that X_i was set to by the intervention; otherwise, $P(x_i \mid \text{pa}_i) = 0$.*

[1] We use $[n]$ to refer to the set $\{1, 2, \ldots, n\}$.

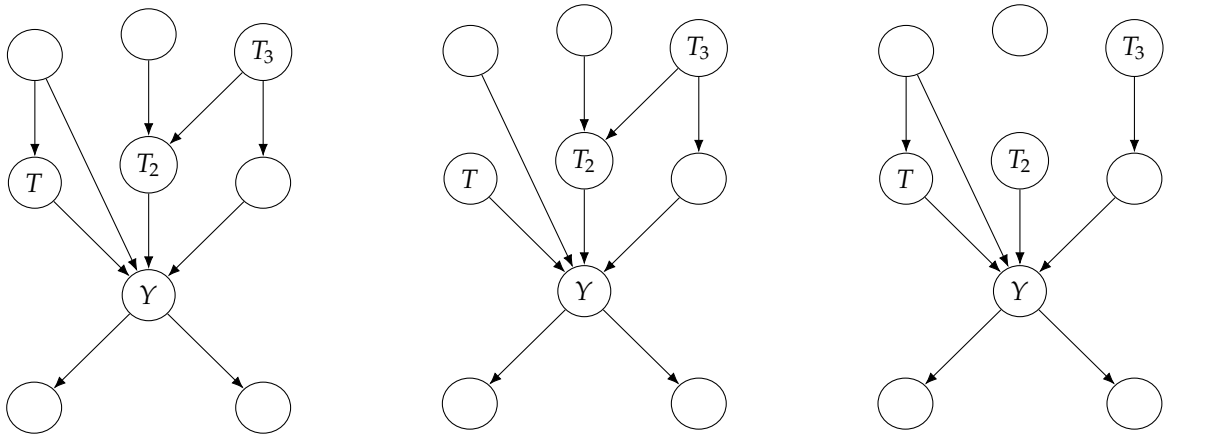
In the second part of the above assumption, we could have alternatively said $P(x_i \mid \text{pa}_i) = 1$ if x_i is *consistent with the intervention*[2] and 0 otherwise. More explicitly, we will say (in the future) that if $i \in S$, a value x_i is consistent with the intervention if x_i equals the value that X_i was set to in the intervention.

[2] Yes, the word "consistent" is extremely overloaded.

The modularity assumption is what allows us to encode many different interventional distributions all in a single graph. For example, it could be the case that $P(Y)$, $P(Y \mid do(T = t))$, $P(Y \mid do(T = t'))$, and $P(Y \mid do(T_2 = t_2))$ are all completely different distributions that share almost nothing. If this were the case, then each of these distributions would need their own graph. However, by assuming modularity, we can encode them all with the same graph that we use to encode the joint $P(Y, T, T_2, \ldots)$, and we can know that all of the factors (except ones that are intervened on) are shared across these graphs.

The causal graph for interventional distributions is simply the same graph that was used for the observational joint distribution, but with all of the edges to the intervened node(s) removed. This is because the probability for the intervened factor has been set to 1, so we can just ignore that factor (this is the focus of the next section). Another way to see that the intervened node has no causal parents is that the intervened node is set to a constant value, so it no longer depends on any of the variables it depends on in the observational setting (its parents). The graph with edges removed is known as the *manipulated graph*.

For example, consider the causal graph for an observational distribution in Figure 4.4a. Both $P(Y \mid do(T = t))$ and $P(Y \mid do(T = t'))$ correspond to the causal graph in Figure 4.4b, where the incoming edge to T has been removed. Similarly, $P(Y \mid do(T_2 = t_2))$ corresponds to the graph in Figure 4.4c, where the incoming edges to T_2 have been removed. Although it is not expressed in the graphs (which only express conditional independencies and causal relations), under the modularity assumption, $P(Y)$, $P(Y \mid T = t')$, and $P(Y \mid do(T_2 = t_2))$ all shared the exact same factors (that are not intervened on).



(a) Causal graph for observational distribution

(b) Causal graph after intervention on T (interventional distribution)

(c) Causal graph after intervention on T_2 (interventional distribution)

Figure 4.4: Intervention as edge deletion in causal graphs

What would it mean for the modularity assumption to be violated? Imagine that you intervene on X_i, and this causes the mechanism that generates a different node X_j to change; an intervention on X_i changes $P(x_j \mid \text{pa}_j)$, where $j \neq i$. In other words, the intervention is not local to the node you intervene on; causal mechanisms are not invariant to when you change other causal mechanisms; the causal mechanisms are not modular.

This assumption is so important that Judea Pearl refers to a closely related version (which we will see in Section 4.5.2) as **The Law of Counterfactuals (and Interventions)**, one of two key principles from which all other causal results follow.[3] Incidentally, taking the modularity assumption (Assumption 4.1) and the Markov assumption (the other key principle) together gives us *causal Bayesian networks*. We'll now move to one of the important results that follow from these assumptions.

[3] The other key principle is the global Markov assumption (Theorem 3.1), which is the assumption that d-separation implies conditional independence.

4.3 Truncated Factorization

Recall the Bayesian network factorization (Definition 3.1), which tells us that if P is Markov with respect to a graph G, then P factorizes as follows:

$$P(x_1, \ldots, x_n) = \prod_i P(x_i \mid \text{pa}_i) \tag{4.3}$$

where pa_i denotes the parents of X_i in G. Now, if we intervene on some set of nodes S and assume modularity (Assumption 4.1), then all of the factors should remain the same except the factors for $X_i \in S$; those factors

should change to 1 (for values consistent with the intervention) because those variables have been intervened on. This is how we get the *truncated factorization*.

Proposition 4.1 (Truncated Factorization) *We assume that P and G satisfy the Markov assumption and modularity. Given, a set of intervention nodes S, if x is consistent with the intervention, then*

$$P(x_1, \ldots, x_n \mid do(S = s)) = \prod_{i \notin S} P(x_i \mid \text{pa}_i)\,. \tag{4.4}$$

Otherwise, $P(x_1, \ldots, x_n \mid do(S = s)) = 0$.

The key thing that changed when we moved from the regular factorization in Equation 4.3 to the truncated factorization in Equation 4.4 is that the latter's product is only over $i \notin S$ rather than all i. In other words, the factors for $i \in S$ have been truncated.

4.3.1 Example Application and Revisiting "Association is Not Causation"

To see the power that the truncated factorization gives us, let's apply it to identify the causal effect of treatment on outcome in a simple graph. Specifically, we will identify the causal quantity $P(y \mid do(t))$. In this example, the distribution P is Markov with respect to the graph in Figure 4.5. The Bayesian network factorization (from the Markov assumption), gives us the following:

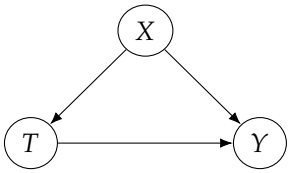


Figure 4.5: Simple causal structure where X counfounds the effect of T on Y and where X is the only confounder.

$$P(y, t, x) = P(x)\,P(t \mid x)\,P(y \mid t, x) \tag{4.5}$$

When we intervene on the treatment, the truncated factorization (from adding the modularity assumption) gives us the following:

$$P(y, x \mid do(t)) = P(x)\,P(y \mid t, x) \tag{4.6}$$

Then, we simply need to marginalize out x to get what we want:

$$P(y \mid do(t)) = \sum_x P(y \mid t, x)\,P(x) \tag{4.7}$$

We assumed X is discrete when we summed over its values, but we can simply replace the sum with an integral if X is continuous. Throughout this book, that will be the case, so we usually won't point it out.

If we massage Equation 4.7 a bit, we can clearly see how association is not causation. The purely associational counterpart of $P(y \mid do(t))$ is $P(y \mid t)$. If the $P(x)$ in Equation 4.7 were $P(x \mid t)$, then we would actually recover $P(y \mid t)$. We briefly show this:

$$\sum_x P(y \mid t, x)\,P(x \mid t) = \sum_x P(y, x \mid t) \tag{4.8}$$

$$= P(y \mid t) \tag{4.9}$$

This gives some concreteness to the difference between association and causation. In this example (which is representative of a broader

phenomenon), the difference between $P(y \mid do(t))$ and $P(y \mid t)$ is the difference between $P(x)$ and $P(x \mid t)$.

To round this example out, say T is a binary random variable, and we want to compute the ATE. $P(y \mid do(T = 1))$ is the distribution for $Y(1)$, so we can just take the expectation to get $\mathbb{E}[Y(1)]$. Similarly, we can do the same thing with $Y(0)$. Then, we can write the ATE as follows:

$$\mathbb{E}[Y(1) - Y(0)] = \sum_y y\, P(y \mid do(T = 1)) - \sum_y y\, P(y \mid do(T = 0)) \quad (4.10)$$

If we then plug in Equation 4.7 for $P(y \mid do(T = 1))$ and $P(y \mid do(T = 0))$, we have a fully identified ATE. Given the simple graph in Figure 4.5, we have shown how we can use the truncated factorization to identify causal effects in Equations 4.5 to 4.7. We will now generalize this identification process to a more general formula.

4.4 The Backdoor Adjustment

Recall from Chapter 3 that causal association flows from T to Y along directed paths and that non-causal association flows along any other paths from T to Y that aren't blocked by either 1) a non-collider that is conditioned on or 2) a collider that isn't conditioned on. These non-directed unblocked paths from T to Y are known as *backdoor paths* because they have an edge that goes in the "backdoor" of the T node. And it turns out that if we can block these paths by conditioning, we can identify causal quantities like $P(Y \mid do(t))$.[4]

This is precisely what we did in the previous section. We blocked the backdoor path $T \leftarrow X \rightarrow Y$ in Figure 4.5 simple by conditioning on X and marginalizing it out (Equation 4.7). In this section, we will generalize Equation 4.7 to arbitrary DAGs. But before we do that, let's graphically consider why the quantity $P(y \mid do(t))$ is purely causal.

As we discussed in Section 4.2, the graph for the interventional distribution $P(Y \mid do(t))$ is the same as the graph for the observational distribution $P(Y, T, X)$, but with the incoming edges to T removed. For example, if we take the graph from Figure 4.5 and intervene on T, then we get the manipulated graph in Figure 4.6. In this manipulated graph, there cannot be any backdoor paths because no edges are going into the backdoor of T. Therefore, all of the association that flows from T to Y in the manipulated graph is purely causal.

With that digression aside, let's prove that we can identify $P(y \mid do(t))$. We want to turn the causal estimand $P(y \mid do(t))$ into a statistical estimand (only relies on the observational distribution). We'll start with assuming we have a set of variables W that satisfy the backdoor criterion:

Definition 4.1 (Backdoor Criterion) *A set of variables W satisfies the backdoor criterion relative to T and Y if the following are true:*

1. *W blocks all backdoor paths from T to Y.*
2. *W does not contain any descendants of T.*

[4] As we mentioned in Section 3.8, blocking all backdoor paths is equivalent to having d-separation in the graph where edges going out of T are removed. This is because these are the only edges that causation flows along, so once they are removed, all that remains is non-causation association.

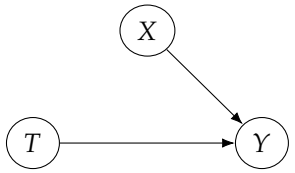


Figure 4.6: Manipulated graph that results from intervening on T, when the original graph is Figure 4.5.

[5] Active reading exercise: In a general DAG, which set of nodes related to T will always be a sufficient adjustment set? Which set of nodes related to Y will always be a sufficient adjustment set?

Satisfying the backdoor criterion makes W a *sufficient adjustment set*.[5] We saw an example of X as a sufficient adjustment set in Section 4.3.1. Because there was only a single backdoor path in Section 4.3.1, a single node (X) was enough to block all backdoor paths, but, in general, there can be multiple backdoor paths.

To introduce W into the proof, we'll use the usual trick of conditioning on variables and marginalizing them out:

$$P(y \mid do(t)) = \sum_w P(y \mid do(t), w)\, P(w \mid do(t)) \tag{4.11}$$

Given that W satisfies the backdoor criterion, we can write the following:

$$\sum_w P(y \mid do(t), w)\, P(w \mid do(t)) = \sum_w P(y \mid t, w)\, P(w \mid do(t)) \tag{4.12}$$

This follows from the modularity assumption (Assumption 4.1). If W is all of the parents for Y (other than T), it should be clear that the modularity assumption immediately implies $P(y \mid do(t), w) = P(y \mid t, w)$. If W isn't the parents of Y but still blocks all backdoor paths another way, then this equality is still true but requires using the graphical knowledge we built up in Chapter 3.

In the manipulated graph (for $P(y \mid do(t), w)$), all of the T-Y association flows along the directed path(s) from T to Y, since there cannot be any backdoor paths because T has no incoming edges. Similarly, in the regular graph (for $P(y \mid t, w)$), all of the T-Y association flows along the directed path(s) from T to Y. This is because, even though there exist backdoor paths, the association that would flow along them is blocked by W, leaving association to only flow along directed paths. In both cases, association flows along the exact same directed paths, which correspond to the exact same conditional distributions (by the modularity assumption).

Although we've justified Equation 4.12, there is still a *do* in the expression: $P(w \mid do(t))$. However, $P(w \mid do(t)) = P(w)$. To see this, consider how T might have influence W in the manipulated graph. It can't be through any path that has an edge into T because T doesn't have any incoming edges in the manipulated graph. It can't be through any path that has an edge going out of T because such a path would have to have a collider, that isn't conditioned on, on the path. We know any such colliders are not conditioned on because we have assumed that W does not contain descendants of T (second part of the backdoor criterion).[6] Therefore, we can write the final step:

$$\sum_w P(y \mid t, w)\, P(w \mid do(t)) = \sum_w P(y \mid t, w)\, P(w) \tag{4.13}$$

This is known as the *backdoor adjustment*.

Theorem 4.2 (Backdoor Adjustment) *Given the modularity assumption (Assumption 4.1), that W satisfies the backdoor criterion (Definition 4.1), and*

[6] We will come back to what goes wrong if we condition on descendants of T in Section 4.5.3, after we cover some important concepts that we need before we can fully explain that.

positivity (Assumption 2.3), we can identify the causal effect of T on Y:

$$P(y \mid do(t)) = \sum_w P(y \mid t, w)\, P(w)$$

Here's a concise recap of the proof (Equations 4.11 to 4.13) without all of the explanation/justification:

Proof.

$$P(y \mid do(t)) = \sum_w P(y \mid do(t), w)\, P(w \mid do(t)) \tag{4.14}$$

$$= \sum_w P(y \mid t, w)\, P(w \mid do(t)) \tag{4.15}$$

$$= \sum_w P(y \mid t, w)\, P(w) \tag{4.16}$$

□

Relation to d-separation We can use the backdoor adjustment if W d-separates T from Y in the manipulated graph. Recall from Section 3.8 that we mentioned that we would be able to isolate the causal association if T is d-separated from Y in the manipulated graph. "Isolation of the causal association" is identification. We can also isolate the causal association if Y is d-separated from T in the manipulated graph, *conditional on* W. This is what the first part of the backdoor criterion is about and what we've codified in the backdoor adjustment.

4.4.1 Relation to Potential Outcomes

Hmm, the backdoor adjustment (Theorem 4.2) looks quite similar to the adjustment formula (Theorem 2.1) that we saw back in the potential outcomes chapter:

$$\mathbb{E}[Y(1) - Y(0)] = \mathbb{E}_W\left[\mathbb{E}[Y \mid T = 1,\, W] - \mathbb{E}[Y \mid T = 0,\, W]\right] \tag{4.17}$$

We can derive this from the more general backdoor adjustment in a few steps. First, we take an expectation over Y:

$$\mathbb{E}[Y \mid do(t)] = \sum_w \mathbb{E}[Y \mid t, w]\, P(w) \tag{4.18}$$

Then, we notice that the sum over w and $P(w)$ is an expectation (for discrete w, but just replace with an integral if not):

$$\mathbb{E}[Y \mid do(t)] = \mathbb{E}_W \mathbb{E}[Y \mid t, W] \tag{4.19}$$

And finally, we look at the difference between $T = 1$ and $T = 0$:

$$\mathbb{E}[Y \mid do(T = 1)] - \mathbb{E}[Y \mid do(T = 0)] = \mathbb{E}_W\left[\mathbb{E}[Y \mid T = 1, W] - \mathbb{E}[Y \mid T = 0, W]\right] \tag{4.20}$$

Since the *do*-notation $\mathbb{E}[Y \mid do(t)]$ is just another notation for the potential outcomes $\mathbb{E}[Y(t)]$, we are done! If you remember, one of the main assumptions we needed to get Equation 4.17 (Theorem 2.1) was conditional

exchangeability (Assumption 2.2), which we repeat below:

$$(Y(1), Y(0)) \perp\!\!\!\perp T \mid W \tag{4.21}$$

However, we had no way of knowing how to choose W or knowing that that W actually gives us conditional exchangeability. Well, using graphical causal models, we know how to choose a valid W: we simply choose W so that it satisfies the backdoor criterion. Then, under the assumptions encoded in the causal graph, conditional exchangeability provably holds; the causal effect is provably identifiable.

4.5 Structural Causal Models (SCMs)

Graphical causal models such as causal Bayesian networks give us powerful ways to encode statistical and causal assumptions, but we have yet to explain exactly what an intervention is or exactly what a causal mechanism is. Moving from causal Bayesian networks to full structural causal models will give us this additional clarity along with the power to compute counterfactuals.

4.5.1 Structural Equations

As Judea Pearl often says, the equals sign in mathematics does not convey any causal information. Saying $A = B$ is the same as saying $B = A$. Equality is symmetric. However, in order to talk about causation, we must have something asymmetric. We need to be able to write that A is a cause of B, meaning that changing A results in changes in B, but changing B does not result in changes in A. This is what we get when we write the following *structural equation*:

$$B := f(A)\,, \tag{4.22}$$

where f is some function that maps A to B. While the usual "=" symbol does not give us causal information, this new ":=" symbol does. This is a major difference that we see when moving from statistical models to causal models. Now, we have the asymmetry we need to describe causal relations. However, the mapping between A and B is deterministic. Ideally, we'd like to allow it to be probabilistic, which allows room for some unknown causes of B that factor into this mapping. Then, we can write the following:

$$B := f(A, U)\,, \tag{4.23}$$

where U is some unobserved random variable. We depict this in Figure 4.7, where U is drawn inside a dashed node to indicate that it is unobserved. The unobserved U is analogous to the randomness that we would see by sampling units (individuals); it denotes all the relevant (noisy) background conditions that determine B. More concretely, there are analogs to every part of the potential outcome $Y_i(t)$: B is the analog of Y, $A = a$ is the analog of $T = t$, and U is the analog of i.

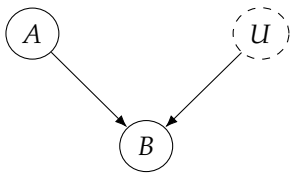


Figure 4.7: Graph for simple structural equation. The dashed node U means that U is unobserved.

The functional form of f does not need to be specified, and when left unspecified, we are in the *nonparametric* regime because we aren't making any assumptions about parametric form. Although the mapping

is deterministic, because it takes a random variable U (a "noise" or "background conditions" variable) as input, it can represent any stochastic mapping, so structural equations generalize the probabilistic factors $P(x_i \mid \text{pa}_i)$ that we've been using throughout this chapter. Therefore, all the results that we've seen such as the truncated factorization and the backdoor adjustment still hold when we introduce structural equations.

Cause and Causal Mechanism Revisited We have now come to the more precise definitions of what a cause is (Definition 3.2) and what a causal mechanism is (introduced in Section 4.2). A causal mechanism that generates a variable is the structural equation that corresponds to that variable. For example, the causal mechanism for B is Equation 4.23. Similarly, X is a *direct cause* of Y if X appears on the right-hand side of the structural equation for Y. We say that X is a *cause* of Y if X is a direct cause of any of the causes of Y[7] or if X is a direct cause of Y.

[7] Trust me; the recursion ends. The base case was specified.

We only showed a single structural equation in Equation 4.23, but there can be a large collection of structural equations in a single model, which we will commonly label M. For example, we write structural equations for Figure 4.8 below:

$$M: \quad \begin{aligned} B &:= f_B(A, U_B) \\ C &:= f_C(A, B, U_C) \\ D &:= f_D(A, C, U_D) \end{aligned} \tag{4.24}$$

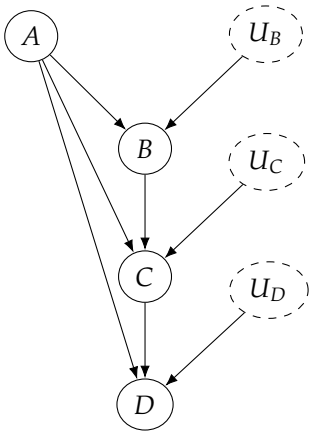


Figure 4.8: Graph for the structural equations in Equation 4.24.

In causal graphs, the noise variables are often implicit, rather than explicitly drawn. The variables that we write structural equations for are known as *endogenous* variables. These are the variables whose causal mechanisms we are modeling – the variables that have parents in the causal graph. In contrast, *exogenous* variables are variables who do not have any parents in the causal graph; these variables are external to our causal model in the sense that we choose not to model their causes. For example, in the causal model described by Figure 4.8 and Equation 4.24, the endogenous variables are $\{B, C, D\}$. And the exogenous variables are $\{A, U_B, U_C, U_D\}$.

Definition 4.2 (Structural Causal Model (SCM)) *A structural causal model is a tuple of the following sets:*

1. *A set of endogenous variables V*
2. *A set of exogenous variables U*
3. *A set of functions f, one to generate each endogenous variable as a function of other variables*

For example, M, the set of three equations above in Equation 4.24 constitutes an SCM with corresponding causal graph in Figure 4.8. Every SCM implies an associated causal graph: for each structural equation, draw an edge from every variable on the right-hand side to the variable on the left-hand side.

If the causal graph contains no cycles (is a DAG) and the noise variables U are independent, then the causal model is *Markovian*; the distribution P is Markov with respect to the causal graph. If the causal graph doesn't contain cycles but the noise terms are dependent, then the model is *semi-Markovian*. For example, if there is unobserved confounding, the model

is semi-Markovian. Finally, the graphs of *non-Markovian* models contain cycles. We will largely be considering Markovian and semi-Markovian models in this book.

4.5.2 Interventions

Interventions in SCMs are remarkably simple. The intervention $do(T = t)$ simply corresponds to replacing the structural equation for T with $T := t$. For example, consider the following causal model M with corresponding causal graph in Figure 4.9:

$$M : \begin{array}{l} T := f_T(X, U_T) \\ Y := f_Y(X, T, U_Y) \end{array} \tag{4.25}$$

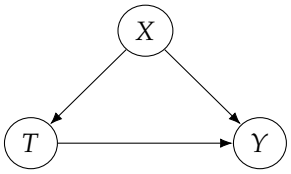


Figure 4.9: Basic causal graph

If we then intervene on T to set it to t, we get the *interventional SCM* M_t below and corresponding manipulated graph in Figure 4.10.

$$M_t : \begin{array}{l} T := t \\ Y := f_Y(X, T, U_Y) \end{array} \tag{4.26}$$

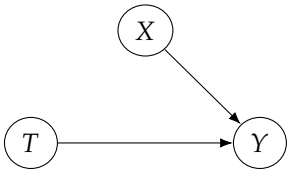


Figure 4.10: Basic causal with the the incoming edges to T removed, due to the intervention $do(T = t)$.

The fact that $do(T = t)$ only changes the equation for T and no other variables is a consequence of the modularity assumption; these causal mechanisms (structural equations) are modular. Assumption 4.1 states the modularity assumption in the context of causal Bayesian networks, but we need a slightly different translation of this assumption for SCMs.

Assumption 4.2 (Modularity Assumption for SCMs) *Consider an SCM M and an interventional SCM M_t that we get by performing the intervention $do(T = t)$. The modularity assumption states that M and M_t share all of their structural equations except the structural equation for T, which is $T := t$ in M_t.*

In other words, the intervention $do(T = t)$ is localized to T. None of the other structural equations change because they are modular; the causal mechanisms are independent. The modularity assumption for SCMs is what gives us what Pearl calls the **The Law of Counterfactuals**, which we briefly mentioned at the end of Section 4.2, after we defined the modularity assumption for causal Bayesian networks. But before we can get to that, we must first introduce a bit more notation.

In the causal inference literature, there are many different ways of writing the unit-level potential outcome. In Chapter 2, we used $Y_i(t)$. However, there are other ways such as Y_i^t or even $Y_t(u)$. For example, in his prominent potential outcomes paper, Holland [5] uses the $Y_t(u)$ notation. In this notation, u is the analog of i, just as we mentioned is the case for the U in Equation 4.23 and the paragraph that followed it. This is the notation that Pearl uses for SCMs as well [see, e.g., 17, Definition 4]. So $Y_t(u)$ denotes the outcome that unit u would observe if they take treatment t, given that the SCM is M. Similarly, we define $Y_{M_t}(u)$ as the outcome that unit u would observe if they take treatment t, given that the SCM is M_t (remember that M_t is the same SCM as M but with the structural equation for T changed to $T := t$). Now, we are ready to

[5]: Holland (1986), 'Statistics and Causal Inference'

[17]: Pearl (2009), 'Causal inference in statistics: An overview'

present one of Pearl's two key principles from which all other causal results follow:[8]

Definition 4.3 (The Law of Counterfactuals (and Interventions))

$$Y_t(u) = Y_{M_t}(u) \tag{4.27}$$

This is called "The Law of Counterfactuals" because it gives us information about counterfactuals. Given an SCM with enough details about it specified, we can actually compute counterfactuals. This is a big deal because this is exactly what the fundamental problem of causal inference (Section 2.2) told us we cannot do. We won't say more about how to do this until we get to the dedicated chapter for counterfactuals: Chapter 11.

[8] Active reading exercise: Can you recall which was the other key principle/assumption?

Active reading exercise: Take what you now know about structural equations, and relate it to other parts of this chapter. For example, how do interventions in structural equations relate to the modularity assumption? How does the modularity assumption for SCMs (Assumption 4.2) relate to the modularity assumption in causal Bayesian networks (Assumption 4.1)? Does this modularity assumption for SCMs still give us the backdoor adjustment?

4.5.3 Collider Bias and Why to Not Condition on Descendants of Treatment

In defining the backdoor criterion (Definition 4.1) for the backdoor adjustment (Theorem 4.2), not only did we specify that the adjustment set W blocks all backdoor paths, but we also specified that W does not contain any descendants of T. Why? There are two categories of things that could go wrong if we condition on descendants of T:

1. We block the flow of causation from T to Y.
2. We induce non-causal association between T and Y.

As we'll see, it is fairly intuitive why we want to avoid the first category. The second category is a bit more complex, and we'll break it up into two different parts, each with their own paragraph. This more complex part is actually why we delayed this explanation to after we introduced SCMs, rather than back when we introduced the backdoor criterion/adjustment in Section 4.4.

If we condition on a node that is on a directed path from T to Y, then we block the flow of causation along that causal path. We will refer to a node on a directed path from T to Y as a *mediator*, as it mediates the effect of T on Y. For example, in Figure 4.11, all of the causal flow is blocked by M. This means that we will measure zero association between T and Y (given that W blocks all backdoor paths). In Figure 4.12, only a portion of the causal flow is blocked by M. This is because causation can still flow along the $T \to Y$ edge. In this case, we will get a non-zero estimate of the causal effect, but it will still be biased, due to the causal flow that M blocks.

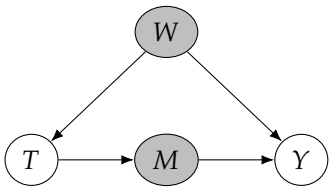


Figure 4.11: Causal graph where all causation is blocked by conditioning on M.

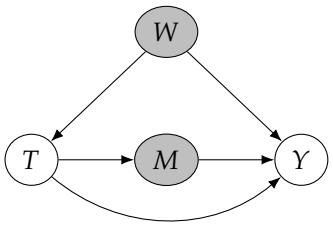


Figure 4.12: Causal graph where part of the causation is blocked by conditioning on M.

If we condition on a descendant of T that isn't a mediator, it could unblock a path from T to Y that was blocked by a collider. For example, this is the case with conditioning on Z in Figure 4.13. This induces non-causal association between T and Y, which biases the estimate of the causal effect. Consider the following general kind of path, where $\to \cdots \to$ denotes a directed path: $T \to \cdots \to Z \leftarrow \cdots \leftarrow Y$. Conditioning on Z, or any descendant of Z in a path like this, will induce *collider bias*. That is, the causal effect estimate will be biased by the non-causal association that we induce when we condition on Z or any of its descendants (see Section 3.6).

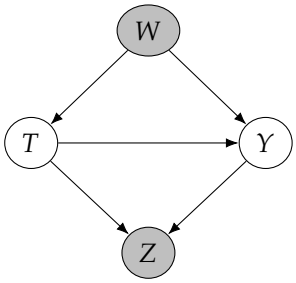


Figure 4.13: Causal graph where conditioning on the collider Z induces bias.

What about conditioning on Z in Figure 4.14? Would that induce bias? Recall that graphs are frequently drawn without explicitly drawing the noise variables. If we *magnify* part of the graph, making M's noise variable explicit, we get Figure 4.15. Now, we see that $T \to M \leftarrow U_M$ forms an immorality. Therefore, conditioning on Z induces an association between T and U_M. This induced non-causal association is another form of collider bias. You might find this unsatisfying because Y is not one of the immoral parents here; rather T and U_M are the ones living the immoral lifestyle. So why would this change the association between T and Y? One way to get the intuition for this is that there is now induced association flowing between T and U_M through the edge $T \to M$, which is also an edge that causal association is flowing along. You can think of these two types of association getting tangled up along the $T \to M$ edge, making the observed association between T and Y not purely causal. See Pearl [18, Section 11.3.1 and 11.3.3] for more information on this topic.

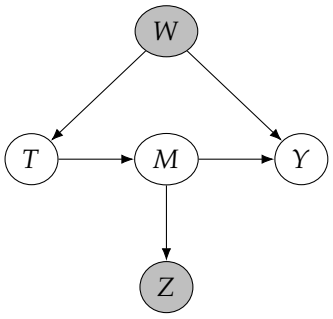


Figure 4.14: Causal graph where the child of a mediator is conditioned on.

Note that we actually can condition on some descendants of T without inducing non-causal associations between T and Y. For example, conditioning on descendants of T that aren't on any causal paths to Y won't induce bias. However, as you can see from the above paragraph, this can get a bit tricky, so it is safest to just not condition on any descendants of T, as the backdoor criterion prescribes. Even outside of graphical causal models (e.g. in potential outcomes literature), this rule is often applied; it is usually described as not conditioning on any *post-treatment covariates*.

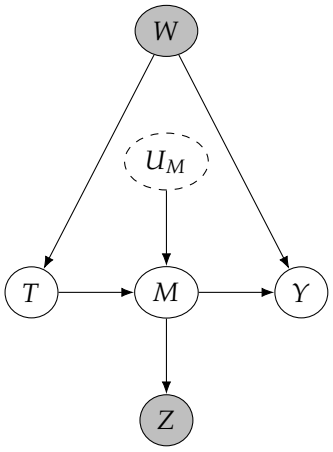


Figure 4.15: Magnified causal graph where the child of a mediator is conditioned on.

M-Bias Unfortunately, even if we only condition on pretreatment covariates, we can still induce collider bias. Consider what would happen if we condition on the collider Z_2 in Figure 4.16. Doing this opens up a backdoor path, along which non-causal association can flow. This is known as *M-bias* due to the M shape that this non-causal association flows along when the graph is drawn with children below their parents. For many examples of collider bias, see Elwert and Winship [19].

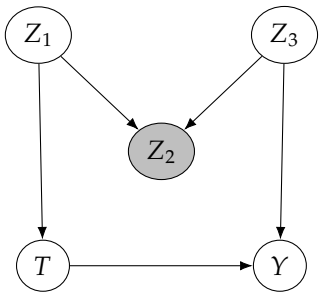


Figure 4.16: Causal graph depicting M-Bias.

4.6 Example Applications of the Backdoor Adjustment

4.6.1 Association vs. Causation in a Toy Example

In this section, we posit a toy generative process and derive the bias of the associational quantity $\mathbb{E}[Y \mid t]$. We compare this to the causal quantity $\mathbb{E}[Y \mid do(t)]$, which gives us exactly what we want. Note that both of these quantities are actually functions of t. If the treatment were binary, then we would just look at the difference between the quantities with $T = 1$ and with $T = 0$. However, because our generative processes will be linear, $\frac{d\,\mathbb{E}[Y|t]}{dt}$ and $\frac{d\,\mathbb{E}[Y|do(t)]}{dt}$ actually gives us all the information about the treatment effect, regardless of if treatment is continuous, binary, or multi-valued. We will assume infinite data so that we can work with expectations. This means this section has nothing to do with estimation; for estimation, see the next section

The generative process that we consider has the causal graph in Figure 4.17

and the following structural equations:

$$T := \alpha_1 X \tag{4.28}$$
$$Y := \beta T + \alpha_2 X \,. \tag{4.29}$$

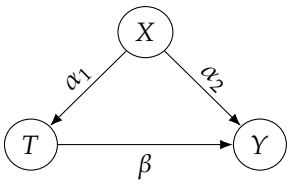


Figure 4.17: Causal graph for toy example

Note that in the structural equation for Y, β is the coefficient in front of T. This means that the causal effect of T on Y is β. Keep this in mind as we go through these calculations.

From the causal graph in Figure 4.17, we can see that X is a sufficient adjustment set. Therefore, $\mathbb{E}[Y \mid do(t)] = \mathbb{E}_X \mathbb{E}[Y \mid t, X]$. Let's calculate the value of this quantity in our example.

$$\mathbb{E}_X \mathbb{E}[Y \mid t, X] = \mathbb{E}_X \left[\mathbb{E}[\beta T + \alpha_2 X \mid T = t,\ X] \right] \tag{4.30}$$
$$= \mathbb{E}_X \left[\beta t + \alpha_2 X \right] \tag{4.31}$$
$$= \beta t + \alpha_2 \mathbb{E}[X] \tag{4.32}$$

Importantly, we made use of the equality that the structural equation for Y (Equation 4.29) gives us in Equation 4.30. Now, we just have to take the derivative to get the causal effect:

$$\frac{d\, \mathbb{E}_X \mathbb{E}[Y \mid t, X]}{dt} = \beta \,. \tag{4.33}$$

We got exactly what we were looking for. Now, let's move to the associational quantity:

$$\mathbb{E}[Y \mid T = t] = \mathbb{E}[\beta T + \alpha_2 X \mid T = t] \tag{4.34}$$
$$= \beta t + \alpha_2 \mathbb{E}[X \mid T = t] \tag{4.35}$$
$$= \beta t + \frac{\alpha_2}{\alpha_1} t \tag{4.36}$$

In Equation 4.36, we made use of the equality that the structural equation for T (Equation 4.28) gives us. If we then take the derivative, we see that there is confounding bias:

$$\frac{d\, \mathbb{E}[Y \mid t]}{dt} = \beta + \frac{\alpha_2}{\alpha_1} \,. \tag{4.37}$$

To recap, $\mathbb{E}_X \mathbb{E}[Y \mid t,\ X]$ gave us the causal effect we were looking for (Equation 4.33), whereas the associational quantity $\mathbb{E}[Y \mid t]$ did not (Equation 4.37). Now, let's go through an example that also takes into account estimation.

4.6.2 A Complete Example with Estimation

Recall that we estimated a concrete value for the causal effect of sodium intake on blood pressure in Section 2.5. There, we used the potential outcomes framework. Here, we will do the same thing, but using causal graphs. The spoiler is that the 19% error that we saw in Section 2.5 was due to conditioning on a collider.

First, we need to write down our causal assumptions in terms of a causal graph. Remember that in Luque-Fernandez et al. [8]'s example from epidemiology, the treatment T is sodium intake, and the outcome Y is

[8]: Luque-Fernandez et al. (2018), 'Educational Note: Paradoxical collider effect in the analysis of non-communicable disease epidemiological data: a reproducible illustration and web application'

blood pressure. The covariates are age W and amount of protein in urine (proteinuria) Z. Age is a common cause of both blood pressure and the body's ability to self-regulate sodium levels. In contrast, high amounts of urinary protein are caused by high blood pressure and high sodium intake. This means that proteinuria is a collider. We depict this causal graph in Figure 4.18.

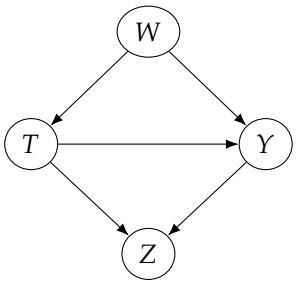


Figure 4.18: Causal graph for the blood pressure example. T is sodium intake. Y is blood pressure. W is age. And, importantly, the amount of protein excreted in urine Z is a collider.

Because Z is a collider, conditioning on it induces bias. Because W and Z were grouped together as "covariates" X in Section 2.5, we conditioned on all of them. This is why we saw that our estimate was 19% off from the true causal effect 1.05. Now that we've made the causal relationships clear with a causal graph, the backdoor criterion (Definition 4.1) tells us to only adjust for W and to not adjust for Z. More precisely, we were doing the following adjustment in Section 2.5:

$$\mathbb{E}_{W,Z}\mathbb{E}[Y \mid t, W, Z] \tag{4.38}$$

And now, we will use the backdoor adjustment (Theorem 4.2) to change our statistical estimand to the following:

$$\mathbb{E}_{W}\mathbb{E}[Y \mid t, W] \tag{4.39}$$

We have simply removed the collider Z from the variables we adjust for. For estimation, just as we did in Section 2.5, we use a model-assisted estimator. We replace the outer expectation over W with an empirical mean over W and replace the conditional expectation $\mathbb{E}[Y \mid t, W]$ with a machine learning model (in this case, linear regression).

Just as writing down the graph has lead us to simply not condition on Z in Equation 4.39, the code for estimation also barely changes. We need to change just a single line of code in our previous program (Listing 2.1). We display the full program with the fixed line of code below:

Listing 4.1: Python code for estimating the ATE, without adjusting for the collider

```
1  import numpy as np
2  import pandas as pd
3  from sklearn.linear_model import LinearRegression
4
5  Xt = df[['sodium', 'age']]
6  y = df['blood_pressure']
7  model = LinearRegression()
8  model.fit(Xt, y)
9
10 Xt1 = pd.DataFrame.copy(Xt)
11 Xt1['sodium'] = 1
12 Xt0 = pd.DataFrame.copy(Xt)
13 Xt0['sodium'] = 0
14 ate_est = np.mean(model.predict(Xt1) - model.predict(Xt0))
15 print('ATE estimate:', ate_est)
```

Full code, complete with simulation, is available at https://github.com/bradyneal/causal-book-code/blob/master/sodium_example.py.

Namely, we've changed line 5 from

```
5  Xt = df[['sodium', 'age', 'proteinuria']]
```

in Listing 2.1 to

```
5  Xt = df[['sodium', 'age']]
```

in Listing 4.1. When we run this revised code, we get an ATE estimate of 1.0502, which corresponds to **0.02%** error (true value is 1.05) when using

a fairly large sample.[9]

[9] Active reading exercise: Given that Y is generated as a linear function of T and W, could we have just used the coefficient in front of T in the linear regression as an estimate for the causal effect?

Progression of Reducing Bias When looking at the total association between T and Y by simply regressing Y on T, we got an estimate that was a staggering **407%** off of the true causal effect, due largely to confounding bias (see Section 2.5). When we adjusted for all covariates in Section 2.5, we reduced the percent error all the way down to **19%**. In this section, we saw this remaining error is due to collider bias. When we removed the collider bias, by not conditioning on the collider Z, the error became **non-existent**.

Potential Outcomes and M-Bias In fairness to the general culture around the potential outcomes framework, it is common to only condition on pretreatment covariates. This would prevent a practitioner who adheres to this rule from conditioning on the collider Z in Figure 4.18. However, there is no reason that there can't be pretreatment colliders that induce M-bias (Section 4.5.3). In Figure 4.19, we depict an example of M-bias that is created by conditioning on Z_2. We could fix this by additionally conditioning on Z_1 and/or Z_3, but in this example, they are unobserved (indicated by the dashed lines). This means that the only way to avoid M-bias in Figure 4.19 is to not condition on the covariates Z_2.

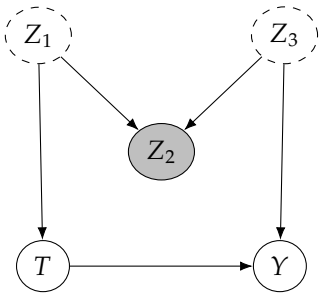


Figure 4.19: Causal graph depicting M-Bias that can only be avoided by not conditioning on the collider Z_2. This is due to the fact that the dashed nodes Z_1 and Z_3 are unobserved.

4.7 Assumptions Revisited

The first main set of assumptions is encoded by the causal graph that we write down. Exactly what this causal graph means is determined by two main assumptions, each of which can take on several different forms:

1. **The Modularity Assumption**
   Different forms:
   - Modularity Assumption for Causal Bayesian Networks (Assumption 4.1)
   - Modularity Assumption for SCMs (Assumption 4.2)
   - The Law of Counterfactuals (Definition 4.3)
2. **The Markov Assumption**
   Different equivalent forms:
   - Local Markov assumption (Assumption 3.1)
   - Bayesian network factorization (Definition 3.1)
   - Global Markov assumption (Theorem 3.1)

Given, these two assumptions (and positivity), if the backdoor criterion (Definition 4.1) is satisfied in our assumed causal graph, then we have identification. Note that although the backdoor criterion is a sufficient condition for identification, it is not a necessary condition. We will see this more in Chapter 6.

Now that you're familiar with causal graphical models and SCMs, it may be worth going back and rereading Chapter 2 while trying to make connections to what you've learned about graphical causal models in these past two chapters.

More Formal If you're really into fancy formalism, there are some relevant sources to check out. You can see the fundamental axioms that underlie The Law of Counterfactuals in [20, 21], or if you want a textbook, you can find them in [18, Chapter 7.3]. To see proofs of the equivalence of all three forms of the Markov assumption, see, for example, [13, Chapter 3].

[20]: Galles and Pearl (1998), 'An Axiomatic Characterization of Causal Counterfactuals'
[21]: Halpern (1998), 'Axiomatizing Causal Reasoning'

[18]: Pearl (2009), *Causality*

[13]: Koller and Friedman (2009), *Probabilistic Graphical Models: Principles and Techniques*

Connections to No Interference, Consistency, and Positivity The no interference assumption (Assumption 2.4) is commonly implicit in causal graphs, since the outcome Y (think Y_i) usually only has a single node T (think T_i) for treatment as a parent, rather than having multiple treatment nodes T_i, T_{i-1}, T_{i+1}, etc. as parents. However, causal DAGs can be extended to settings where there is interference [22]. Consistency (Assumption 2.5) follows from the axioms of SCMs (see [18, Corollary 7.3.2] and [23]). Positivity (Assumption 2.3) is still a very important assumption that we must make, though it is sometimes neglected in the graphical models literature.

[22]: Ogburn and VanderWeele (2014), 'Causal Diagrams for Interference'

[18]: Pearl (2009), *Causality*

[23]: Pearl (2010), 'On the consistency rule in causal inference: axiom, definition, assumption, or theorem?'

5 Randomized Experiments

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5.3 No Backdoor Paths 51

Randomized experiments are noticeably different from observational studies. In randomized experiments, the experimenter has complete control over the *treatment assignment mechanism* (how treatment is assigned). For example, in the most simple kind of randomized experiment, the experimenter randomly assigns (e.g. via coin toss) each participant to either the treatment group or the control group. This complete control over how treatment is chosen is what distinguishes randomized experiments from observational studies. In this simple experimental setup, the treatment isn't a function of covariates at all! In contrast, in observational studies, the treatment is almost always a function of some covariate(s). As we will see, this difference is key to whether or not confounding is present in our data.

In randomized experiments, association *is* causation. This is because randomized experiments are special in that they guarantee that there is no confounding. As a consequence, this allows us to measure the causal effect $\mathbb{E}[Y(1)] - \mathbb{E}[Y(0)]$ via the associational difference $\mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0]$. In the following sections, we explain why this is the case from a variety of different perspectives. If any one of these explanations clicks with you, that might be good enough. Definitely stick through to the most visually appealing explanation in Section 5.3.

5.1 Comparability and Covariate Balance

Ideally, the treatment and control groups would be the same, in all aspects, except for treatment. This would mean they only differ in the treatment they receive (i.e. they are *comparable*). This would allow us to attribute any difference in the outcomes of the treatment and control groups to the treatment. Saying that these treatment groups are the same in everything other than their treatment and outcomes is the same as saying they have the same distribution of confounders. Because people often check for this property on observed variables (often what people mean by "covariates"), this concept is known as *covariate balance*.

Definition 5.1 (Covariate Balance) *We have covariate balance if the distribution of covariates X is the same across treatment groups. More formally,*

$$P(X \mid T = 1) \overset{d}{=} P(X \mid T = 0) \tag{5.1}$$

The symbol $\overset{d}{=}$ means "equal in distribution."

Randomization implies covariate balance, across all covariates, even unobserved ones. Intuitively, this is because the treatment is chosen at random, regardless of X, so the treatment and control groups should look very similar. The proof is simple. Because T is not at all determined by X (solely by a coin flip), T is independent of X. This means that

$P(X \mid T = 1) \overset{d}{=} P(X)$. Similarly, it means $P(X \mid T = 0) \overset{d}{=} P(X)$. Therefore, we have $P(X \mid T = 1) \overset{d}{=} P(X \mid T = 0)$.

Although we have proven that randomization implies covariate balance, we have not proven that that covariate balance implies that association is causation.[1] We'll now prove that by showing that $P(y \mid do(t)) = P(y \mid t)$. For the proof, the main property we utilize is that covariate balance implies X and T are independent.

[1] Recall that the intuition is that covariate balance means that everything is the same between the treatment groups, except for the treatment, so the treatment must be the explanation for the change in Y.

Proof. First, let X be a sufficient adjustment set that potentially contains *unobserved* variables (randomization also balances unobserved covariates). Such an adjustment set must exist because we allow it to contain any variables, observed or unobserved. Then, we have the following from the backdoor adjustment (Theorem 4.2):

$$P(y \mid do(t)) = \sum_x P(y \mid t, x)P(x) \tag{5.2}$$

By multiplying by $\frac{P(t|x)}{P(t|x)}$, we get the joint distribution in the numerator:

$$= \sum_x \frac{P(y \mid t, x)P(t \mid x)P(x)}{P(t \mid x)} \tag{5.3}$$

$$= \sum_x \frac{P(y, t, x)}{P(t \mid x)} \tag{5.4}$$

Now, we use the important property that $X \perp\!\!\!\perp T$:

$$= \sum_x \frac{P(y, t, x)}{P(t)} \tag{5.5}$$

An application of Bayes rule and marginalization gives us the rest:

$$= \sum_x P(y, x \mid t) \tag{5.6}$$

$$= P(y \mid t) \tag{5.7}$$

□

5.2 Exchangeability

Exchangeability (Assumption 2.1) gives us another perspective on why randomization makes causation equal to association. To see why, consider the following thought experiment. We decide an individual's treatment group using a random coin flip as follows: if the coin is heads, we assign the individual to the treatment group ($T = 1$), and if the coins is tails, we assign the individual to the control group ($T = 0$). If the groups are exchangeable, we could exchange these groups, and the average outcomes would remain the same. This is intuitively true if we chose the groups with a coin flip. Imagine simply swapping the meaning of "heads" and "tails" in this experiment. Would you expect that to change the results at all? No. This is why randomized experiments give us exchangeability.

Recall from Section 2.3.2 that mean exchangeability is formally the following:

$$\mathbb{E}[Y(1) \mid T = 1] = \mathbb{E}[Y(1) \mid T = 0] \tag{5.8}$$
$$\mathbb{E}[Y(0) \mid T = 0] = \mathbb{E}[Y(0) \mid T = 1] \tag{5.9}$$

The "exchange" is when we go from $Y(1)$ in the treatment group to $Y(1)$ in the control group (Equation 5.8) and from $Y(0)$ in the control group to $Y(0)$ in the treatment group (Equation 5.9).

To see the proof of why association is causation in randomized experiments through the lens of exchangeability, recall the proof from Section 2.3.2. First, recall that Equation 5.8 means that both quantities in it are equal to the marginal expected outcome $\mathbb{E}[Y(1)]$ and, similarly, that Equation 5.8 means that both quantities in it are equal to the marginal expected outcome $\mathbb{E}[Y(0)]$. Then, we have the following proof:

$$\begin{aligned}\mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] &= \mathbb{E}[Y(1) \mid T = 1] - \mathbb{E}[Y(0) \mid T = 0] && \text{(2.3 revisited)} \\ &= \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0] && \text{(2.4 revisited)}\end{aligned}$$

5.3 No Backdoor Paths

The final perspective that we'll look at to see why association is causation in randomized experiments is that of graphical causal models. In regular observational data, there is almost always confounding. For example, in Figure 5.1, we see that X is a confounder of the effect of T on Y. Non-causal association flows along the backdoor path $T \leftarrow X \rightarrow Y$.

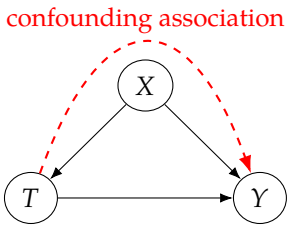

Figure 5.1: Causal structure of X confounding the effect of T on Y.

However, if we randomize T, something magical happens: T no longer has any causal parents, as we depict in Figure 5.2. This is because T is purely random. It doesn't depend on anything other than the output of a coin toss (or a quantum random number generator, if you're into the kind of stuff). Because T has no incoming edges, under randomization, there are no backdoor paths. So the empty set is a sufficient adjustment set. This means that all of the association that flows from T to Y is causal. We can identify $P(Y \mid do(T = t))$ by simply applying the backdoor adjustment (Theorem 4.2), adjusting for the empty set:

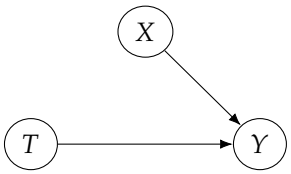

Figure 5.2: Causal structure when we randomize treatment.

$$P(Y \mid do(T = t)) = P(Y \mid T = t)$$

With that, we conclude our discussion of why association is causation in randomized experiments. Hopefully, at least one of these three explanations is intuitive to you and easy to store in long-term memory.

6 Nonparametric Identification

In Section 4.4, we saw that satisfying the backdoor criterion is sufficient to give us identifiability, but is the backdoor criterion also necessary? In other words, is it possible to get identifiability without being able to block all backdoor paths?

As an example, consider that we have data generated according to the graph in Figure 6.1. We don't observe W in this data, so we can't block the backdoor path through W and the confounding association that flows along it. But we still need to identify the causal effect. It turns out that it is possible to identify the causal effect in this graph, using the frontdoor criterion. We'll see the frontdoor criterion and corresponding adjustment in Section 6.1. Then, we'll consider even more general identification in Section 6.2 when we introduce *do*-calculus. We'll conclude with graphical conditions for identifiability in Section 6.3.

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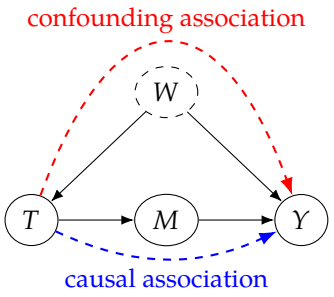


Figure 6.1: Causal graph where W is unobserved, so we cannot block the backdoor path. We depict the flow of causal association and the flow of confounding association with dashed lines.

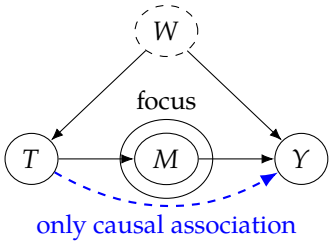


Figure 6.2: In contrast to Figure 6.1, when we focus our analysis on M, we are able to isolate only the *causal* association.

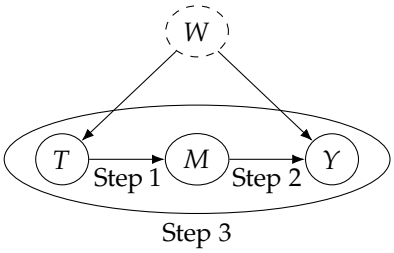


Figure 6.3: Illustration of steps to get to the frontdoor adjustment.

6.1 Frontdoor Adjustment

The high-level intuition for why we can identify the causal effect of T on Y in the graph in Figure 6.1 (even when we can't adjust for the confounder W because it is unobserved) is as follows: a mediator like M is very helpful; we can isolate the association that flows through M by focusing our statistical analysis on M, and the only association that flows through M is causal association (association flowing along directed paths from T to Y). We illustrate this intuition in Figure 6.2, where we depict only the *causal* association. In this section, we will focus our analysis on M using a three step procedure (see Figure 6.3 for our corresponding illustration):

1. Identify the causal effect of T on M.
2. Identify the causal effect of M on Y.
3. Combine the above steps to identify the causal effect of T on Y.

Step 1 First, we will identify the effect of T on M: $P(m \mid do(t))$. Because Y is a collider on the $T - M$ path through W, it blocks that backdoor path. So there are no unblocked backdoor paths from T to M. This means that the only association that flows from T to M is the causal association that flows along the edge connecting them. Therefore, we have the following identification via the backdoor adjustment (Theorem 4.2, using the empty set as the adjustment set):[1]

$$P(m \mid do(t)) = P(m \mid t) \tag{6.1}$$

Step 2 Second, we will identify the effect of M on Y: $P(y \mid do(m))$. Because T blocks the backdoor path $M \leftarrow T \leftarrow W \rightarrow Y$, we can simply

[1] Active reading exercise: Write a proof for Equation 6.1 without using the backdoor adjustment. Instead, start from the truncated factorization (Proposition 4.1) like we did in Section 4.3.1. Hint: The proof can be quite short. We provide a proof in Appendix A.1, in case you get stuck.

adjust for T. Therefore, using the backdoor adjustment again, we have the following:

$$P(y \mid do(m)) = \sum_t P(y \mid m, t)\, P(t) \tag{6.2}$$

Step 3 Now that we know how changing T changes M (step 1) and how changing M changes Y (step 2), we can combine these two to get how changing T changes Y (through M):

$$P(y \mid do(t)) = \sum_m P(m \mid do(t))\, P(y \mid do(m)) \tag{6.3}$$

The first factor on the right-hand side corresponds to setting T to t and observing the resulting value of M. The second factor corresponds to setting M to exactly the value m that resulted from setting T and then observing what value of Y results. We must sum over m because $P(m \mid do(t))$ is probabilistic, so we must sum over its support. In other words, we must sum over all possible realizations m of the random variables whose distribution is $P(M \mid do(t))$.

If we then plug in Equations 6.1 and 6.2 into Equation 6.3, we get the *frontdoor adjustment* (keep reading to see the definition of the frontdoor criterion):

Theorem 6.1 (Frontdoor Adjustment) *If (T, M, Y) satisfy the frontdoor criterion and we have positivity, then*

$$P(y \mid do(t)) = \sum_m P(m \mid t) \sum_{t'} P(y \mid m, t')\, P(t') \tag{6.4}$$

The causal graph we've been using (Figure 6.4) is an example of a simple graph that satisfies the *frontdoor criterion*. To get the full definition, we must first define *complete/full mediation*: a set of variables M completely mediates the effect of T on Y if all causal (directed) paths from T to Y go through M. We now give the general definition of the frontdoor criterion:

Definition 6.1 (Frontdoor Criterion) *A set of variables M satisfies the frontdoor criterion relative to T and Y if the following are true:*

1. *M completely mediates the effect of T on Y (i.e. all causal paths from T to Y go through M).*
2. *There is no unblocked backdoor path from T to M.*
3. *All backdoor paths from M to Y are blocked by T.*[2]

Although Equations 6.1 and 6.2 are straightforward applications of the backdoor adjustment, we hand-waved our way to Equation 6.3, which was key to the frontdoor adjustment (Theorem 6.1). We'll now walk through how to get Equation 6.3. Active reading exercise: Feel free to stop reading here and do this yourself.

We are about to enter *Equationtown* (Figure 6.5), so if you are satisfied with the intuition we gave for step 3 and prefer to not see a lot of equations, feel free to skip to the end of the proof (denoted by the □ symbol).

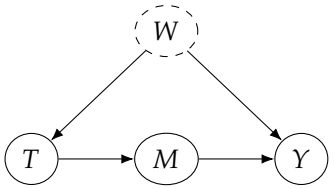


Figure 6.4: Simple causal graph that satisfies the frontdoor criterion

[2] Active reading exercise: Think of a graph other than Figure 6.4 that satisfies the frontdoor criterion. Also, for each condition, think of a graph that does *not* satisfy only that condition.

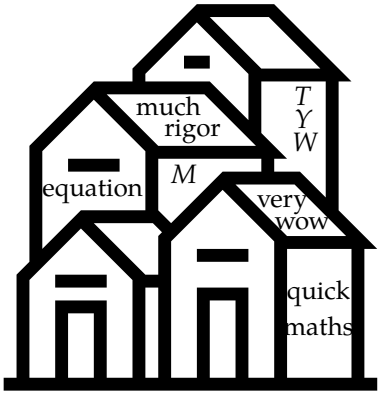


Figure 6.5: Equationtown

Proof. As usual, we start with the truncated factorization, using the causal graph in Figure 6.4. From the Bayesian network factorization (Definition 3.1), we have the following:

$$P(w, t, m, y) = P(w)\,P(t \mid w)\,P(m \mid t)\,P(y \mid w, m) \tag{6.5}$$

Then, using the truncated factorization (Proposition 4.1), we remove the factor for T:

$$P(w, m, y \mid do(t)) = P(w)\,P(m \mid t)\,P(y \mid w, m) \tag{6.6}$$

Next, we marginalize out w and m:

$$\sum_m \sum_w P(w, m, y \mid do(t)) = \sum_m \sum_w P(w)\,P(m \mid t)\,P(y \mid w, m) \tag{6.7}$$

$$P(y \mid do(t)) = \sum_m P(m \mid t) \sum_w P(y \mid w, m)\,P(w) \tag{6.8}$$

Even though we've removed all the *do* operators, recall that we are not done because W is unobserved. So we must also remove the w from the expression. This is where we have to get a bit creative.

We want to be able to combine $P(y \mid w, m)$ and $P(w)$ into a joint factor over both y and w so that we can marginalize out w. To do this, we need to get m behind the conditioning bar of the $P(w)$ factor. This would be easy if we could just swap $P(w)$ out for $P(w \mid m)$ in Equation 6.8.[3] The key thing to notice is that we actually can include m behind the conditioning bar if t were also there because T d-separates W from M in Figure 6.6. In math, this means that the following equality holds:

$$P(w \mid t) = P(w \mid t, m) \tag{6.9}$$

[3] Active reading exercise: Why would it be easy to marginalize out w if it were the case that $P(w) = P(w \mid m)$? And why does this equality not hold?

Great, so how do we get t into this party? The usual trick of conditioning on it and marginalizing it out:

$$\begin{aligned}
P(y \mid do(t)) &= \sum_m P(m \mid t) \sum_w P(y \mid w, m)\,P(w) && \text{(6.8 revisited)} \\
&= \sum_m P(m \mid t) \sum_w P(y \mid w, m) \sum_{t'} P(w \mid t')\,P(t') && (6.10) \\
&= \sum_m P(m \mid t) \sum_w P(y \mid w, m) \sum_{t'} P(w \mid t', m)\,P(t') && (6.11) \\
&= \sum_m P(m \mid t) \sum_{t'} P(t') \sum_w P(y \mid w, m)\,P(w \mid t', m) && (6.12)
\end{aligned}$$

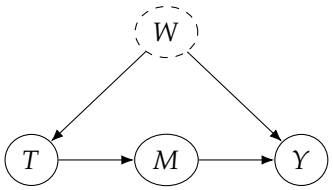


Figure 6.6: Simple causal graph that satisfies the frontdoor criterion

Great, but now we can't combine $P(y \mid w, m)$ and $P(w \mid t', m)$ because $P(y \mid w, m)$ is missing this newly introduced t' behind its conditioning bar. Luckily, we can fix that[4] and combine the two factors:

[4] Active reading exercise: Why is $P(y \mid w, m)$ equal to $P(y \mid w, t', m)$?

$$\begin{aligned}
&= \sum_m P(m \mid t) \sum_{t'} P(t') \sum_w P(y \mid w, m)\,P(w \mid t', m) && (6.13) \\
&= \sum_m P(m \mid t) \sum_{t'} P(t') \sum_w P(y \mid w, t', m)\,P(w \mid t', m) && (6.14) \\
&= \sum_m P(m \mid t) \sum_{t'} P(t') \sum_w P(y, w \mid t', m) && (6.15) \\
&= \sum_m P(m \mid t) \sum_{t'} P(t') P(y \mid t', m) && (6.16)
\end{aligned}$$

This matches the result stated in Theorem 6.1, so we've completed the derivation of the frontdoor adjustment without using the backdoor adjustment. However, we still need to show that Equation 6.3 is correct to justify step 3. To do that, all that's left is to recognize that these parts match Equations 6.1 and 6.2 and plug those in:

$$P(m \mid do(t)) = P(m \mid t) \tag{6.1}$$

$$P(y \mid do(m)) = \sum_t P(y \mid m, t)\, P(t) \tag{6.2}$$

$$= \sum_m P(m \mid do(t))\, P(y \mid do(m)) \tag{6.17}$$

□

And we're done! We just needed to be a bit clever with our uses of d-separation and marginalization. Part of why we went through that proof is because we will prove the frontdoor adjustment using *do*-calculus in Section 6.2. This way you can easily compare a proof using the truncated factorization to a proof using *do*-calculus to prove the same result.

6.2 *do*-calculus

As we saw in the last section, it turns out that satisfying the backdoor criterion (Definition 4.1) isn't necessary to identify causal effects. For example, if the frontdoor criterion (Definition 6.1) is satisfied, that also gives us identifiability. This leads to the following questions: can we identify causal estimands when the associated causal graph satisfies neither the backdoor criterion nor the frontdoor criterion? If so, how? Pearl's *do-calculus* [24] gives us the answer to these questions.

[24]: Pearl (1995), 'Causal diagrams for empirical research'

As we will see, the *do*-calculus gives us tools to identify causal effects using the causal assumptions encoded in the causal graph. It will allow us to identify any causal estimand that is identifiable. More concretely, consider an arbitrary causal estimand $P(Y \mid do(T = t), X = x)$, where Y is an arbitrary set of outcome variables, T is an arbitrary set of treatment variables, and X is an arbitrary (potentially empty) set of covariates that we want to choose how specific the causal effect we're looking at is. Note that this means we can use *do*-calculus to identify causal effects where there are multiple treatments and/or multiple outcomes.

In order to present the rules of *do*-calculus, we must define a bit of notation for augmented versions of the causal graph G. Let $G_{\overline{X}}$ denote the graph that we get if we take G and remove all of the *incoming* edges to nodes in the set X; recall from Section 4.2 that this is known as the *manipulated graph*. Let $G_{\underline{X}}$ denote the graph that we get if we take G and remove all of the *outgoing* edges from nodes in the set X. The mnemonic meaning to help you remember this is to think of parents as drawn above their children in the graph, so the bar above X is cutting its incoming edges and the bar below X is cutting its outgoing edges. Combining these two, we'll use $G_{\overline{X}\underline{Z}}$ to denote the graph with the incoming edges to X and the outgoing edges from Z removed. And recall from Section 3.7 that we use $\perp\!\!\!\perp_G$ to denote d-separation in G. We're now ready; *do*-calculus consists of just three rules:

Theorem 6.2 (Rules of *do*-calculus) *Given a causal graph G, an associated*

distribution P, and disjoint sets of variables Y, T, Z, and W, the following rules hold.

Rule 1:

$$P(y \mid do(t), z, w) = P(y \mid do(t), w) \quad \text{if } Y \perp\!\!\!\perp_{G_{\overline{T}}} Z \mid T, W \tag{6.18}$$

Rule 2:

$$P(y \mid do(t), do(z), w) = P(y \mid do(t), z, w) \quad \text{if } Y \perp\!\!\!\perp_{G_{\overline{T},\underline{Z}}} Z \mid T, W \tag{6.19}$$

Rule 3:

$$P(y \mid do(t), do(z), w) = P(y \mid do(t), w) \quad \text{if } Y \perp\!\!\!\perp_{G_{\overline{T},\overline{Z(W)}}} Z \mid T, W \tag{6.20}$$

where $Z(W)$ denotes the set of nodes of Z that aren't ancestors of any node of W in $G_{\overline{T}}$.

Now, rather than recreate the proofs for these rules from Pearl [24], we'll give intuition for each of them in terms of concepts we've already seen in this book.

[24]: Pearl (1995), 'Causal diagrams for empirical research'

Rule 1 Intuition If we take Rule 1 and simply remove the intervention $do(t)$, we get the following (Active reading exercise: what familiar concept is this?):

$$P(y \mid z, w) = P(y \mid w) \quad \text{if } Y \perp\!\!\!\perp_G Z \mid W \tag{6.21}$$

This is just what d-separation gives us under the Markov assumption; recall from Theorem 3.1 that d-separation in the graph implies conditional independence in P. This means that Rule 1 is simply a generalization of Theorem 3.1 to interventional distributions.

Rule 2 Intuition Just as with Rule 1, we'll remove the intervention $do(t)$ from Rule 2 and see what this reminds us of (Active reading exercise: what concept does this remind you of?):

$$P(y \mid do(z), w) = P(y \mid z, w) \quad \text{if } Y \perp\!\!\!\perp_{G_{\underline{Z}}} Z \mid W \tag{6.22}$$

This is exactly what we do when we justify the backdoor adjustment (Theorem 4.2) using the backdoor criterion (Definition 4.1). As we saw at the ends of Section 3.8 and Section 4.4. Association *is* causation if the outcome Y and the treatment Z are d-separated by some set of variables that are conditioned on W. So rule 2 is a generalization of the backdoor adjustment to interventional distributions.

Rule 3 Intuition This is the trickiest rule to understand. Just as with the other two rules, we'll first remove the intervention $do(t)$ to make thinking about this simpler:

$$P(y \mid do(z), w) = P(y \mid w) \quad \text{if } Y \perp\!\!\!\perp_{G_{\overline{Z(W)}}} Z \mid W \tag{6.23}$$

To get the equality in this equation, it must be the case that removing the intervention $do(z)$ (which is like taking the manipulated graph and reintroducing the edges going into Z) introduces no new association that can affect Y. Because $do(z)$ removes the incoming edges to Z to give us $G_{\overline{Z}}$, the main association that we need to worry about is association flowing from Z to Y in $G_{\overline{Z}}$ (causal association). Therefore, you might

expect that the condition that gives us the equality in Equation 6.23 is $Y \perp\!\!\!\perp_{G_{\overline{Z}}} Z \mid W$. However, we have to refine this a bit to prevent inducing association by conditioning on the descendants of colliders (recall from Section 3.6). Namely, Z could contain colliders in G, and W could contain descendants of these colliders. Therefore, to not induce new association through colliders in Z when we reintroduce the incoming edges to Z to get G, we must limit the set of manipulated nodes to those that are not ancestors of nodes in the conditioning set W: $Z(W)$.

Completeness of *do*-calculus Maybe there could exist causal estimands that are identifiable but that can't be identified using only the rules of *do*-calculus in Theorem 6.2. Fortunately, Shpitser and Pearl [25] and Huang and Valtorta [26] independently proved that this is not the case. They proved that *do*-calculus is complete, which means that these three rules are sufficient to identify all identifiable causal estimands. Because these proofs are constructive, they also admit algorithms that identify any causal estimand in polynomial time.

[25]: Shpitser and Pearl (2006), 'Identification of Joint Interventional Distributions in Recursive Semi-Markovian Causal Models'
[26]: Huang and Valtorta (2006), 'Pearl's Calculus of Intervention is Complete'

Nonparametric Identification Note that all of this is about *nonparametric* identification; in other words, *do*-calculus tells us if we can identify a given causal estimand using only the causal assumptions encoded in the causal graph. If we introduce more assumptions about the distribution (e.g. linearity), we can identify more causal estimands. That would be known as *parametric* identification. We don't discuss parametric identification in this chapter, though we will in later chapters.

6.2.1 Application: Frontdoor Adjustment

Recall the simple graph we used that satisfies the frontdoor criterion (Figure 6.7), and recall the frontdoor adjustment:

$$P(y \mid do(t)) = \sum_m P(m \mid t) \sum_{t'} P(y \mid m, t') P(t') \qquad \text{(6.4 revisited)}$$

At the end of Section 6.1, we saw a proof for the frontdoor adjustment using just the truncated factorization. To get an idea for how *do*-calculus works and the intuition we use in proofs that use it, we'll now do the frontdoor adjustment proof using the rules of *do*-calculus.

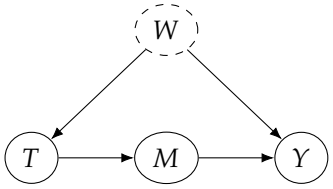


Figure 6.7: Simple causal graph that satisfies the frontdoor criterion

Proof. Our goal is to identify $P(y \mid do(t))$. Because we have the intuition we described in Section 6.1 that the full mediator M will help us out, the first thing we'll do is introduce M into the equation via the marginalization trick:

$$P(y \mid do(t)) = \sum_m P(y \mid do(t), m) P(m \mid do(t)) \qquad (6.24)$$

Because the backdoor path from T to M in Figure 6.7 is blocked by the collider Y, all of the association that flows from T to M is causal, so we can apply Rule 2 to get the following:

$$= \sum_m P(y \mid do(t), m) P(m \mid t) \qquad (6.25)$$

Now, because M is a full mediator of the causal effect of T on Y, we should be able to replace $P(y \mid do(t), m)$ with $P(y \mid do(m))$, but this will

take two steps of *do*-calculus. To remove $do(t)$, we'll need to use Rule 3, which requires that T have no causal effect on Y in the relevant graph. We can get to a graph like that by removing the edge from T to M (Figure 6.9); in *do*-calculus, we do this by using Rule 2 (in the opposite direction as before) to $do(m)$. We can do this because the existing $do(t)$ makes it so there are no backdoor paths from M to Y in $G_{\overline{T}}$ (Figure 6.8).

$$= \sum_m P(y \mid do(t), do(m))\, P(m \mid t) \tag{6.26}$$

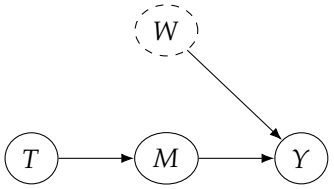


Figure 6.8: $G_{\overline{T}}$

Now, as we planned, we can remove the $do(t)$ using Rule 3. We can use Rule 3 here because there is no causation flowing from T to Y in $G_{\overline{M}}$ (Figure 6.9).

$$= \sum_m P(y \mid do(m))\, P(m \mid t) \tag{6.27}$$

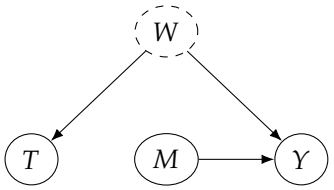


Figure 6.9: $G_{\overline{M}}$

All that's left is to remove this last *do*-operator. As we discussed in Section 6.1, T blocks the only backdoor path from M to Y in the graph (Figure 6.10). This means, that if we can condition on T, we can get rid of this last *do*-operator. As usual, we do that by conditioning on and marginalizing out T. Rearranging a bit and using t' for the marginalization since t is already present:

$$= \sum_m P(m \mid t) \sum_{t'} P(y \mid do(m), t')\, P(t' \mid do(m)) \tag{6.28}$$

Now, we can simply apply Rule 2, since T blocks the backdoor path from M to Y:

$$= \sum_m P(m \mid t) \sum_{t'} P(y \mid m, t')\, P(t' \mid do(m)) \tag{6.29}$$

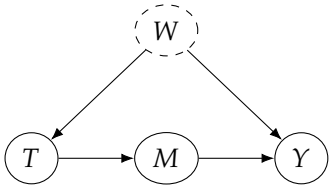


Figure 6.10: G

And finally, we can apply Rule 3 to remove the last $do(m)$ because there is no causal effect of M on T (i.e. there is no directed path from M to T in the graph in (Figure 6.10).

$$= \sum_m P(m \mid t) \sum_{t'} P(y \mid m, t')\, P(t') \tag{6.30}$$

□

That concludes our proof of the frontdoor adjustment using *do*-calculus. It follows a different path than the proof we gave at the end of Section 6.1, where we used the truncated factorization, but both proofs rely heavily on intuition we get from looking at the graph.

Active reading exercise: Assuming the backdoor criterion, prove the backdoor adjustment using the rules of *do*-calculus.

6.3 Determining Identifiability from the Graph

It's nice to know that we can identify any causal estimand that is possible to identify using *do*-calculus, but this isn't as satisfying as knowing whether a causal estimand is identifiable by simply looking at the causal graph. For example, the backdoor criterion (Definition 4.1) and the frontdoor criterion (Definition 6.1) gave us simple ways to know for sure that a causal estimand is identifiable. However, there are plenty of

causal estimands that are identifiable, even though the corresponding causal graphs don't satisfy the backdoor or frontdoor criterion. More general graphical criteria exist that will tell us that these estimands are identifiable. We will discuss these more general graphical criteria for identifiability in this section.

Single Variable Intervention When we care about causal effects of an intervention on a single variable, Tian and Pearl [27] provide a relatively simple graphical criterion that is sufficient for identifiability: the *unconfounded children criterion*.

[27]: Tian and Pearl (2002), 'A General Identification Condition for Causal Effects'

Definition 6.2 (Unconfounded Children Criterion) *This criterion is satisfied if it is possible to block all backdoor paths from the treatment variable T to all of its children that are ancestors of Y with a single conditioning set.*

This criterion generalizes the backdoor criterion (Definition 4.1) and the frontdoor criterion (Definition 6.1). Like them, it is a *sufficient* condition for identifiability:

Theorem 6.3 (Unconfounded Children Identifiability) *Let Y be the set of outcome variables and T be a single variable. If the unconfounded children criterion and positivity are satisfied, then $P(Y = y \mid do(T = t))$ is identifiable [27].*

The intuition for unconfounded children criterion implies identifiability is similar to the intuition for the frontdoor criterion; if we can isolate all of the causal association flowing out of treatment along directed paths to Y, we have identifiability. To see this intuition, first, consider that all of the causal association from T must flow through its children. We can isolate this causal association if there is no confounding between T and any of its children.[5] This isolation of all of the causal association is what gives us identifiability of the causal effect of T on any other node in the graph. This intuition might lead you to suspect that this criterion *is necessary* in the very specific case where the outcome set Y is all of the other variables in the graph other than T; it turns out that this is true [27]. But this condition is *not necessary* if Y is a smaller set than that.

[5] This is analogous to what we saw with the frontdoor criterion in Section 6.1, where we could isolate the causal association flowing through the full mediator M if the $T - M$ relationship is unconfounded (no unblocked backdoor paths).

To give you a more visual grasp of the intuition for why the unconfounded children criterion is sufficient for identification, we give an example graph in Figure 6.12. In Figure 6.12a, we visualize the flow of confounding association and causal association that flows in this graph. Then, we depict the isolation of the causal association in that graph in Figure 6.12b.

Necessary Condition The unconfounded children criterion is not necessary for identifiability, but it might aid your graphical intuition to have a necessary condition in mind. Here is one: For each backdoor path from T to any child M of T that is an ancestor of Y, it is possible to block that path [18, p. 92]. The intuition for this is that because the causal association that flows from T to Y must go through children of T that are ancestors of Y, to be able to isolate this causal association, the effect of T on these mediating children must be unconfounded. And a prerequisite to these $T - M$ (parent-child) relationships being unconfounded is that any single backdoor path from T to M must be blockable (what we state in this condition). Unfortunately, this condition is *not sufficient*. To see why, consider Figure 6.11. The backdoor path $T \leftarrow W_1 \rightarrow W_2 \leftarrow W_3 \rightarrow Y$

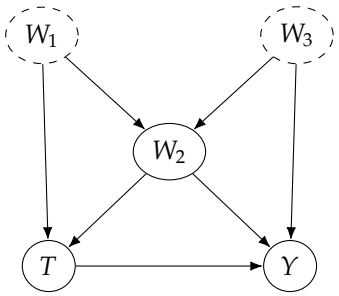
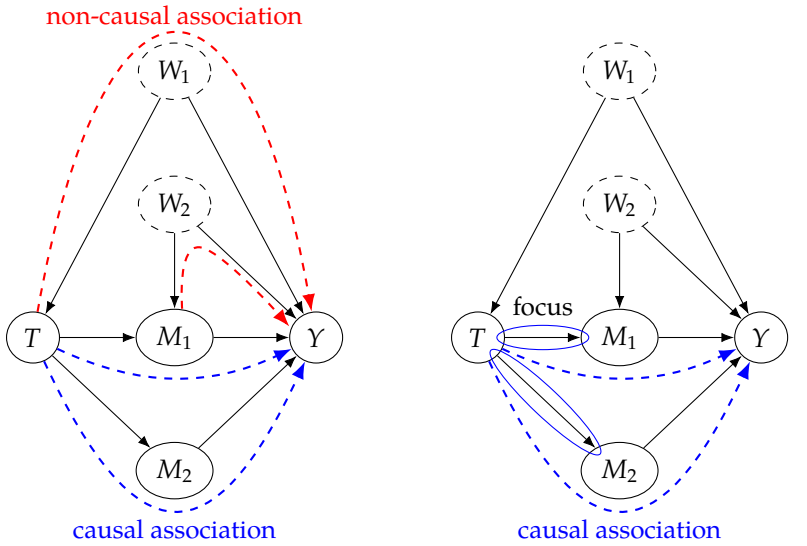


Figure 6.11: Graph where blocking one backdoor path unblocks another

[18]: Pearl (2009), *Causality*



(a) Visualization of the flow of confounding association and causal association.

(b) Visualization of the isolation of the causal association flowing from T to its children, allowing the unconfounded children criterion to imply identifiability.

Figure 6.12: Example graph that satisfies the unconfounded children criterion

is blocked by the collider W_2. And we can block the the backdoor path $T \leftarrow W_2 \rightarrow Y$ by conditioning on W_2. However, conditioning on W_2 unblocks the other backdoor path where W_2 is a collider. Being able to block both paths individually does not mean we can block them both with a single conditioning set. In sum, the unconfounded children criterion is sufficient but not necessary, and this related condition is necessary but not sufficient. Also, everything we've seen in this section so far is for a single variable intervention.

Necessary and Sufficient Conditions for Multiple Variable Interventions Shpitser and Pearl [25] provide a necessary and sufficient criterion for identifiability of $P(Y = y \mid do(T = t))$ when Y and T are arbitrary sets of variables: *the hedge criterion*. However, this is outside the scope of this book, as it requires more complex objects such as hedges, C-trees, and other leafy objects. Moving further along, Shpitser and Pearl [28] provide a necessary and sufficient criterion for the most general type of causal estimand: *conditional causal effects*, which take the form $P(Y = y \mid do(T = t), X = x)$, where Y, T, and X are all arbitrary sets of variables.

[25]: Shpitser and Pearl (2006), 'Identification of Joint Interventional Distributions in Recursive Semi-Markovian Causal Models'

[28]: Shpitser and Pearl (2006), 'Identification of Conditional Interventional Distributions'

Active reading exercises:

1. Is the unconfounded criterion (Definition 6.2) satisfied in Figure 6.13a?
2. Is the unconfounded criterion satisfied in Figure 6.13b?
3. Can we get identifiability in Figure 6.13b via any simpler criterion that we've seen before?

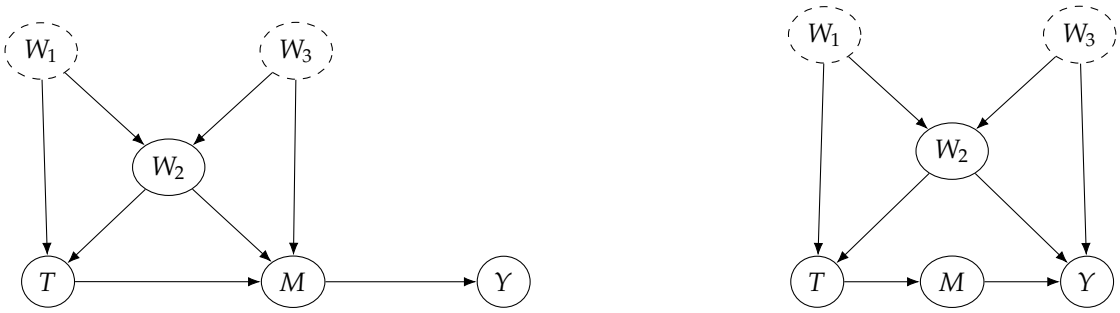


Figure 6.13: Graphs for the questions about the unconfounded children criterion

7 Estimation

In the previous chapter, we covered identification. Once we identify some causal estimand by reducing it to a statistical estimand, we still have more work to do. We need to get a corresponding estimate. In this chapter, we'll cover a variety of estimators that we can use to do this. This isn't meant to be anywhere near exhaustive as there are many different estimators of causal effects, but it is meant to give you a solid introduction to them.

All of the estimators that we include full sections on are model-assisted estimators (recall from Section 2.4). And they all work with arbitrary statistical models such as the ones you might get from scikit-learn Pedregosa et al. [29].

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[29]: Pedregosa et al. (2011), 'Scikit-learn: Machine Learning in Python'

7.1 Preliminaries

Recall from Chapter 2 that we denote the individual treatment effect (ITE) with τ_i and average treatment effect (ATE) with τ:

$$\tau_i \triangleq Y_i(1) - Y_i(0) \tag{7.1}$$

$$\tau \triangleq \mathbb{E}[Y_i(1) - Y_i(0)] \tag{7.2}$$

ITEs are the most specific kind of causal effects, but they are hard to estimate without strong assumptions (on top of those discussed in Chapters 2 and 4). However, we often want to estimate causal effects that are a bit more individualized than the ATE.

For example, say we've observed an individual's covariates x; we might like to use those to estimate a more specific effect for that individual (and anyone else with covariates x). This brings us to the *conditional average treatment effect* (CATE) $\tau(x)$:

$$\tau(x) \triangleq \mathbb{E}[Y_i(1) - Y_i(0) \mid X = x] \tag{7.3}$$

The X that is conditioned on does not need to consist of all of the observed covariates, but this is often the case when people refer to CATEs. We call that *individualized average treatment effects* (IATEs).

ITEs and "CATEs" (what we call IATEs) are sometimes conflated, but they are not the same. For example, two individuals could have the same covariates, but their potential outcomes could be different because of other unobserved differences between these individuals. If we encompass everything about an individual that is relevant to their potential outcomes in the vector I, then ITEs and "CATEs" are the same if $X = I$. In a causal graph, I corresponds to all of the exogenous variables in the magnified graph that have causal association flowing to Y.[1]

[1] This paragraph contains a lot of information. Active reading exercise:
1) Convince yourself that ITEs and "CATEs" (what we call IATEs) are the same if $X = I$.
2) Convince yourself that I corresponds to the exogenous variables in the magnified graph that have causal association flowing to Y.

Unconfoundedness Throughout this chapter, whenever we are estimating an ATE, we will assume that W is a sufficient adjustment set, and whenever we are estimating a CATE, we will assume that $W \cup X$ is a sufficient adjustment set. In other words, for ATE estimation, we assume that W satisfies the backdoor criterion (Definition 4.1); equivalently for ATE estimation, we assume that we have conditional exchangeability given W (Assumption 2.2). And similarly for CATE estimation, assuming $W \cup X$ is a sufficient adjustment set means that we are assuming that $W \cup X$ satisfies the backdoor criterion / gives us unconfoundedness. This unconfoundedness assumption gives us parametric identification[2] and allows us to focus on estimation in this chapter.

[2] By "parametric identification," we mean identification under the parametric assumptions of our statistical models. For example, these assumptions are for extrapolation if we don't have positivity.

7.2 Conditional Outcome Modeling (COM)

We are interested in estimating the ATE τ. We'll start with recalling the adjustment formula (Theorem 2.1), which can be derived as a corollary of the backdoor adjustment (Theorem 4.2), as we saw in Section 4.4.1:

$$\tau \triangleq \mathbb{E}[Y(1) - Y(0)] = \mathbb{E}_W\left[\mathbb{E}[Y \mid T = 1, W] - \mathbb{E}[Y \mid T = 0, W]\right] \quad (7.4)$$

On the left-hand side of Equation 7.4, we have a causal estimand, and on the right-hand side, we have a statistical estimand (i.e. we have identified this causal quantity). Then, the next step in the Identification-Estimation Flowchart (see Figure 7.1 reproduced from Section 2.4) is to get an estimate of this (statistical) estimand.

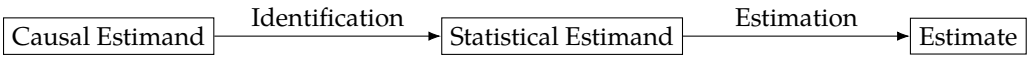


Figure 7.1: The Identification-Estimation Flowchart – a flowchart that illustrates the process of moving from a target causal estimand to a corresponding estimate, through identification and estimation.

The most straightforward thing to do is to just fit a statistical model (machine learning model) to the conditional expectation $\mathbb{E}[Y \mid T, W]$ and then approximate $\mathbb{E}_W$ with an empirical mean over the n data points ($\frac{1}{n}\sum_i$). And this is exactly what we did in the simple examples of estimation in Sections 2.5 and 4.6.2. To make this more clear, we introduce μ in place of this conditional expectation:

$$\mu(1, w) - \mu(0, w) \triangleq \mathbb{E}[Y \mid T = 1, W = w] - \mathbb{E}[Y \mid T = 0, W = w] \quad (7.5)$$

Then, we can fit a statistical model to μ. We will denote that these fitted models are approximations of μ with a hat: $\hat{\mu}$. We will refer to a model $\hat{\mu}$ as a *conditional outcome model*. Now, we can cleanly write the model-assisted estimator (for the ATE) that we've described:

$$\hat{\tau} = \frac{1}{n}\sum_i \left(\hat{\mu}(1, w_i) - \hat{\mu}(0, w_i)\right) \quad (7.6)$$

Active reading exercise: What are the two different approximations we make in this estimator and what parts of the statistical estimand in Equation 7.4 do each of them replace?

We will refer to estimators that take this form as *conditional outcome model (COM) estimators*. Because minimizing the mean-squared error (MSE) of predicting Y from (T, X) pairs is equivalent to modeling this conditional expectation [see, e.g., 10, Section 2.4], there are many different models we

[10]: Hastie et al. (2001), *The Elements of Statistical Learning*

can use for $\hat{\mu}$ in Equation 7.6 to get a COM estimator (see, e.g., scikit-learn [29]).

[29]: Pedregosa et al. (2011), 'Scikit-learn: Machine Learning in Python'

For CATE estimation, because we assumed that $W \cup X$ is a sufficient adjustment set, rather than just W,[3] we must additionally add X as an input to our conditional outcome model. More precisely, for CATE estimation, we define μ as follows:

[3] Active reading exercise: Why do we additionally add X to the adjustment set when we are interested in CATEs?

$$\mu(t, w, x) \triangleq \mathbb{E}[Y \mid T = t, W = w, X = x] \tag{7.7}$$

Then, we train a statistical model $\hat{\mu}$ to predict Y from (T, W, X). And this gives us the following COM estimator for the CATE $\tau(x)$:

$$\hat{\tau}(x) = \frac{1}{n_x} \sum_{i:x_i=x} \big(\hat{\mu}(1, w_i, x) - \hat{\mu}(0, w_i, x)\big) \tag{7.8}$$

Active reading exercise: Write down the causal estimand and statistical estimand that lead us to the estimator in Equation 7.8, and proof that they're equal under unconfoundedness and positivity. In other words, identify the CATE.

where n_x is the number of data points that have $x_i = x$. When we are interested in the IATE (CATE where X is all of the observed covariates), n_x is often 1, which simplifies our estimator to a simple difference between predictions:

$$\hat{\tau}(x_i) = \hat{\mu}(1, w_i, x_i) - \hat{\mu}(0, w_i, x_i) \tag{7.9}$$

Even, though IATEs are different from ITEs ($\tau(x_i) \neq \tau_i$), if we really want to give estimates for ITEs, it is relatively common to take this estimator as our estimator of the ITE τ_i as well:

$$\hat{\tau}_i = \hat{\tau}(x_i) = \hat{\mu}(1, w_i, x_i) - \hat{\mu}(0, w_i, x_i) \tag{7.10}$$

Though, this will likely be unreliable due to severe positivity violation.[4]

[4] Active reading exercise: Why is there a severe positivity violation here? Does this only apply in Equation 7.10 or also in Equation 7.9? What if there were multiple units with $x_i = x$?

The Many-Faced Estimator COM estimators have many different names in the literature. For example, they are often called G-computation estimators, parametric G-formula, or standardization in epidemiology and biostatistics. Because we are fitting a single statistical model for μ here, "COM estimator" is sometimes referred to as an "S-learner," where the "S" stands for "single."

7.3 Grouped Conditional Outcome Modeling (GCOM)

In order to get the estimate in Equation 7.6, we must train a model that predicts Y from (T, W). However, T is often one-dimensional, whereas W can be high-dimensional. But the input to $\hat{\mu}$ for t is the only thing that changes between the two terms inside the sum $\hat{\mu}(1, w_i) - \hat{\mu}(0, w_i)$. Imagine concatenating T to a 100-dimensional vector W and then feeding that through a neural network that we're using for $\hat{\mu}$. It seems reasonable that the network could ignore T while focusing on the other 100 dimensions of its input. This would result in an ATE estimate of zero. And, indeed, there is some evidence of COM estimators being biased toward zero [30].

[30]: Künzel et al. (2019), 'Metalearners for estimating heterogeneous treatment effects using machine learning'

So how can we ensure that the model $\hat{\mu}$ doesn't ignore T? Well, we can just train two different models $\hat{\mu}_1(w)$ and $\hat{\mu}_0(w)$ that model $\mu_1(w)$ and

$\mu_0(w)$, respectively, where

$$\mu_1(w) \triangleq \mathbb{E}[Y \mid T = 1, W = w] \quad \text{and} \quad \mu_0(w) \triangleq \mathbb{E}[Y \mid T = 0, W = w]\,. \tag{7.11}$$

Using two separate models for the values of treatment ensures that T cannot be ignored. To train these statistical models, we first group the data into a group where $T = 1$ and a group where $T = 0$. Then, we train $\hat{\mu}_1(w)$ to predict Y from W in the group where $T = 1$. And, similarly, we train $\hat{\mu}_0(w)$ to predict Y from W in the group where $T = 0$. This gives us a natural derivative of COM estimators (Equation 7.6), *grouped conditional outcome model (GCOM) estimators*:[5]

$$\hat{\tau} = \frac{1}{n} \sum_i \big(\hat{\mu}_1(w_i) - \hat{\mu}_0(w_i)\big) \tag{7.12}$$

[5] Künzel et al. [30] call a GCOM estimator a "T-learner" where the "T" is for "two" because it requires fitting two different models: $\hat{\mu}_1$ and $\hat{\mu}_0$.

And just as we saw, in Equation 7.8, we can add X as an input to $\hat{\mu}_1$ and $\hat{\mu}_0$ to get a GCOM estimator for the CATE $\tau(x)$:

$$\hat{\tau}(x) = \frac{1}{n_x} \sum_{i:x_i=x} \big(\hat{\mu}_1(w_i, x) - \hat{\mu}_0(w_i, x)\big) \tag{7.13}$$

While GCOM estimation seems to fix the problem that COM estimation can have regarding bias toward zero treatment effect, it does have an important downside. In COM estimation, we were able to make use of all the data when we estimate the single model $\hat{\mu}$. However, in *grouped* conditional outcome model estimation, we only use the $T = 1$ group to estimate $\hat{\mu}_1$, and we only use the $T = 0$ group to estimate $\hat{\mu}_0$. Importantly, we are missing out on making the most of our data by not using all of the data to estimate $\hat{\mu}_1$ and all of the data to estimate $\hat{\mu}_0$.

7.4 Increasing Data Efficiency

In this section, we'll cover two ways to address the problem of data efficiency that we mentioned is present in GCOM estimation at the end of the last section: TARNet (Section 7.4.1) and X-Learner (Section 7.4.2).

7.4.1 TARNet

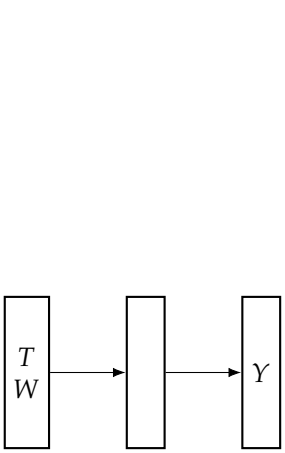
Consider that we're using neural networks for our statistical models; starting with that, we'll contrast, vanilla COM estimation, GCOM estimation, and TARNet. In vanilla COM estimation, the neural network is used to predict Y from (T, W) (see Figure 7.2a). This has the problem of potentially yielding ATE estimates that are biased toward zero, as the network might ignore the scalar T, especially when W is high-dimensional. We ensure that T can't be ignored in GCOM estimation by using two separate neural networks for the two treatment groups (Figure 7.2b). However, this is inefficient as we only use the treatment group data for training one network and the control group data for training the other network.

We can achieve a middle ground between vanilla COM estimation and GCOM estimation using Shalit et al. [31]'s TARNet. With TARNet, we use a single network that takes only W as input but then branches off into

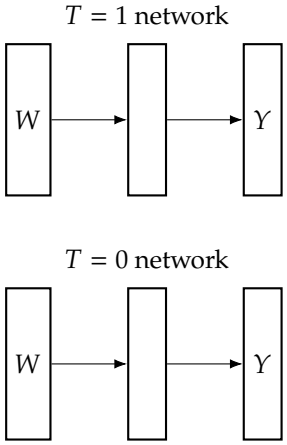
[31]: Shalit et al. (2017), 'Estimating individual treatment effect: generalization bounds and algorithms'

two separate heads (sub-networks) for each treatment group. We then use this model for $\mu(t, w)$ to get a COM estimator. This has the advantage of learning a treatment-agnostic representation (TAR) of W using *all* of the data while still forcing the model to not ignore T by branching into two heads for the different values of T. In other words, TARNet uses the knowledge we have about T (as a uniquely important variable) in its architecture. Still, the sub-networks for each of these heads are only trained with the data for the corresponding treatment group, rather than all of the data.[6]

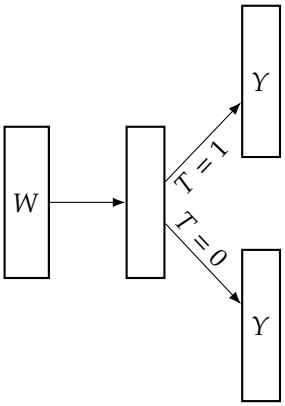
[6] Active reading exercise: Which parts of TARNet are like Figure 7.2a and which parts are like Figure 7.2b? What advantages/disadvantages do Figures 7.2a to 7.2c have relative to each other?



(a) A single neural network to model $\mu(t, w)$, used in vanilla COM estimation (Section 7.2).



(b) Two neural networks: a network to model $\mu_1(w)$ (top) and a network to model $\mu_0(w)$ (bottom), used in GCOM estimation (Section 7.3).



(c) TARNet [31]. A single neural network to model $\mu(t, w)$ that branches off into two heads: one for $T = 1$ and one for $T = 0$.

Figure 7.2: Coarse neural networks architectures for vanilla COM estimation (left), GCOM estimation (middle), and TARNet (right). In this figure, we use each arrow to denote a sub-network that has an arbitrary number of layers.

7.4.2 X-Learner

We just saw that one way to increase data efficiency relative to GCOM estimation is to use TARNet, a COM estimator that shares some qualities with GCOM estimators. However, TARNet still doesn't use all of the data for the full model (neural network). In this section, we will start with GCOM estimation and build on it to create a class of estimators that use all of the data for both models that are part of the estimators. An estimator in this class is known as an X-learner [30]. Unlike TARNet, X-learners are neither COM estimators nor GCOM estimators.

[30]: Künzel et al. (2019), 'Metalearners for estimating heterogeneous treatment effects using machine learning'

There are three steps to X-learning, and the **first step** is the exact same as what's used in GCOM estimation: estimate $\hat{\mu}_1(x)$ using the treatment group data and estimate $\hat{\mu}_0(x)$ using the control group data.[7] As before, this can be done with any models that minimize MSE. For simplicity, in this section, we'll be considering IATEs (X is all of the observed variables) where X satisfies the backdoor criterion (X contains W and no descendants of T).

[7] Recall that $\hat{\mu}_1(w)$ and $\hat{\mu}_0(w)$ are approximations of $\mathbb{E}[Y \mid T = 1, W = w]$ and $\mathbb{E}[Y \mid T = 0, W = w]$, respectively.

The **second step** is the most important part as it is both where we end up using all of the data for both models and where the "X" comes from. We specify $\hat{\tau}_{1,i}$ for the treatment group ITE estimates and $\hat{\tau}_{0,i}$ for the control

group ITE estimates:

$$\hat{\tau}_{1,i} = Y_i(1) - \hat{\mu}_0(x_i) \tag{7.14}$$
$$\hat{\tau}_{0,i} = \hat{\mu}_1(x_i) - Y_i(0) \tag{7.15}$$

Here, $\hat{\tau}_{1,i}$ is estimated using the treatment group outcomes and the imputed counterfactual that we get from $\hat{\mu}_0$. Similarly, $\hat{\tau}_{0,i}$ is estimated using the control group outcomes and the imputed counterfactual that we get from $\hat{\mu}_1$. If you draw a line between the observed potential outcomes and a line between the imputed potential outcomes, you can see the "X" shape. Importantly, this "X" tells us that each treatment group ITE estimate $\hat{\tau}_{1,i}$ uses both treatment group data (its observed potential outcome under treatment), *and* control group data (in $\hat{\mu}_0$). Similarly, $\hat{\tau}_{0,i}$ is estimated with data from both treatment groups.

However, each ITE estimate only uses a single data point from its corresponding treatment group. We can fix this by fitting a model $\hat{\tau}_1(x)$ to predict $\hat{\tau}_{1,i}$ from the corresponding treatment group x_i's. Finally, we have a model $\hat{\tau}_1(x)$ that was fit using all of the data (treatment group data just now and control group data when μ_0 was fit in step 1). Similarly, we can fit a model $\hat{\tau}_0(x)$ to predict $\hat{\tau}_{0,i}$ from the corresponding *control* group x_i's. The **output of step 2** is two different estimators for the IATE: $\hat{\tau}_1(x)$ and $\hat{\tau}_0(x)$.

Finally, in **step 3**, we combine $\hat{\tau}_1(x)$ and $\hat{\tau}_0(x)$ together to get our IATE estimator:

$$\hat{\tau}(x) = g(x)\,\hat{\tau}_0(x) + (1 - g(x))\,\hat{\tau}_1(x) \tag{7.16}$$

where $g(x)$ is some weighting function that produces values between 0 and 1. Künzel et al. [30] report that an estimate of the propensity score (introduced in next section) works well, but that choosing the constant function 0 or 1 also makes sense if the treatment groups are very different sizes. Or that choosing $g(x)$ to minimize the variance of $\hat{\tau}(x)$ could also be attractive.

[30]: Künzel et al. (2019), 'Metalearners for estimating heterogeneous treatment effects using machine learning'

Active reading exercise: In this section, we covered the X-learner for IATE estimation. What would an X-learner for more general CATE estimation (X is arbitrary and doesn't necessarily contain all confounders W) look like?

7.5 Propensity Scores

Given that the vector of variables W satisfies the backdoor criterion (or, equivalently, that $(Y(1), Y(0)) \perp\!\!\!\perp T \mid W$), we might wonder if it is really necessary to condition on that whole vector to isolate causal association, especially when W is high-dimensional. It turns out that it isn't. If W satisfies unconfoundedness and positivity, then we can actually get away with only conditioning on the *scalar* $P(T = 1 \mid W)$. We'll let $e(w)$ denote $P(T = 1 \mid W = w)$, as we'll refer to $e(w)$ as the *propensity score* since it is the propensity for (probability of) receiving treatment given that W is w. The magic of being able to condition on the scalar $e(W)$ in the place of the vector W is due to Rosenbaum and Rubin [32]'s propensity score theorem:

[32]: Rosenbaum and Rubin (1983), 'The central role of the propensity score in observational studies for causal effects'

Theorem 7.1 (Propensity Score Theorem) *Given positivity, unconfoundedness given W implies unconfoundedness given the propensity score $e(W)$.*

Equivalently,

$$(Y(1), Y(0)) \perp\!\!\!\perp T \mid W \implies (Y(1), Y(0)) \perp\!\!\!\perp T \mid e(W)\,. \tag{7.17}$$

We provide a more traditional mathematical proof in Appendix A.2 and give a graphical proof here. Consider the graph in Figure 7.3. Because the edge from W to T is a symbol for the mechanism $P(T \mid W)$ and because the propensity score completely describes that distribution ($P(T = 1 \mid W) = e(W)$), we can think of the propensity score as a full mediator of the effect of W on T. This means that we can redraw this graph with $e(W)$ situated between W and T. And in this redrawned graph in Figure 7.4, we can see that $e(W)$ blocks all backdoor paths that W blocks, so $e(W)$ must be a sufficient adjustment set if W is. Therefore, we have a graphical proof of the propensity score theorem using the backdoor adjustment (Theorem 4.2).

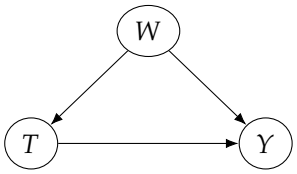


Figure 7.3: Simple graph where W satisfies the backdoor criterion

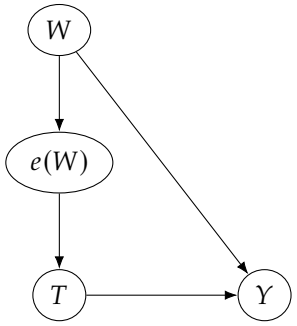


Figure 7.4: Graph illustrating that $e(W)$ blocks the backdoor path(s) that W blocks.

Importantly, this theorem means that we can swap in $e(W)$ in place of W wherever we are adjusting for W in a given estimator in this chapter. For example, this seems very useful when W is high-dimensional.

Recall The Positivity-Unconfoundedness Tradeoff from Section 2.3.4. As we condition on more non-collider-bias-inducing variables, we decrease confounding. However, this comes at the cost of decreasing overlap because the W in $P(T = 1 \mid W)$ becomes higher and higher dimensional. The propensity score seems to allow us to magically fix that issue since the $e(W)$ remains a scalar, even as W grows in dimension. Fantastic, right?

Well, unfortunately, we usually don't have access to $e(W)$. Rather, the best we can do is model it. We do this by training a model to predict T from W. For example, logistic regression (logit model) is very commonly used to do this. And because this model is fit to the high-dimensional W, in some sense, we have just shifted the positivity problem to our model for $e(W)$.

7.6 Inverse Probability Weighting (IPW)

What if we could resample the data in a way to make it so that association is causation? This is the motivation behind creating "pseudo-populations" that are made up of reweighted versions of the observed population. To get to this, let's recall why association is not causation in general.

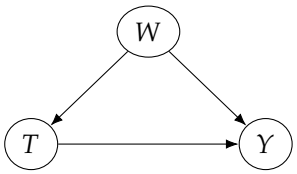


Figure 7.5: Simple graph where W confounds the effect of T on Y

Association is not causation in the graph in Figure 7.5 because W is a common cause of T and Y. In other words, the mechanism that generates T depends on W, and the mechanism that generates Y depends on W. Focusing on the mechanism that generates T, we can write this mathematically as $P(T \mid W) \neq P(T)$. It turns out that we can reweight the data to get a *pseudo-population* where $P(T \mid W) = P(T)$ or $P(T \mid W)$ equals some constant; the important part is that we make T independent of W. The corresponding graph for such a pseudo-population has no edge from W to T because T does not depend on W; we depict this in Figure 7.6.

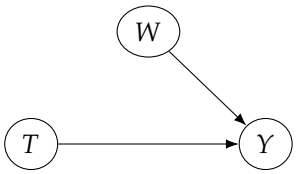


Figure 7.6: Effective graph for pseudo-population that we get by reweighting the data generated according to the graph in Figure 7.5 using inverse probability weighting.

It turns out that the propensity score is key to this reweighting. All we have to do is reweight each data point with treatment T and confounders

W by its inverse probability of receiving its value of treatment given that it has its value of W. This is why this technique is called *inverse probability weighting (IPW)*. For individuals that received treatment 1, this weight is $\frac{1}{e(W)}$, and for individuals that received treatment 0, this weight is $\frac{1}{1-e(W)}$.[8] If the treatment were continuous, the weight would be $\frac{1}{P(T|W)}$, which happens to also be the reciprocal of the generalization of the propensity score to continuous treatment.

[8] Active reading exercise: Why is the denominator $1 - e(W)$ when $T = 0$. Hint: recall the precise definition of $e(W)$.

Why does what we described in the above paragraph work? Well, recall that our goal is to undo confounding by "removing" the edge that goes from W to T (i.e. move from Figure 7.5 to Figure 7.6). And the mechanism that edge describes is $P(T \mid W)$. By weighting the data points by $\frac{1}{P(T|W)}$, we are effectively canceling it out. That's the intuition. Formally, we have the following identification equation:

$$\mathbb{E}[Y(t)] = \mathbb{E}\left[\frac{\mathbb{1}(T = t)Y}{P(t \mid W)}\right] \tag{7.18}$$

where $\mathbb{1}(T = t)$ is an indicator random variable that takes on the value 1 if $T = t$ and 0 otherwise. We provide a proof of Equation 7.18 using the familiar adjustment formula $\mathbb{E}[Y(t)] = \mathbb{E}[\mathbb{E}[Y \mid t, W]]$ (Theorem 2.1) in Appendix A.3.

Assuming binary treatment, the following identification equation for the ATE follows from Equation 7.18:

$$\tau \triangleq \mathbb{E}[Y(1) - Y(0)] = \mathbb{E}\left[\frac{\mathbb{1}(T = 1)Y}{e(W)}\right] - \mathbb{E}\left[\frac{\mathbb{1}(T = 0)Y}{1 - e(W)}\right] \tag{7.19}$$

Now that we have a statistical estimand in the form of IPW, we can get an IPW estimator. Replacing expectations by empirical means and $e(W)$ by a propensity score model $\hat{e}(W)$, we get the following equivalent formulations of the basic IPW estimator[9] for the ATE:

$$\hat{\tau} = \frac{1}{n}\sum_i \left(\frac{\mathbb{1}(t_i = 1)y_i}{\hat{e}(w_i)} - \frac{\mathbb{1}(t_i = 0)y_i}{1 - \hat{e}(w_i)}\right) \tag{7.20}$$

$$= \frac{1}{n_1}\sum_{i:t_i=1} \frac{y_i}{\hat{e}(w_i)} - \frac{1}{n_0}\sum_{i:t_i=0} \frac{y_i}{1 - \hat{e}(w_i)} \tag{7.21}$$

where n_1 and n_0 are the number of treatment group units and control group units, respectively.

[9] This estimator is originally from Horvitz and Thompson [33].

[33]: Horvitz and Thompson (1952), 'A Generalization of Sampling Without Replacement from a Finite Universe'

Active reading exercise: What would be the corresponding formulations of the basic IPW estimator for $\mathbb{E}[Y(t)]$?

Weight Trimming As you can see in Equations 7.20 and 7.21, if the propensity scores are very close to 0 or 1, the estimates will blow up. In order to prevent this, it is not uncommon to trim the propensity scores that are less than ϵ to ϵ and those that are greater than $1 - \epsilon$ to $1 - \epsilon$ (effectively trimming the weights to be no larger than $\frac{1}{\epsilon}$), though this introduces its own problems such as bias.

CATE Estimation We can extend the ATE estimator in Equation 7.20 to get an IPW estimator for the CATE $\tau(x)$ by just restricting to the data points where $x_i = x$:

$$\hat{\tau}(x) = \frac{1}{n_x}\sum_{i:x_i=x} \left(\frac{\mathbb{1}(t_i = 1)y_i}{\hat{e}(w_i)} - \frac{\mathbb{1}(t_i = 0)y_i}{1 - \hat{e}(w_i)}\right) \tag{7.22}$$

where n_x is the number of data points with $x_i = x$. However, the estimator in Equation 7.22 may quickly run into the problem of using very small amounts of data, leading to high variance. More general CATE estimation with IPW estimators is more complex and outside the scope of this book. See, for example, Abrevaya et al. [34] and references therein.

[34]: Abrevaya et al. (2015), 'Estimating Conditional Average Treatment Effects'

7.7 Doubly Robust Methods

We've seen that we can estimate causal effects by modeling $\mu(t, w) \triangleq \mathbb{E}[Y \mid t, w]$ (Sections 7.2 to 7.4) or by modeling $e(w) \triangleq P(T = 1 \mid w)$ (Section 7.6). What if we modeled both $\mu(t, w)$ *and* $e(w)$? Well, we can and estimators that do this are sometimes *doubly robust*. A doubly robust estimator has the property that it is a consistent[10] estimator of τ if either $\hat{\mu}$ is a consistent estimator of μ *or* $\hat{e}$ is a consistent estimate of e. In other words, only one of $\hat{\mu}$ and $\hat{e}$ needs to be well-specified. Additionally, the rate at which a doubly robust estimator converges to τ is the *product* of the rate at which $\hat{\mu}$ converges to μ and the rate at which $\hat{e}$ converges to e. This makes double robustness is very useful when we are using flexible machine learning models in high-dimensions because, in this setting, each of our individual models ($\hat{\mu}$ and $\hat{e}$) converge more slowly that the ideal rate of $n^{-1/2}$.

[10] An estimator is consistent if it converges in probability to its estimand as the number of samples n grows.

However, there is some controversy over how well doubly robust methods work in practice if not at least one of $\hat{\mu}$ or $\hat{e}$ is well-specified [35]. Though, this might be contested as we get better at using doubly robust estimators with flexible machine learning models (see, e.g., [36]). Meanwhile, the estimators that currently seem to do the best all flexibly model μ (unlike pure IPW estimators) [37]. This is why we began this chapter with estimators that model μ and dedicated several sections to such estimators.

[35]: Kang and Schafer (2007), 'Demystifying Double Robustness: A Comparison of Alternative Strategies for Estimating a Population Mean from Incomplete Data'

[36]: Zivich and Breskin (2020), *Machine learning for causal inference: on the use of cross-fit estimators*

[37]: Dorie et al. (2019), 'Automated versus Do-It-Yourself Methods for Causal Inference: Lessons Learned from a Data Analysis Competition'

Doubly robust methods are largely outside the scope of this book, so we refer the reader to an introduction by Seaman and Vansteelandt [38], along with other seminal works on the topic: [39–41]. Additionally, there is a large body of doubly robust work on methods that have performed reasonably well in competitions [37]; this category is known as *targeted maximum likelihood estimation (TMLE)*. [42–44].

[38]: Seaman and Vansteelandt (2018), 'Introduction to Double Robust Methods for Incomplete Data'

[39]: Tsiatis (2007), *Semiparametric theory and missing data*

[40]: Robins et al. (1994), 'Estimation of Regression Coefficients When Some Regressors are not Always Observed'

[41]: Bang and Robins (2005), 'Doubly Robust Estimation in Missing Data and Causal Inference Models'

[42]: Van Der Laan and Rubin (2006), 'Targeted maximum likelihood learning'

[43]: Schuler and Rose (2017), 'Targeted Maximum Likelihood Estimation for Causal Inference in Observational Studies'

[44]: Van der Laan and Rose (2011), *Targeted learning: causal inference for observational and experimental data*

7.8 Other Methods

As this chapter is only an introduction to estimation in causal inference, there are some methods that we've entirely left out. We'll briefly describe some of the most popular ones in this section.

Matching In matching methods, we try to match units in the treatment group with units in the control group and throw away the non-matches to create comparable groups. We can match in raw covariate space, coarsened covariate space, or propensity score space. There are different distance functions for deciding how close two units are. Furthermore, there are different criteria for deciding whether a given distance is close enough to count as a match (one criterion requires an exact match), how many matches each treatment group unit can have, how many matches

each control group unit can have, etc. See, for example, Stuart [45] for a review.

[45]: Stuart (2010), 'Matching Methods for Causal Inference: A Review and a Look Forward'

Double Machine Learning In double machine learning, we fit three models in two stages: two in the first stage and a final model in the second stage. First stage:

1. Fit a model to predict Y from W to get the predicted $\hat{Y}$.[11]
2. Fit a model to predict T from W to get the to get the predicted $\hat{T}$.

[11] Active reading exercise: How is this model different from $\hat{\mu}$?

Then, in the second stage, we "partial out" W by looking at $Y - \hat{Y}$ and $T - \hat{T}$. In a sense, we have deconfounded the effect of treatment on the outcome with this partialling out. Then, we fit a model to predict $Y - \hat{Y}$ from $T - \hat{T}$. This gives us our causal effect estimates. For more on this topic, see, for example [46–49].

[46]: Chernozhukov et al. (2018), 'Double/debiased machine learning for treatment and structural parameters'
[47]: Felton (2018), *Chernozhukov et al. on Double / Debiased Machine Learning*
[48]: Syrgkanis (2019), *Orthogonal/Double Machine Learning*
[49]: Foster and Syrgkanis (2019), *Orthogonal Statistical Learning*

Causal Trees and Forests Another popular estimation method is to recursively partition the data into subsets that have the same treatment effects [50]. This forms a *causal tree* where the leaves are subsets of the population with similar causal effects. Since random forests generally perform better than decision trees, it would be great if this kind of strategy can be extended to random forests. And it can. This extensions is known as *causal forests* [51], which are part of more general class known as *generalized random forests* [52]. Importantly, these methods were developed with the goal in mind of yielding valid confidence intervals for the estimates.

[50]: Athey and Imbens (2016), 'Recursive partitioning for heterogeneous causal effects'
[51]: Wager and Athey (2018), 'Estimation and Inference of Heterogeneous Treatment Effects using Random Forests'
[52]: Athey et al. (2019), 'Generalized random forests'

7.9 Concluding Remarks

7.9.1 Confidence Intervals

So far, in this chapter, we have only discussed point estimates for causal effects. We haven't discussed how we can gauge our uncertainty due to data sampling. We haven't discussed how to calculate confidence intervals on these estimates. This is a machine learning perspective, after all; who cares about confidence intervals... Jokes aside, because we are allowing for arbitrary machine learning models in all of the estimators we discuss, it is actually quite difficult to get valid confidence intervals.

Bootstrapping One way to get confidence intervals is to use bootstrapping. With bootstrapping, we repeat the causal effect estimation process many times, each time with a different sample (with replacement) from our data. This allows us to build an empirical distribution for the estimate. We can then compute whatever confidence interval we like from that empirical distribution. Unfortunately, bootstrapped confidence intervals are not always valid. For example, if we take a bootstrapped 95% confidence interval, it might not contain the true value (estimand) 95% of the time.

Specialized Models Another way to get confidence intervals is to analyze very specific models, rather than allowing for arbitrary models Linear models are the simplest example of this; it is easy to get confidence intervals in linear models. Similarly, if we use a linear model as the second stage model in double machine learning, we can get confidence intervals. Noticeably, causal trees and causal forests were developed with the goal in mind of getting confidence intervals.

7.9.2 Comparison to Randomized Experiments

You might read somewhere that some of these adjustment techniques ensure that we've addressed confounding and isolated a causal effect. Of course, this is not true when there is unobserved confounding. These methods only address *observed* confounding. If there are any unobserved confounders, these methods don't fix that like randomization does (Chapter 5). These adjustment methods aren't magic. And it's hard to know when it is reasonable to assume we've observed all confounders. That's why it is important to run a sensitivity analysis where we gauge how robust our causal effect estimates are to unobserved confounding. This is the topic of the next chapter.

Active reading exercise: What kind of estimator did we use back in the estimation examples in Sections 2.5 and 4.6.2?

Unobserved Confounding: Bounds and Sensitivity Analysis

8

All of the methods in Chapter 7 assume that we don't have any unobserved confounding. However, unconfoundedness is an untestable assumption. In observational studies, there could also be some unobserved confounder(s). Therefore, we'd like to know how robust our estimates are to unobserved confounding. The first way we can do is by getting an upper and lower bound on the causal effect using credible assumptions (Section 8.1). Another way we can do this is by simulating how strong the confounder's effect on the treatment and the confounder's effect on the outcome need to be to make the true causal effect substantially different from our estimate (Section 8.2).

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(a) No unobserved confounding **(b)** Unobserved confounding (U)

Figure 8.1: On the left, we have the setting we have considered up till now, where we have unconfoundedness / the backdoor criterion. On the right, we have a simple graph where the unobserved confounder U make the causal effect of T on Y not identifiable.

8.1 Bounds

There is a tradeoff between how realistic or credible our assumptions are and how precise of an identification result we can get. Manski [53] calls this "*The Law of Decreasing Credibility*: the credibility of inference decreases with the strength of the assumptions maintained."

[53]: Manski (2003), *Partial Identification of Probability Distributions: Springer Series in Statistics*

Depending on what assumptions we are willing to make, we can derive various nonparametric bounds on causal effects. We have seen that if we are willing to assume unconfoundedness (or some causal graph in which the causal effect is identifiable) and positivity, we can identify a single point for the causal effect. However, this might be unrealistic. For example, there could always be unobserved confounding in observational studies.

This is what motivates Charles Manski's work on bounding causal effects [53–60]. This gives us an interval that the causal effect must be in, rather than telling us exactly what point in that interval the causal effect must be. In this section, we will give an introduction to these nonparametric bounds and how to derive them.

[54]: Manski (1989), 'Anatomy of the Selection Problem'
[55]: Manski (1990), 'Nonparametric Bounds on Treatment Effects'
[56]: Manski (1993), 'Identification Problems in the Social Sciences'
[57]: Manski (1994), 'The selection problem'
[58]: Manski (1997), 'Monotone Treatment Response'
[59]: Manski and Pepper (2000), 'Monotone Instrumental Variables: With an Application to the Returns to Schooling'
[53]: Manski (2003), *Partial Identification of Probability Distributions: Springer Series in Statistics*
[60]: Manski (2013), *Public Policy in an Uncertain World*

The assumptions that we consider are weaker than unconfoundedness, so they give us intervals that the causal effect must fall in (under these

assumptions). If we assumed the stronger assumption of unconfoundedness, these intervals would collapse to a single point. This illustrates the law of decreasing credibility.

8.1.1 No-Assumptions Bound

Say all we know about the potential outcomes $Y(0)$ and $Y(1)$ is that they are between 0 and 1. Then, the maximum value of an ITE $Y_i(1) - Y_i(0)$ is 1 (1 - 0), and the minimum is -1 (0 - 1):

$$-1 \leq Y_i(1) - Y_i(0) \leq 1 \qquad \text{if } \forall t,\ 0 \leq Y(t) \leq 1 \tag{8.1}$$

So we know that all ITEs must be in an interval of length 2. Because all the ITEs must fall inside this interval of length 2, the ATE must also fall inside this interval of length 2. Interestingly, for ATEs, it turns out that we can cut the length of this interval in half without making any assumptions (beyond the min/max value of outcome); the interval that the ATE must fall in is only of length 1.

We'll show this result from Manski [55] in the more general scenario where the outcome is bounded between a and b:

[55]: Manski (1990), 'Nonparametric Bounds on Treatment Effects'

Assumption 8.1 (Bounded Potential Outcomes)

$$\forall t,\ a \leq Y(t) \leq b \tag{8.2}$$

By the same reasoning as above, this implies the following bounds on the ITEs and ATE:

Active reading exercise: Ensure you follow how we get to these bounds.

$$a - b \leq Y_i(1) - Y_i(0) \leq b - a \tag{8.3}$$
$$a - b \leq \mathbb{E}[Y(1) - Y(0)] \leq b - a \tag{8.4}$$

These are intervals of length $(b-a)-(a-b) = 2(b-a)$. And the bounds for the ITEs cannot be made tighter without further assumptions. However, seemingly magically, we can halve the length of the interval for the ATE. To see this, we rewrite the ATE as follows:

$$\begin{aligned}\mathbb{E}[Y(1) - Y(0)] &= \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] &(8.5)\\ &= P(T=1)\,\mathbb{E}[Y(1) \mid T=1] + P(T=0)\,\mathbb{E}[Y(1) \mid T=0] \\ &\quad - P(T=1)\,\mathbb{E}[Y(0) \mid T=1] - P(T=0)\,\mathbb{E}[Y(0) \mid T=0] &(8.6)\end{aligned}$$

We immediately recognize the first and last terms as friendly conditional expectations that we can estimate from observational data:

Active reading exercise: What assumption are we using here?

$$\begin{aligned}&= P(T=1)\,\mathbb{E}[Y \mid T=1] + P(T=0)\,\mathbb{E}[Y(1) \mid T=0] \\ &\quad - P(T=1)\,\mathbb{E}[Y(0) \mid T=1] - P(T=0)\,\mathbb{E}[Y \mid T=0] &(8.7)\end{aligned}$$

Because this is such an important decomposition, we'll give it a name and box before moving on with the bound derivation. We will call this the *observational-counterfactual decomposition* (of the ATE). Also, to have

a bit more concise notation, we'll use $\pi \triangleq P(T = 1)$ moving forward.

Proposition 8.1 (Observational-Counterfactual Decomposition)

$$\begin{aligned}\mathbb{E}[Y(1) - Y(0)] = \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,\mathbb{E}[Y(1) \mid T = 0] \\ - \pi\,\mathbb{E}[Y(0) \mid T = 1] - (1 - \pi)\,\mathbb{E}[Y \mid T = 0]\end{aligned} \tag{8.8}$$

Unfortunately, $\mathbb{E}[Y(1) \mid T = 0]$ and $\mathbb{E}[Y(0) \mid T = 1]$ are counterfactual. However, we know that they're bounded between a and b. Therefore, we get an upper bound on the complete expression by letting the quantity that's being added ($\mathbb{E}[Y(1) \mid T = 0]$) equal b and letting the quantity that's being subtracted ($\mathbb{E}[Y(0) \mid T = 1]$) equal a. Similarly, we can get a lower bound by letting the term that's being added equal a and the term that's being subtracted equal b.

Proposition 8.2 (No-Assumptions Bound) *Let π denote $P(T = 1)$, where T is a binary random variable. Given that the outcome Y is bounded between a and b (Assumption 8.1), we have the following upper and lower bounds on the ATE:*

$$\mathbb{E}[Y(1) - Y(0)] \leq \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,b - \pi\,a - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \tag{8.9}$$

$$\mathbb{E}[Y(1) - Y(0)] \geq \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,a - \pi\,b - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \tag{8.10}$$

Importantly, the length of this interval is $b - a$, half the length of the naive interval that we saw in Equation 8.4. We can see this by subtracting the lower bound from the upper bound:

$$\begin{aligned}&\pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,b - \pi\,a - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \\ -&(\pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,a - \pi\,b - (1 - \pi)\,\mathbb{E}[Y \mid T = 0]) \\ &= (1 - \pi)\,b + \pi\,b - \pi\,a - (1 - \pi)\,a && (8.11) \\ &= b - a && (8.12)\end{aligned}$$

This is sometimes referred to as the "no-assumptions bound" because we made no assumptions other than that the outcomes are bounded. If the outcomes are not bounded, then the ATE and ITEs can be anywhere between $-\infty$ and ∞.

Running Example

Consider that we know that the outcomes are bounded between 0 and 1 (e.g., because we're in a binary outcomes setting). This means that the ITEs and must be bounded between -1 (0 - 1) and 1 (1 - 0), which means that the ATE must also be bounded between -1 and 1. For this example, also consider that $\pi = 0.3$, $\mathbb{E}[Y \mid T = 1] = .9$, and $\mathbb{E}[Y \mid T = 0] = .2$.[1] Then, by plugging these in to Equations 8.9 and 8.10, we get the following bounds on the ATE:

$$\mathbb{E}[Y(1) - Y(0)] \leq (.3)(.9) + (1 - .3)(1) - (.3)(0) - (1 - .3)(.2) \tag{8.13}$$

$$\mathbb{E}[Y(1) - Y(0)] \geq (.3)(.9) + (1 - .3)(0) - (.3)(1) - (1 - .3)(.2) \tag{8.14}$$

[1] Active reading exercise: How would we estimate these conditional expectations?

$$-0.17 \leq \mathbb{E}[Y(1) - Y(0)] \leq 0.83 \tag{8.15}$$

Notice that this interval is of length 1 ($b - a = 1$), half the length of the naive interval $-1 \leq \mathbb{E}[Y(1) - Y(0)] \leq 1$ (Equation 8.4). We will use this running example throughout Section 8.1.

Active reading exercises:

1. What kind of bounds can we get for CATEs $\mathbb{E}[Y(1) - Y(0) \mid X]$, assuming we have positivity? What goes wrong if we don't have positivity?
2. Say the potential outcomes are bounded in different ways: $a_1 \leq Y(1) \leq b_1$ and $a_0 \leq Y(0) \leq b_0$. Derive the corresponding no-assumptions bounds in this more general setting.

The bounds in Proposition 8.2 are as tight as we can get without further assumptions. Unfortunately, the corresponding interval always contains 0,[2] which means that we cannot use this bound to distinguish "no causal effect" from "causal effect." Can we get tighter bounds?

[2] To see why the no-assumptions bound always contains zero, consider what we would need for it to not contain zero: we would either need the upper bound to be less than zero or the lower bound to be greater than zero. However, this cannot be the case. To see why, note that the minimum upper bound is achieved when $\mathbb{E}[Y \mid T = 1] = a$ and $\mathbb{E}[Y \mid T = 0] = b$, which gives us an (inclusive) upper bound of zero. Same with the lower bound. Active reading exercise: Show that the maximum lower bound is 0.

In order to bound the ATE, we must have some information about the counterfactual part of this decomposition. We can easily estimate the observational part from data. In the no-assumptions bound (Proposition 8.2), all we assumed is that the outcomes are bounded by a and b. If we make more assumptions, we can get smaller intervals. In the next few sections, we will cover some assumptions that are sometimes fairly reasonable, depending on the setting, and what tighter bounds these assumptions get us. The general strategy we will use for all of them is to start with the observational-counterfactual decomposition of the ATE (Proposition 8.1),

$$\begin{aligned}\mathbb{E}[Y(1) - Y(0)] = \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,\mathbb{E}[Y(1) \mid T = 0] \\ - \pi\,\mathbb{E}[Y(0) \mid T = 1] - (1 - \pi)\,\mathbb{E}[Y \mid T = 0]\,,\end{aligned} \tag{8.8 revisited}$$

and get smaller intervals by bounding the counterfactual parts using the different assumptions we make.

The intervals we will see in the next couple of subsections will all contain zero. We won't see an interval that is purely positive or purely negative until Section 8.1.4, so feel free to skip to that section if you only want to see those intervals.

8.1.2 Monotone Treatment Response

For our first assumption beyond assuming bounded outcomes, consider that we find ourselves in a setting where it is feasible that the treatment can only help; it can't hurt. This is the setting that Manski [58] considers in context. In this setting, we can justify the *monotone treatment response (MTR)* assumption:

[58]: Manski (1997), 'Monotone Treatment Response'

Assumption 8.2 (Nonnegative Monotone Treatment Response)

$$\forall i \; Y_i(1) \geq Y_i(0) \tag{8.16}$$

This means that every ITE is nonnegative, so we can bring our lower bound on the ITEs up from $a - b$ (Equation 8.3) to 0. So, intuitively, this should mean that our lower bound on the ATE should move up to 0. And we will now see that this is the case.

Now, rather than lower bounding $\mathbb{E}[Y(1) \mid T = 0]$ with a and $-\mathbb{E}[Y(0) \mid T = 1]$ with $-b$, we can do better. Because the the treatment only helps, $\mathbb{E}[Y(1) \mid T = 0] \geq \mathbb{E}[Y(0) \mid T = 0] = \mathbb{E}[Y \mid T = 0]$, so we can lower bound $\mathbb{E}[Y(1) \mid T = 0]$ with $\mathbb{E}[Y \mid T = 0]$. Similarly, $-\mathbb{E}[Y(0) \mid T = 1] \geq -\mathbb{E}[Y(1) \mid T = 1] = \mathbb{E}[Y \mid T = 1]$ (since multiplying by a negative flips the inequality), so we can lower bound $-\mathbb{E}[Y(0) \mid T = 1]$ with $-\mathbb{E}[Y \mid T = 1]$. Therefore, we can improve on the no-assumptions lower bound[3] to get 0, as our intuition suggested:

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0)] &= \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,\mathbb{E}[Y(1) \mid T = 0] \\
&\quad - \pi\,\mathbb{E}[Y(0) \mid T = 1] - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] && \text{(8.8 revisited)} \\
&\geq \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \\
&\quad - \pi\,\mathbb{E}[Y \mid T = 1] - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] && (8.17) \\
&= 0 && (8.18)
\end{aligned}$$

[3] Recall that by only assuming that outcomes are bounded between a and b, we get the no-assumptions lower bound (Proposition 8.2):

$$\begin{aligned}
&\mathbb{E}[Y(1) - Y(0)] \\
&\quad \geq \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,a \\
&\quad\quad - \pi\,b - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \\
&\quad\quad\quad \text{(8.10 revisited)}
\end{aligned}$$

Proposition 8.3 (Nonnegative MTR Lower Bound) *Under the nonnegative MTR assumption, the ATE is bounded from below by 0. Mathematically,*

$$\mathbb{E}[Y(1) - Y(0)] \geq 0 \tag{8.19}$$

Running Example The no-assumptions upper bound[4] still applies here, so in our running example from Section 8.1.1 where $\pi = .3$, $\mathbb{E}[Y \mid T = 1] = .9$, and $\mathbb{E}[Y \mid T = 0] = .2$, our ATE interval improves from $[-0.17, 0.83]$ (Equation 8.15) to $[0, 0.83]$.

[4] Recall the no-assumptions upper bound (Proposition 8.2):

$$\begin{aligned}
&\mathbb{E}[Y(1) - Y(0)] \\
&\quad \leq \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,b \\
&\quad\quad - \pi\,a - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \\
&\quad\quad\quad \text{(8.9 revisited)}
\end{aligned}$$

Alternatively, say the treatment can only hurt people; it can't help them (e.g. a gunshot wound only hurts chances of staying alive). In those cases, we would have the *nonpositive* monotone treatment response assumption and the nonpositive MTR *upper* bound:

Assumption 8.3 (Nonpositive Monotone Treatment Response)

$$\forall i\ Y_i(1) \leq Y_i(0) \tag{8.20}$$

Proposition 8.4 (Nonpositive MTR Upper Bound) *Under the nonpositive MTR assumption, the ATE is bounded from above by 0. Mathematically,*

$$\mathbb{E}[Y(1) - Y(0)] \leq 0 \tag{8.21}$$

Active reading exercise: Prove Proposition 8.4.

Running Example And in this setting, the no-assumptions lower bound[5] still applies. That means that the ATE interval in our example improves from $[-0.17, 0.83]$ (Equation 8.15) to $[-0.17, 0]$.

[5] Recall the no-assumptions lower bound (Proposition 8.2):

$$\begin{aligned}
&\mathbb{E}[Y(1) - Y(0)] \\
&\quad \geq \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,a \\
&\quad\quad - \pi\,b - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \\
&\quad\quad\quad \text{(8.10 revisited)}
\end{aligned}$$

Active reading exercise: What is the ATE interval if we assume *both* nonnegative MTR *and* nonpositive MTR? Does this make sense, intuitively?

8.1.3 Monotone Treatment Selection

The next assumption that we'll consider is the assumption that the people who selected treatment would have better outcomes than those who didn't select treatment, under either treatment scenario. Manski and Pepper [59] introduced this as the *monotone treatment selection (MTS)* assumption.

[59]: Manski and Pepper (2000), 'Monotone Instrumental Variables: With an Application to the Returns to Schooling'

Assumption 8.4 (Monotone Treatment Selection)

$$\mathbb{E}[Y(1) \mid T = 1] \geq \mathbb{E}[Y(1) \mid T = 0] \tag{8.22}$$
$$\mathbb{E}[Y(0) \mid T = 1] \geq \mathbb{E}[Y(0) \mid T = 0] \tag{8.23}$$

As Morgan and Winship [12, Section 12.2.2] point out, you might think of this as positive self-selection. Those who generally get better outcomes self-select into the treatment group. Again, we start with the observational-counterfactual decomposition, and we now obtain an upper bound using the MTS assumption (Assumption 8.4):

[12]: Morgan and Winship (2014), *Counterfactuals and Causal Inference: Methods and Principles for Social Research*

Proposition 8.5 (Monotone Treatment Selection Upper Bound) *Under the MTS assumption, the ATE is bounded from above by the associational difference. Mathematically,*

$$\mathbb{E}[Y(1) - Y(0)] \leq \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0] \tag{8.24}$$

Proof.

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0)] &= \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,\mathbb{E}[Y(1) \mid T = 0] \\
&\quad - \pi\,\mathbb{E}[Y(0) \mid T = 1] - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \\
&\qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad \text{(8.8 revisited)} \\
&\leq \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,\mathbb{E}[Y \mid T = 1] \\
&\quad - \pi\,\mathbb{E}[Y \mid T = 0] - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \qquad (8.25) \\
&= \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0] \qquad (8.26)
\end{aligned}$$

where Equation 8.25 followed from the fact that (a) Equation 8.22 of the MTS assumption allows us to upper bound $\mathbb{E}[Y(1) \mid T = 0]$ by $\mathbb{E}[Y(1) \mid T = 1] = \mathbb{E}[Y(1) \mid T = 1]$ and (b) Equation 8.23 of the MTS assumption allows us to upper bound $-\mathbb{E}[Y(0) \mid T = 1]$ by $-\mathbb{E}[Y \mid T = 0]$. □

Running Example Recall our running example from Section 8.1.1 where $\pi = .3$, $\mathbb{E}[Y \mid T = 1] = .9$, and $\mathbb{E}[Y \mid T = 0] = .2$. The MTS assumption gives us an upper bound, and we still have the no-assumptions lower bound.[6] That means that the ATE interval in our example improves from $[-0.17, 0.83]$ (Equation 8.15) to $[-0.17, 0.7]$.

Both MTR and MTS Then, we can combine the nonnegative MTR assumption (Assumption 8.2) with the MTS assumption (Assumption 8.4) to get the lower bound in Proposition 8.3 and the upper bound in Proposition 8.5, respectively. In our running example, this yields the following interval for the ATE: $[0, 0.7]$.

[6] Recall the no-assumptions lower bound (Proposition 8.2):

$$\begin{aligned}
&\mathbb{E}[Y(1) - Y(0)] \\
&\quad \geq \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,a \\
&\qquad - \pi\,b - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \\
&\qquad\qquad\qquad\qquad \text{(8.10 revisited)}
\end{aligned}$$

Intervals Contain Zero Although bounds from the MTR and MTS assumptions can be useful for ruling out very large or very small causal effects, the corresponding intervals still contain zero. This means that these assumptions are not enough to identify whether there is an effect or not.

8.1.4 Optimal Treatment Selection

We now consider what we will call the *optimal treatment selection (OTS)* assumption from Manski [55]. This assumption means that the individuals always receive the treatment that is best for them (e.g. if an expert doctor is deciding which treatment to give people). We write this mathematically as follows:

[55]: Manski (1990), 'Nonparametric Bounds on Treatment Effects'

Assumption 8.5 (Optimal Treatment Selection)

$$T_i = 1 \implies Y_i(1) \geq Y_i(0)\,, \qquad T_i = 0 \implies Y_i(0) > Y_i(1) \tag{8.27}$$

From the OTS assumption, we know that

$$\mathbb{E}[Y(1) \mid T = 0] \leq \mathbb{E}[Y(0) \mid T = 0] = \mathbb{E}[Y \mid T = 0]\,. \tag{8.28}$$

Therefore, we can give an upper bound, by upper bounding $\mathbb{E}[Y(1) \mid T = 0]$ with $\mathbb{E}[Y \mid T = 0]$ and upper bounding $-\mathbb{E}[Y(0) \mid T = 1]$ with $-a$ (same as in the no-assumptions upper bound[7]):

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0)] &= \pi\,\mathbb{E}[Y \mid T = 1] + (1-\pi)\,\mathbb{E}[Y(1) \mid T = 0] \\
&\quad - \pi\,\mathbb{E}[Y(0) \mid T = 1] - (1-\pi)\,\mathbb{E}[Y \mid T = 0] && \text{(8.8 revisited)} \\
&\leq \pi\,\mathbb{E}[Y \mid T = 1] + (1-\pi)\,\mathbb{E}[Y \mid T = 0] \\
&\quad - \pi\,a - (1-\pi)\,\mathbb{E}[Y \mid T = 0] && (8.29) \\
&= \pi\,\mathbb{E}[Y \mid T = 1] - \pi\,a && (8.30)
\end{aligned}$$

[7] Recall the no-assumptions upper bound (Proposition 8.2):

$$\begin{aligned}
&\mathbb{E}[Y(1) - Y(0)] \\
&\quad \leq \pi\,\mathbb{E}[Y \mid T = 1] + (1-\pi)\,b \\
&\quad\quad - \pi\,a - (1-\pi)\,\mathbb{E}[Y \mid T = 0] \qquad \text{(8.9 revisited)}
\end{aligned}$$

The OTS assumption also tells us that

$$\mathbb{E}[Y(0) \mid T = 1] \leq \mathbb{E}[Y(1) \mid T = 1] = \mathbb{E}[Y \mid T = 1]\,, \tag{8.31}$$

which is equivalent to saying $-\mathbb{E}[Y(0) \mid T = 1] \geq -\mathbb{E}[Y \mid T = 1]$. So we can lower bound $-\mathbb{E}[Y(0) \mid T = 1]$ with $-\mathbb{E}[Y \mid T = 1]$, and we can lower bound $\mathbb{E}[Y(1) \mid T = 0]$ with a (just as we did in the no-assumptions lower bound[8]) to get the following lower bound:

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0)] &= \pi\,\mathbb{E}[Y \mid T = 1] + (1-\pi)\,\mathbb{E}[Y(1) \mid T = 0] \\
&\quad - \pi\,\mathbb{E}[Y(0) \mid T = 1] - (1-\pi)\,\mathbb{E}[Y \mid T = 0] && \text{(8.8 revisited)} \\
&\geq \pi\,\mathbb{E}[Y \mid T = 1] + (1-\pi)\,a \\
&\quad - \pi\,\mathbb{E}[Y \mid T = 1] - (1-\pi)\,\mathbb{E}[Y \mid T = 0] && (8.32) \\
&= (1-\pi)\,a - (1-\pi)\,\mathbb{E}[Y \mid T = 0] && (8.33)
\end{aligned}$$

[8] Recall the no-assumptions lower bound (Proposition 8.2):

$$\begin{aligned}
&\mathbb{E}[Y(1) - Y(0)] \\
&\quad \geq \pi\,\mathbb{E}[Y \mid T = 1] + (1-\pi)\,a \\
&\quad\quad - \pi\,b - (1-\pi)\,\mathbb{E}[Y \mid T = 0] \qquad \text{(8.10 revisited)}
\end{aligned}$$

Proposition 8.6 (Optimal Treatment Selection Bound 1) *Let π denote $P(T = 1)$, where T is a binary random variable. Given that the outcome Y is bounded from below by a (Assumption 8.1) and that the optimal treatment is always selection (Assumption 8.5), we have the following upper and lower bounds on the ATE:*

$$\mathbb{E}[Y(1) - Y(0)] < \pi\,\mathbb{E}[Y \mid T = 1] - \pi\, a \tag{8.34}$$
$$\mathbb{E}[Y(1) - Y(0)] \geq (1 - \pi)\, a - (1 - \pi)\,\mathbb{E}[Y \mid T = 0] \tag{8.35}$$
$$\textit{Interval Length} = \pi\,\mathbb{E}[Y \mid T = 1] + (1 - \pi)\,\mathbb{E}[Y \mid T = 0] - a \tag{8.36}$$

Unfortunately, this interval also always contains zero![9] This means that Proposition 8.6 doesn't tell us whether the causal effect is non-zero or not.

[9] Active reading exercise: Show that this interval always contains zero.

Running Example Recall our running example from Section 8.1.1 where $a = 0$, $b = 1$, $\pi = .3$, $\mathbb{E}[Y \mid T = 1] = .9$, and $\mathbb{E}[Y \mid T = 0] = .2$. Plugging these in to Proposition 8.6 gives us the following:

$$\mathbb{E}[Y(1) - Y(0)] \leq (.3)\,(.9) - (.3)\,(0) \tag{8.37}$$
$$\mathbb{E}[Y(1) - Y(0)] \geq (1 - .3)\,(0) - (1 - .3)\,(.2) \tag{8.38}$$
$$-0.14 \leq \mathbb{E}[Y(1) - Y(0)] \leq 0.27 \tag{8.39}$$
$$\text{Interval Length} = 0.41 \tag{8.40}$$

We'll now give an interval that can be purely positive or purely negative, potentially identifying the ATE as non-zero.

A Bound That Can Identify the Sign of the ATE

It turns out that, although we take the OTS assumption from Manski [55], the bound we gave in Proposition 8.6 is not actually the bound that Manski [55] derives with that assumption. For example, where we used $\mathbb{E}[Y(1) \mid T = 0] \leq \mathbb{E}[Y \mid T = 0]$, Manski uses $\mathbb{E}[Y(1) \mid T = 0] \leq \mathbb{E}[Y \mid T = 1]$. We'll quickly prove this inequality that Manski uses from the OTS assumption:[10] We start by applying Equation 8.42:

[55]: Manski (1990), 'Nonparametric Bounds on Treatment Effects'

$$\mathbb{E}[Y(1) \mid T = 0] = \mathbb{E}[Y(1) \mid Y(0) > Y(1)] \tag{8.45}$$

Because the random variable we are taking the expectation of is $Y(1)$, if we flip $Y(0) > Y(1)$ to $Y(0) \leq Y(1)$, then we get an upper bound:

$$\leq \mathbb{E}[Y(1) \mid Y(0) \leq Y(1)] \tag{8.46}$$

Finally, applying Equation 8.44, we have the result:

$$= \mathbb{E}[Y(1) \mid T = 1] \tag{8.47}$$
$$= \mathbb{E}[Y \mid T = 1] \tag{8.48}$$

Now that we have that $\mathbb{E}[Y(1) \mid T = 0] \leq \mathbb{E}[Y \mid T = 1]$, we can prove Manski [55]'s upper bound, where we use this key inequality in

[10] Recall the OTS assumption (Assumption 8.5):

$$T_i = 1 \implies Y_i(1) \geq Y_i(0) \tag{8.41}$$
$$T_i = 0 \implies Y_i(0) > Y_i(1) \tag{8.42}$$

Because there are only two values that T can take on, this is equivalent to the following (contrapositives):

$$T_i = 0 \impliedby Y_i(1) < Y_i(0) \tag{8.43}$$
$$T_i = 1 \impliedby Y_i(0) \leq Y_i(1) \tag{8.44}$$

[55]: Manski (1990), 'Nonparametric Bounds on Treatment Effects'

Equation 8.49:

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0)] &= \pi\,\mathbb{E}[Y \mid T=1] + (1-\pi)\,\mathbb{E}[Y(1) \mid T=0] \\
&\quad - \pi\,\mathbb{E}[Y(0) \mid T=1] - (1-\pi)\,\mathbb{E}[Y \mid T=0] \\
&\qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad \text{(8.8 revisited)} \\
&\leq \pi\,\mathbb{E}[Y \mid T=1] + (1-\pi)\,\mathbb{E}[Y(1) \mid T=1] \\
&\quad - \pi\,a - (1-\pi)\,\mathbb{E}[Y \mid T=0] \qquad\qquad\qquad\qquad (8.49) \\
&= \pi\,\mathbb{E}[Y \mid T=1] + (1-\pi)\,\mathbb{E}[Y \mid T=1] \\
&\quad - \pi\,a - (1-\pi)\,\mathbb{E}[Y \mid T=0] \qquad\qquad\qquad\qquad (8.50) \\
&= \mathbb{E}[Y \mid T=1] - \pi\,a - (1-\pi)\,\mathbb{E}[Y \mid T=0] \qquad\qquad (8.51)
\end{aligned}$$

Similarly, we can perform an analogous derivation[11] to get the lower bound:

$$\mathbb{E}[Y(1) - Y(0)] \geq \pi\,\mathbb{E}[Y \mid T=1] + (1-\pi)\,a - \mathbb{E}[Y \mid T=0] \qquad (8.52)$$

[11] Active reading exercise: Derive Equation 8.52 yourself.

Proposition 8.7 (Optimal Treatment Selection Bound 2) *Let π denote $P(T=1)$, where T is a binary random variable. Given that the outcome Y is bounded from below by a (Assumption 8.1) and that the optimal treatment is always selection (Assumption 8.5), we have the following upper and lower bounds on the ATE:*

$$\mathbb{E}[Y(1) - Y(0)] \leq \mathbb{E}[Y \mid T=1] - \pi\,a - (1-\pi)\,\mathbb{E}[Y \mid T=0] \qquad (8.53)$$

$$\mathbb{E}[Y(1) - Y(0)] \geq \pi\,\mathbb{E}[Y \mid T=1] + (1-\pi)\,a - \mathbb{E}[Y \mid T=0] \qquad (8.54)$$

$$\textit{Interval Length} = (1-\pi)\,\mathbb{E}[Y \mid T=1] + \pi\,\mathbb{E}[Y \mid T=0] - a \qquad (8.55)$$

This interval can also include zero, but it doesn't have to. For example, in our running example, it doesn't.

Running Example Recall our running example from Section 8.1.1 where $a=0$, $b=1$, $\pi=.3$, $\mathbb{E}[Y \mid T=1]=.9$, and $\mathbb{E}[Y \mid T=0]=.2$. Plugging these in to Proposition 8.7 gives us the following for the OTS bound 2:

$$\mathbb{E}[Y(1) - Y(0)] \leq (.9) - (.3)\,(0) - (1-.3)\,(.2) \qquad (8.56)$$

$$\mathbb{E}[Y(1) - Y(0)] \geq (.3)\,(.9) + (1-.3)\,(0) - (.2) \qquad (8.57)$$

$$0.07 \leq \mathbb{E}[Y(1) - Y(0)] \leq 0.76 \qquad (8.58)$$

$$\text{Interval Length} = 0.69 \qquad (8.59)$$

Application of OTS bound 1 (Proposition 8.6) to our running example:

$$-0.14 \leq \mathbb{E}[Y(1) - Y(0)] \leq 0.27 \qquad \text{(8.39 revisited)}$$

$$\text{Interval Length} = 0.41 \qquad \text{(8.40 revisited)}$$

So while the OTS bound 2 from Manski [55] identifies the sign of the ATE in our running example, unlike the OTS bound 1, the OTS bound 2 gives us a 68% larger interval. You can see this by comparing Equation 8.40 (in the above margin) with Equation 8.59.

[55]: Manski (1990), 'Nonparametric Bounds on Treatment Effects'

This illustrates some important takeaways:

1. Different bounds are better in different cases.[12]
2. Different bounds can be better in different ways (e.g., identifying the sign vs. getting a smaller interval).

[12] Active reading exercise: Using Equations 8.40 and 8.59, derive the conditions under which OTS bound 1 yields a smaller interval and the conditions under which OTS bound 2 yields a smaller interval.

Mixing Bounds Fortunately because both the OTS bound 1 and OTS bound 2 come from the same assumption (Assumption 8.5), we can take the lower bound from OTS bound 2 and the upper bound from OTS

bound 1 to get the following tighter interval that still identifies the sign:

$$0.07 \leq \mathbb{E}[Y(1) - Y(0)] \leq 0.27 \tag{8.60}$$

Similarly, we could have mixed the lower bound from OTS bound 1 and the upper bound from OTS bound 2, but that would have given the worst interval in this subsection for this specific example. It could be the best in a different example, though.

In this section we've given you a taste of what kind of results we can get from nonparametric bounds, but, of course, this is just an introduction. For more literature on this, see, e.g., [53–60].

[54]: Manski (1989), 'Anatomy of the Selection Problem'
[55]: Manski (1990), 'Nonparametric Bounds on Treatment Effects'
[56]: Manski (1993), 'Identification Problems in the Social Sciences'
[57]: Manski (1994), 'The selection problem'
[58]: Manski (1997), 'Monotone Treatment Response'
[59]: Manski and Pepper (2000), 'Monotone Instrumental Variables: With an Application to the Returns to Schooling'
[53]: Manski (2003), *Partial Identification of Probability Distributions: Springer Series in Statistics*
[60]: Manski (2013), *Public Policy in an Uncertain World*

8.2 Sensitivity Analysis

8.2.1 Sensitivity Basics in Linear Setting

Before this chapter, we have exclusively been working in the setting where causal effects are identifiable. We illustrate the common example of the confounders W as common causes of T and Y in Figure 8.2. In this example, the causal effect of T on Y is identifiable. However, what if there is a single unobserved confounder U, as we illustrate in Figure 8.3. Then, the causal effect is not identifiable.

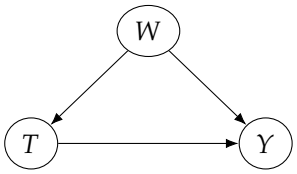


Figure 8.2: Simple causal structure where W confounds the effect of T on Y and where W is the only confounder.

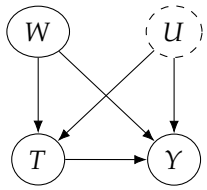


Figure 8.3: Simple causal structure where W is the observed confounders and U is the unobserved confounders.

What would be the bias we'd observe if we only adjusted for the observed confounders W? To illustrate this simply, we'll start with a noiseless[13] linear data generating process. So consider data that are generated by the following structural equations:

$$T := \alpha_w W + \alpha_u U \tag{8.61}$$
$$Y := \beta_w W + \beta_u U + \delta T \tag{8.62}$$

So the relevant quantity that describes causal effects of T on Y is δ since it is the coefficient in front of T in the structural equation for Y. From the backdoor adjustment (Theorem 4.2) / adjustment formula (Theorem 2.1), we know that

$$\mathbb{E}[Y(1) - Y(0)] = \mathbb{E}_{W,U}\left[\mathbb{E}[Y \mid T = 1, W, U] - \mathbb{E}[Y \mid T = 0, W, U]\right] = \delta \tag{8.63}$$

But because U isn't observed, the best we can do is adjust for only W. This leads to a confounding bias of $\frac{\beta_u}{\alpha_u}$. We'll be focusing on identification, not estimation, here, so we'll consider that we have infinite data. This means that we have access to $P(W, T, Y)$. Then, we'll write down and prove the following proposition about confounding bias:

Proposition 8.8 *When T and Y are generated by the noiseless linear process in Equations 8.61 and 8.62, the confounding bias of adjusting for just W (and*

[13] Active reading exercise: What assumption is violated when the data are generated by a noiseless process?

not U) is $\frac{\beta_u}{\alpha_u}$. Mathematically:

$$\mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W] - \mathbb{E}[Y \mid T=0,W]\right] - \mathbb{E}_{W,U}\left[\mathbb{E}[Y \mid T=1,W,U] - \mathbb{E}[Y \mid T=0,W,U]\right] = \frac{\beta_u}{\alpha_u} \tag{8.64}$$

Proof. We'll prove Proposition 8.8 in 3 steps:

1. Get a closed-form expression for $\mathbb{E}_W\left[\mathbb{E}[Y \mid T=t,W]\right]$ in terms of α_w, α_u, β_w, and β_u.
2. Use step 1 to get a closed-form expression for the difference $\mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W] - \mathbb{E}[Y \mid T=0,W]\right]$.
3. Subtract off $\mathbb{E}_{W,U}\left[\mathbb{E}[Y \mid T=1,W,U] - \mathbb{E}[Y \mid T=0,W,U]\right] = \delta$.[14]

[14] Active reading exercise: Show that $\mathbb{E}_{W,U}\left[\mathbb{E}[Y \mid T=1,W,U] - \mathbb{E}[Y \mid T=0,W,U]\right]$ equals δ.

First, we use the structural equation for Y (Equation 8.62):

$$Y := \beta_w W + \beta_u U + \delta T \quad \text{(8.62 revisited)}$$

$$\mathbb{E}_W\left[\mathbb{E}[Y \mid T=t,W]\right] = \mathbb{E}_W\left[\mathbb{E}[\beta_w W + \beta_u U + \delta T \mid T=t,W]\right] \tag{8.65}$$

$$= \mathbb{E}_W\left[\beta_w W + \beta_u \mathbb{E}[U \mid T=t,W] + \delta t\right] \tag{8.66}$$

This is where we use the structural equation for T (Equation 8.61). Rearranging it gives us $U = \frac{T-\alpha_w W}{\alpha_u}$. We can then use that for the remaining conditional expectation:

$$T := \alpha_w W + \alpha_u U \quad \text{(8.61 revisited)}$$

$$= \mathbb{E}_W\left[\beta_w W + \beta_u\left(\frac{t-\alpha_w W}{\alpha_u}\right) + \delta t\right] \tag{8.67}$$

$$= \mathbb{E}_W\left[\beta_w W + \frac{\beta_u}{\alpha_u} t - \frac{\beta_u \alpha_w}{\alpha_u} W + \delta t\right] \tag{8.68}$$

$$= \beta_w \mathbb{E}[W] + \frac{\beta_u}{\alpha_u} t - \frac{\beta_u \alpha_w}{\alpha_u}\mathbb{E}[W] + \delta t \tag{8.69}$$

Then, rearranging a bit, we have the following:

$$= \left(\delta + \frac{\beta_u}{\alpha_u}\right) t + \left(\beta_w - \frac{\beta_u \alpha_w}{\alpha_u}\right)\mathbb{E}[W] \tag{8.70}$$

The only parts of this that matter are the parts that depend on t because we want to know the effect of T on Y. For example, consider the expected ATE estimate we would get if we were to only adjust for W:

$$\mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W] - \mathbb{E}[Y \mid T=0,W]\right] \tag{8.71}$$

$$= \left(\delta + \frac{\beta_u}{\alpha_u}\right)(1) + \left(\beta_w - \frac{\beta_u \alpha_w}{\alpha_u}\right)\mathbb{E}[W] - \left[\left(\delta + \frac{\beta_u}{\alpha_u}\right)(0) + \left(\beta_w - \frac{\beta_u \alpha_w}{\alpha_u}\right)\mathbb{E}[W]\right] \tag{8.72}$$

$$= \delta + \frac{\beta_u}{\alpha_u} \tag{8.73}$$

Finally, subtracting off $\mathbb{E}_{W,U}\left[\mathbb{E}[Y \mid T=1,W,U]-\mathbb{E}[Y \mid T=0,W,U]\right]$:

$$\begin{aligned}\text{Bias} &= \mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W]-\mathbb{E}[Y \mid T=0,W]\right] \\ &\quad - \mathbb{E}_{W,U}\left[\mathbb{E}[Y \mid T=1,W,U]-\mathbb{E}[Y \mid T=0,W,U]\right] && (8.74)\\ &= \delta + \frac{\beta_u}{\alpha_u} - \delta && (8.75)\\ &= \frac{\beta_u}{\alpha_u} && (8.76)\end{aligned}$$

□

Generalization to Arbitrary Graphs/Estimands Here, we've performed a sensitivity analysis for the ATE for the simple graph structure in Figure 8.4. For arbitrary estimands in arbitrary graphs, where the structural equations are linear, see Cinelli et al. [61].

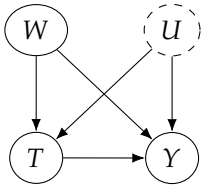


Figure 8.4: Simple causal structure where W is the observed confounders and U is the unobserved confounders.

[61]: Cinelli et al. (2019), 'Sensitivity Analysis of Linear Structural Causal Models'

Sensitivity Contour Plots

Because Proposition 8.8 gives us a closed-form expression for the bias in terms of the unobserved confounder parameters α_u and β_u, we can plot the levels of bias in contour plots. We show this in Figure 8.5a, where we have $\frac{1}{\alpha_u}$ on the x-axis and β_u on the y-axis.

If we rearrange Equation 8.73[15] to solve for δ, we get the following:

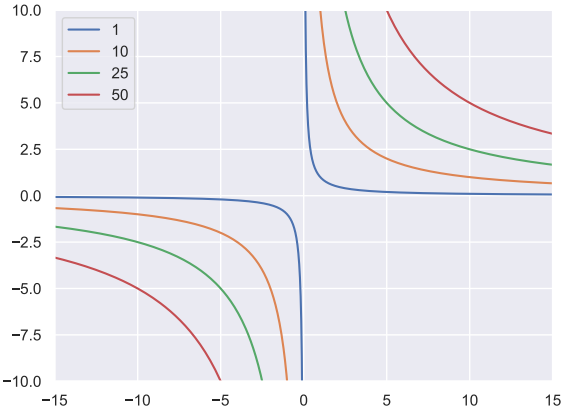
$$\delta = \mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W]-\mathbb{E}[Y \mid T=0,W]\right] - \frac{\beta_u}{\alpha_u} \tag{8.77}$$

So for given values of α_u and β_u, we can compute the true ATE δ, from the observational quantity $\mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W]-\mathbb{E}[Y \mid T=0,W]\right]$. This allows us to get sensitivity curves that allow us to know how robust conclusions like "$\mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W]-\mathbb{E}[Y \mid T=0,W]\right] = 25$ is positive, so δ is likely positive" are to unobserved confounding. We plot such relevant contours of δ in in Figure 8.5b.

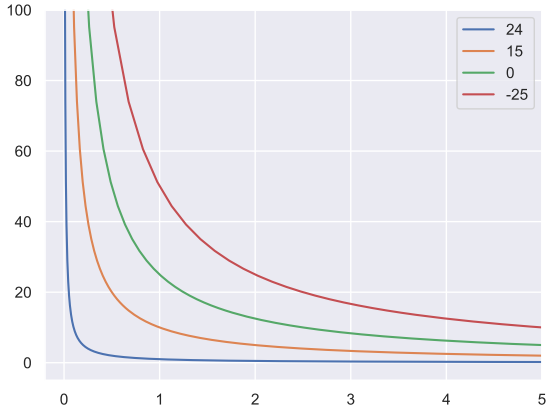
[15] Recall Equation 8.73:

$$\mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W]-\mathbb{E}[Y \mid T=0,W]\right] = \delta + \frac{\beta_u}{\alpha_u}$$

(8.73 revisited)



(a) Contours of confounding bias $\frac{\beta_u}{\alpha_u}$



(b) Contours of the true ATE δ, given that $\mathbb{E}_W\left[\mathbb{E}[Y \mid T=1,W]-\mathbb{E}[Y \mid T=0,W]\right] = 25$

Figure 8.5: Contour plots for sensitivity where the x-axis for both is $\frac{1}{\alpha_u}$ and the y-axis is β_u. There is a color-coded correspondence between the curves in the upper right of Figure 8.5b and the curves in Figure 8.5

In the example we depict in Figure 8.5, the figure tells us that the green curve (third from the bottom/left) indicates how strong the confounding would need to be in order to completely explain the observed association. In other words, $(\frac{1}{\alpha_u}, \beta_u)$ would need be large enough to fall on the green curve or above in order for the true ATE δ to be zero or the opposite sign of $\mathbb{E}_W\left[\mathbb{E}[Y \mid T = 1, W] - \mathbb{E}[Y \mid T = 0, W]\right] = 25$.

8.2.2 More General Settings

We consider a simple linear setting in Section 8.2.1 in order to easily convey the important concepts in sensitivity analysis. However, there is existing that allows us to do sensitivity analysis in more general settings.

Say we are in the common setting where T is binary. This is not the case in the previous section (see Equation 8.61). Rosenbaum and Rubin [62] and Imbens [63][16] consider a simple binary treatment setting with binary U by just putting a logistic sigmoid function around the right-hand side of Equation 8.61 and using that for the probability of treatment instead of the actual value of treatment:

$$T := \alpha_w W + \alpha_u U \qquad \text{(8.61 revisited)}$$

$$P(T = 1 \mid W, U) := \frac{1}{1 + \exp(-(\alpha_w W + \alpha_u U))} \tag{8.78}$$

[62]: Rosenbaum and Rubin (1983), 'Assessing Sensitivity to an Unobserved Binary Covariate in an Observational Study with Binary Outcome'
[63]: Imbens (2003), 'Sensitivity to Exogeneity Assumptions in Program Evaluation'

[16] Imbens [63] is the first to introduce contour plots like the ones in our Figure 8.5.

No Assumptions on T or U Fortunately, we can drop a lot of the assumptions that we've seen so far. Unlike the linear form that we assumed for T in Section 8.2.1 and the linearish form that Rosenbaum and Rubin [62] and Imbens [63] assume, Cinelli and Hazlett [64] develop a method for sensitivity analysis that is agnostic to the functional form of T. Their method also allows for U to be non-binary and for U to be a vector, rather than just a single unobserved confounder.

[64]: Cinelli and Hazlett (2020), 'Making sense of sensitivity: extending omitted variable bias'

Arbitrary Machine Learning Models for Parametrization of T and Y Recall that all of the estimators that we considered in Chapter 7 allowed us to plug in arbitrary machine learning models to get model-assisted estimators. It might be attractive to have an analogous option in sensitivity analysis, potentially using the exact same models for the conditional outcome model μ and the propensity score e that we used for estimation. And this is exactly what Veitch and Zaveri [65] give us. And they are even able to derive a closed-form expression for confounding bias, assuming the models we use for μ and e are well-specified, something that Rosenbaum and Rubin [62] and Imbens [63] didn't do in their simple setting.

[65]: Veitch and Zaveri (2020), *Sense and Sensitivity Analysis: Simple Post-Hoc Analysis of Bias Due to Unobserved Confounding*

Holy Shit; There Are a Lot of Options Although we only highlighted a few options above, there are many different approaches to sensitivity analysis, and people don't agree on which ones are best. This means that sensitivity analysis is an active area of current research. See Liu et al. [66] for a review of methods that preceeded 2013. Rosenbaum is another key figure in sensitivity analysis with his several different approaches [67–69]. Here is a non-exhaustive list of a few other flexible sensitivity analysis methods that you might be interested in looking into: Franks et al. [70], Yadlowsky et al. [71], Vanderweele and Arah [72], and Ding and VanderWeele [73].

[66]: Liu et al. (2013), 'An introduction to sensitivity analysis for unobserved confounding in nonexperimental prevention research'
[67]: Rosenbaum (2002), *Observational Studies*
[68]: Rosenbaum (2010), *Design of Observational Studies*
[69]: Rosenbaum (2017), *Observation and Experiment*
[70]: Franks et al. (2019), 'Flexible Sensitivity Analysis for Observational Studies Without Observable Implications'
[71]: Yadlowsky et al. (2020), *Bounds on the conditional and average treatment effect with unobserved confounding factors*
[72]: Vanderweele and Arah (2011), 'Bias formulas for sensitivity analysis of unmeasured confounding for general outcomes, treatments, and confounders'
[73]: Ding and VanderWeele (2016), 'Sensitivity Analysis Without Assumptions'

9 Instrumental Variables

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How can we identify causal effects when we are in the presence of unobserved confounding? One popular way is to find and use *instrumental variables*. An instrument (instrumental variable) Z has three key qualities. It affects on treatment T, it affects Y only through T, and the effect of Z on Y is unconfounded. We depict these qualities in Figure 9.1. These qualities allow us to use Z to isolate the causal association flowing from T to Y. The intuition is that changes in Z will be reflected in T and lead to corresponding changes in Y. And these specifically Z-focused changes are unconfounded (unlike the changes to T induced by the unobserved confounder U), so they allow us to isolate the causal association that flows from T to Y.

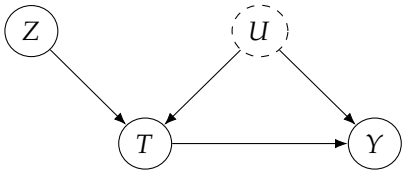

Figure 9.1: Graph where U is an unobserved confounder of the effect of T on Y and Z is an instrumental variable.

9.1 What is an Instrument?

There are three main assumptions that must be satisfied for a variable Z to be considered an instrument. The first is that Z must be relevant in the sense that it must influence T.

Assumption 9.1 (Relevance) *Z has a causal effect on T*

Graphically, the relevance assumption corresponds to the existence of an active edge from Z to T in the causal graph. The second assumption is known as the *exclusion restriction*.

Assumption 9.2 (Exclusion Restriction) *Z causal effect on Y is fully mediated by T*

This assumption is known as the exclusion restriction because it excludes Z from the structural equation for Y and from any other structural equations that would make causal association flow from Z to Y without going through T. Graphically, this means that we've excluded enough potential edges between variables in the causal graph so that all causal paths from Z to Y go through T. Finally, we assume that the causal effect of Z on Y is unconfounded:

Assumption 9.3 (Instrumental Unconfoundedness) *There are no backdoor paths from Z to Y.*

Conditional Instruments We phrased Assumption 9.3 as unconditional unconfoundedness, but all the math for instrumental variables still works if we have unconfoundedness conditional on observed variables as well. We just have to make sure we condition on those relevant variables. In this case, you might see Z referred to as a *conditional instrument*.

9.2 No Nonparametric Identification of the ATE

You might be wondering "if instrumental variables allow us to identify causal effects, then why didn't we see them back in Chapter 6 Nonparametric Identification?" The answer is that instrumental variables don't *nonparametrically* identify the causal effect. We have nonparametric identification when we don't have to make any assumptions about the parametric form. With instrumental variables, we must make assumptions about the parametric form (e.g. linear) to identify causal effects.

We saw the following useful necessary condition for nonparametric identification in Section 6.3: For each backdoor path from T to any child that is an ancestor of Y, it is possible to block that path [18, p. 92]. And we can see in Figure 9.2 that there is a backdoor path from T to Y that cannot be blocked: $T \leftarrow U \rightarrow Y$. So this necessary condition tells us that we can't use the instrument Z to nonparametrically identify the effect of T on Y.

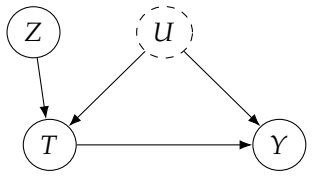

Figure 9.2: Graph where U is an unobserved confounder of the effect of T on Y and Z is an instrumental variable.

[18]: Pearl (2009), *Causality*

9.3 Warm-Up: Binary Linear Setting

As a warm-up, we'll start in the setting where T and Z are binary and where we make the parametric assumption that Y is a linear function of T and U:

Assumption 9.4 (Linear Outcome)

$$Y := \delta T + \alpha_u U \tag{9.1}$$

The fact that Z doesn't appear in Equation 9.1 is a consequence of the exclusion restriction (Assumption 9.2).

Then, with this assumption in mind, we'll try to identify the causal effect δ. Because we have the intuition that Z will be useful for identifying the effect of T on Y, we'll start with the associational difference for the Z-Y relationship: $\mathbb{E}[Y \mid Z = 1] - \mathbb{E}[Y \mid Z = 0]$. By immediately applying Assumption 9.4, we have the following:

$$\mathbb{E}[Y \mid Z = 1] - \mathbb{E}[Y \mid Z = 0] \tag{9.2}$$
$$= \mathbb{E}[\delta T + \alpha_u U \mid Z = 1] - \mathbb{E}[\delta T + \alpha_u U \mid Z = 0] \tag{9.3}$$

Using linearity of expectation and rearranging a bit:

$$= \delta \left(\mathbb{E}[T \mid Z = 1] - \mathbb{E}[T \mid Z = 0]\right) + \alpha_u \left(\mathbb{E}[U \mid Z = 1] - \mathbb{E}[U \mid Z = 0]\right) \tag{9.4}$$

Now, we use the instrumental unconfoundedness assumption (Assumption 9.3). This means that Z and U are independent, which allows us to get rid of the U term:

$$= \delta \left(\mathbb{E}[T \mid Z = 1] - \mathbb{E}[T \mid Z = 0]\right) + \alpha_u \left(\mathbb{E}[U] - \mathbb{E}[U]\right) \tag{9.5}$$

$$= \delta \left(\mathbb{E}[T \mid Z = 1] - \mathbb{E}[T \mid Z = 0]\right) \tag{9.6}$$

Then, we can solve for δ to get the *Wald estimand*:

Proposition 9.1

$$\delta = \frac{\mathbb{E}[Y \mid Z = 1] - \mathbb{E}[Y \mid Z = 0]}{\mathbb{E}[T \mid Z = 1] - \mathbb{E}[T \mid Z = 0]} \tag{9.7}$$

Because of Assumption 9.1, we know that the denominator is non-zero, so the right-hand side isn't undefined. Then, we just plug in empirical means in place of these conditional expectations to get the *Wald estimator* [74]:

$$\hat{\delta} = \frac{\frac{1}{n_1}\sum_{i:z_i=1} Y_i - \frac{1}{n_0}\sum_{i:z_i=0} Y_i}{\frac{1}{n_1}\sum_{i:z_i=1} T_i - \frac{1}{n_0}\sum_{i:z_i=0} T_i} \tag{9.8}$$

where n_1 is the number of samples where $Z = 1$ and n_0 is the number of samples where $Z = 0$.

[74]: Wald (1940), 'The Fitting of Straight Lines if Both Variables are Subject to Error'

Active reading exercise: Where did we use each of Assumptions 9.1 to 9.4 in the above derivation of Equation 9.7.

Causal Effects as Multiplying Path Coefficients When the structural equations are linear, you can think of the causal association flowing from a variable A to a variable B as the product of the coefficients along the directed path from A to B. If there are multiple paths, you just sum the causal associations along all those paths. However, we don't have direct access to the causal association. Rather, we can measure total association, and unblocked backdoor paths also contribute to total association, which is why $\mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0] \neq \delta$. So how can we identify the effect of T on Y in Figure 9.3? Because there are no backdoor paths from the instrument Z to Y, we can trivially identify the effect of Z on Y: $\mathbb{E}[Y \mid Z = 1] - \mathbb{E}[Y \mid Z = 0] = \alpha_z \delta$. Similarly, we can identify the effect of the instrument on T: $\mathbb{E}[T \mid Z = 1] - \mathbb{E}[T \mid Z = 0] = \alpha_z$. Then, we can divide the effect of Z on Y by the effect of the Z on T to identify δ $\left(\frac{\alpha_z \delta}{\alpha_z}\right)$. And this quotient is exactly the Wald estimand in Proposition 9.1.

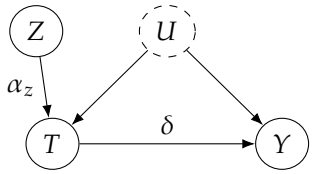

Figure 9.3: Graph where U is an unobserved confounder of the effect of T on Y and Z is an instrumental variable.

9.4 Continuous Linear Setting

We'll now consider the setting where T and Z are continuous, rather than binary. We'll still assume the linear form for Y (Assumption 9.4), which means that the causal efffect of T on Y is δ. In the continuous setting, we get the natural continuous analog of the Wald estimand:

Proposition 9.2

$$\delta = \frac{\text{Cov}(Y, Z)}{\text{Cov}(T, Z)} \tag{9.9}$$

Proof. Just as we started with $\mathbb{E}[Y \mid Z = 1] - \mathbb{E}[Y \mid Z = 0]$ in the previous section, here, we'll start with the continuous analog $\text{Cov}(Y, Z)$. We start with a classic covariance identity:

$$\text{Cov}(Y, Z) = \mathbb{E}[YZ] - \mathbb{E}[Y]\mathbb{E}[Z] \tag{9.10}$$

Then, applying the linear outcome assumption (Assumption 9.4):

$$= \mathbb{E}[(\delta T + \alpha_u U)Z] - \mathbb{E}[\delta T + \alpha_u U]\mathbb{E}[Z] \tag{9.11}$$

Distributing and rearranging:

$$= \delta\mathbb{E}[TZ] + \alpha_u\mathbb{E}[UZ] - \delta\mathbb{E}[T]\mathbb{E}[Z] - \alpha_u\mathbb{E}[U]\mathbb{E}[Z] \tag{9.12}$$
$$= \delta\left(\mathbb{E}[TZ] - \mathbb{E}[T]\mathbb{E}[Z]\right) + \alpha_u\left(\mathbb{E}[UZ] - \mathbb{E}[U]\mathbb{E}[Z]\right) \tag{9.13}$$

Now, we see that we can apply the same covariance identity again:

$$= \delta\text{Cov}(T, Z) + \alpha_u\text{Cov}(U, Z) \tag{9.14}$$

And $\text{Cov}(U, Z) = 0$ by the instrumental unconfoundedness assumption (Assumption 9.3):

$$= \delta\text{Cov}(T, Z) \tag{9.15}$$

Finally, we solve for δ:

$$\delta = \frac{\text{Cov}(Y, Z)}{\text{Cov}(T, Z)} \tag{9.16}$$

where the relevance assumption (Assumption 9.1) tells us that the denominator is non-zero. □

This leads us to the following natural estimator, similar to the Wald estimator:

$$\hat{\delta} = \frac{\widehat{\text{Cov}}(Y, Z)}{\widehat{\text{Cov}}(T, Z)} \tag{9.17}$$

Another equivalent estimator is what's known as the *two-stage least squares estimator (2SLS)*. The two stages are as follows:

1. Linearly regress T on Z to estimate $\mathbb{E}[T \mid Z]$. This gives us the projection of T onto Z: $\hat{T}$.
2. Linearly regress Y on $\hat{T}$ to estimate $\mathbb{E}[Y \mid \hat{T}]$. Obtain our estimate $\hat{\delta}$ as the fitted coefficient in front of $\hat{T}$.

There is helpful intuition that comes with the 2SLS estimator. To see this, start with the canonical instrumental variable graph we've been using (Figure 9.4). In stage one, we are projecting T onto Z to get $\hat{T}$ as a function of only Z: $\hat{T} = \hat{\mathbb{E}}[T \mid Z]$. Then, imagine a graph where T is replaced with $\hat{T}$ (Figure 9.5). Because $\hat{T}$ isn't a function of U, we can think of removing the $U \to \hat{T}$ edge in this graph. Now, because there are no backdoor paths

Active reading exercise: Where did we use the exclusion restriction assumption (Assumption 9.2) in this proof?

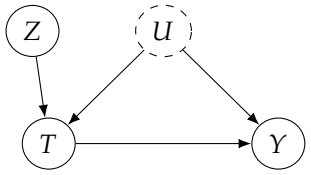

Figure 9.4: Graph where U is an unobserved confounder of the effect of T on Y and Z is an instrumental variable.

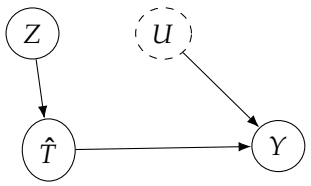

Figure 9.5: Augmented version of Figure 9.4, where T is replaced with $\hat{T} = \hat{\mathbb{E}}[T \mid Z]$, which doesn't depend on U, so there it no longer has an incoming edge from U.

from $\hat{T}$ to Y, we can get that association is causation in stage two, where we simply regress Y on $\hat{T}$ to estimate the causal effect. Note: We can also use 2SLS in the binary setting we discussed in Section 9.3.

9.5 Nonparametric Identification of Local ATE

The problem with the previous two sections is that we've made the strong parametric assumption of linearity (Assumption 9.4). For example, this assumption requires *homogeneity* (that the treatment effect is the same for every unit). There are other variants that encode the homogeneity assumption (see, e.g., Hernán and Robins [7, Section 16.3]), and they are all strong assumptions. Ideally, we'd be able to use instrumental variables for identification without making any parametric assumptions such as linearity or homogeneity. And we can. We just need to settle for a more specific causal estimand than the ATE and swap the linearity assumption out for a new assumption. We will do this in the binary setting, so both T and Z are binary. Before we can do that, we must define a bit of new notation in Section 9.5.1 and introduce principal stratification in Section 9.5.2.

[7]: Hernán and Robins (2020), *Causal Inference: What If*

9.5.1 New Potential Notation with Instruments

Just like we use $Y(1) \triangleq Y(T = 1)$ to denote the potential outcome we would observe if we were to take treatment and $Y(0) \triangleq Y(T = 0)$ to denote the potential outcome we would observe if we were to not take treatment, we will define similar potential notation with instruments.

We'll think of the instrument Z as encouragement for the treatment, so if we have $Z = 1$, we're encouraged to take the treatment, and if we have $Z = 0$, we're encourage to not take the treatment. Let $T(1) \triangleq T(Z = 1)$ denote the treatment we would take if we were to get instrument value 1. Similarly, let $T(0) \triangleq T(Z = 0)$ denote the treatment we would take if we were to get instrument value .

Then, we have the same for potential outcomes where we're intervening on the instrument, rather than the treatment: $Y(Z = 1)$ denotes the outcome we would observe if we were to be encouraged to take the treatment and $Y(Z = 0)$ denotes the outcome we would observe if we were to be encouraged to not take the treatment.

9.5.2 Principal Stratification

We will segment the population into four principal strata, based on the relationship between the encouragement Z and the treatment taken T. There are four strata because there is one for each combination of the values the binary variables Z and T can take on.

Definition 9.1 (Principal Strata)

1. *Compliers - always take the treatment that they're encouraged to take. Namely,* $T(1) = 1$ *and* $T(0) = 0$.

2. *Always-takers - always take the treatment, regardless of encouragement. Namely,* $T(1) = 1$ *and* $T(0) = 1$.
3. *Never-takers - never take the treatment, regardless of encouragement. Namely,* $T(1) = 0$ *and* $T(0) = 0$.
4. *Defiers - always take the opposite treatment of the treatment that they are encouraged to take. Namely,* $T(1) = 0$ *and* $T(0) = 1$.

Different Causal Graphs Importantly, these strata have different causal graphs. While the treatment that the compliers and defiers take depends on the encouragement (instrument), the treatment that the always-takers and never-takers take does not. Therefore, the compliers and defiers have the normal causal graph (Figure 9.6), whereas the always-takers and never-takers have the same causal graph but with the $Z \to T$ edge removed (Figure 9.7). This means that the causal effect of Z on T is zero for always-takers and never-takers. Then, because of the exclusion restriction, this means that the causal effect of Z on Y is zero for the always-takers and never-takers. This will be important for the upcoming derivation.

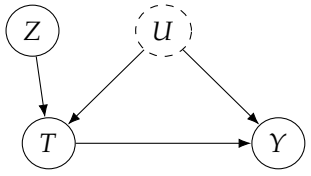

Figure 9.6: Causal graph for the compliers and defiers.

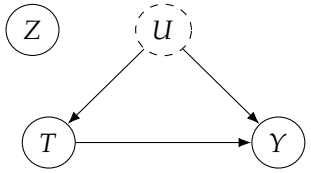

Figure 9.7: Causal graph for the always-takers and never-takers.

Can't Identify Stratum Given some observed value of Z and T, we can't actually identify which stratum we're in. There are four combinations of the binary variables Z and T; for each of these combinations, we'll note that more than one stratum is compatible with the observed combinations of values.

1. $Z = 0$, $T = 0$. Compatible strata: compliers or never-takers
2. $Z = 0$, $T = 1$. Compatible strata: defiers or always-takers
3. $Z = 1$, $T = 0$. Compatible strata: defiers or never-takers
4. $Z = 1$, $T = 1$. Compatible strata: compliers or always-takers

This means that we can't identify if a given unit is a complier, a defier, an always-taker, or a never-taker.

Active reading exercise: Ensure that you follow why these are the compatible strata for each of these combinations of observed values.

9.5.3 Local ATE

Although we won't be able to use instrumental variables to nonparametrically identify the ATE in the presence of unobserved confounding (Section 9.2), we will be able to nonparametrically identify what's known as the *local* ATE. The *local average treatment effect* (LATE) is also known as the *complier average causal effect* (CACE), as it is the ATE among the compliers.

Definition 9.2 (Local Average Treatment Effect (LATE) / Complier Average Causal Effect (CACE))

$$\mathbb{E}[Y(T = 1) - Y(T = 0) \mid T(Z = 1) = 1, T(Z = 0) = 0] \tag{9.18}$$

To identify the LATE, although we will no longer need the linearity assumption (Assumption 9.4), we will need to introduce a new assumption known as *monotonicity*.

Assumption 9.5 (Monotonicity)

$$\forall i, \quad T_i(Z = 1) \geq T_i(Z = 0) \tag{9.19}$$

Monotonicity means that if we are encouraged to take the treatment ($Z = 1$), we are either more likely or equally likely to take the treatment than we would be if we were encouraged to not take the treatment ($Z = 0$). Importantly, this means that we are assuming that there are no defiers. This is because the compliers satisfy $T(1) > T(0)$, the always-takers and never-takers satisfy $T(1) = T(0)$, but the defiers don't satisfy either of these; among the defiers, $T(1) < T(0)$, which is a violation of the monotonicity assumption.

Compliers: $T(1) = 1$, $T(0) = 0$
Always-takers: $T(1) = 1$, $T(0) = 1$
Never-takers: $T(1) = 0$, $T(0) = 0$
Defiers: $T(1) = 0$, $T(0) = 1$

We've now introduced the key concepts of principal strata and the monotonicity assumption. Importantly, we saw that the causal effect of Z on Y is zero among the always-takers and never-takers (Section 9.5.2), and we just saw that monotonicity assumption implies that there are no defiers. With this in mind, we are now ready to derive the nonparametric identification result for the LATE estimand.

Theorem 9.3 (LATE Nonparametric Identification) *Given that Z is an instrument, Z and T are binary variables, and that monotonicity holds, the following is true:*

$$\mathbb{E}[Y(1) - Y(0) \mid T(1) = 1, T(0) = 0] = \frac{\mathbb{E}[Y \mid Z = 1] - \mathbb{E}[Y \mid Z = 0]}{\mathbb{E}[T \mid Z = 1] - \mathbb{E}[T \mid Z = 0]} \tag{9.20}$$

Proof. Because we're interested in the causal effect of T on Y and because know that we'll use the instrument Z, we'll start with the causal effect of Z on Y and decompose it into weighted stratum-specific causal effects using the law of total probability:

$$\begin{aligned}
&\mathbb{E}[Y(Z = 1) - Y(Z = 0)] \\
&\quad = \mathbb{E}[Y(Z = 1) - Y(Z = 0) \mid T(1) = 1, T(0) = 0]\, P(T(1) = 1, T(0) = 0) \\
&\qquad + \mathbb{E}[Y(Z = 1) - Y(Z = 0) \mid T(1) = 0, T(0) = 1]\, P(T(1) = 0, T(0) = 1) \\
&\qquad + \mathbb{E}[Y(Z = 1) - Y(Z = 0) \mid T(1) = 1, T(0) = 1]\, P(T(1) = 1, T(0) = 1) \\
&\qquad + \mathbb{E}[Y(Z = 1) - Y(Z = 0) \mid T(1) = 0, T(0) = 0]\, P(T(1) = 0, T(0) = 0)
\end{aligned} \tag{9.21}$$

The first term correponds to the compliers, the second term corresponds to the the defiers, the third term corresponds to the always-takers, and the last term corresponds to the never takers. As we discussed in Section 9.5.2, the causal effect of Z on Y among the always-takers and never-takers is zero, so we can remove those terms.

$$\begin{aligned}
&= \mathbb{E}[Y(Z = 1) - Y(Z = 0) \mid T(1) = 1, T(0) = 0]\, P(T(1) = 1, T(0) = 0) \\
&\quad + \mathbb{E}[Y(Z = 1) - Y(Z = 0) \mid T(1) = 0, T(0) = 1]\, P(T(1) = 0, T(0) = 1)
\end{aligned} \tag{9.22}$$

Because we've made the monotonicity assumption, we know that there are no defiers ($P(T(1) = 0, T(0) = 1) = 0$), so the defiers term is also zero.

$$= \mathbb{E}[Y(Z = 1) - Y(Z = 0) \mid T(1) = 1, T(0) = 0]\, P(T(1) = 1, T(0) = 0) \tag{9.23}$$

Now, if we solve for this effect of Z on Y among the compliers, we get

the following:

$$\mathbb{E}[Y(Z=1) - Y(Z=0) \mid T(1)=1, T(0)=0] = \frac{\mathbb{E}[Y(Z=1) - Y(Z=0)]}{P(T(1)=1, T(0)=0)} \tag{9.24}$$

And because these are the compliers, people who will take whichever treatment they are encouraged to take, $Y(Z=1)$ and $Y(Z=0)$ are really equal to $Y(T=1)$ and $Y(T=0)$, respectively, so we can change the left-hand side of Equation 9.24 to the LATE, the causal estimand that we're trying to identify:

$$\mathbb{E}[Y(T=1) - Y(T=0) \mid T(1)=1, T(0)=0] \tag{9.25}$$

$$= \frac{\mathbb{E}[Y(Z=1) - Y(Z=0)]}{P(T(1)=1, T(0)=0)} \tag{9.26}$$

Now, we apply the the instrumental unconfoundedness assumption (Assumption 9.3) to identify the numerator.

$$= \frac{\mathbb{E}[Y \mid Z=1] - \mathbb{E}[Y \mid Z=0]}{P(T(1)=1, T(0)=0)} \tag{9.27}$$

All that's left is to identify the denominator, the probability of being a complier. However, we mentioned that we can't identify the compliers in Section 9.5.2, so how can we do this? This is where we'll need to be a bit clever. We'll get this probability by taking everyone (probability 1) and subtracting out the the always-takers and the compliers, since there are no defiers, due to monotonicity (Assumption 9.5).

$$= \frac{\mathbb{E}[Y \mid Z=1] - \mathbb{E}[Y \mid Z=0]}{1 - P(T=0 \mid Z=1) - P(T=1 \mid Z=0)} \tag{9.28}$$

To understand how we got the above equality, consider that everyone either has $Z=1$ or $Z=0$. We can subtract out all of the never-takers by removing those that had $T=0$ among the $Z=1$ subpopulation ($P(T=0 \mid Z=1)$). Similarly, we can subtract out all of the always-takers by removing those that had $T=1$ among the $Z=0$ subpopulation ($P(T=1 \mid Z=0)$). We know that this removes all of the never-takers and always-takers because there are no defiers and because we've looked at both the $Z=1$ subpopulation and the $Z=0$ subpopulation. Now, we just do a bit of manipulation:

$$= \frac{\mathbb{E}[Y \mid Z=1] - \mathbb{E}[Y \mid Z=0]}{1 - (1 - P(T=1 \mid Z=1)) - P(T=1 \mid Z=0)} \tag{9.29}$$

$$= \frac{\mathbb{E}[Y \mid Z=1] - \mathbb{E}[Y \mid Z=0]}{P(T=1 \mid Z=1) - P(T=1 \mid Z=0)} \tag{9.30}$$

Finally, because T is a binary variable, we can swap out probabilities of $T=1$ for expectations:

$$= \frac{\mathbb{E}[Y \mid Z=1] - \mathbb{E}[Y \mid Z=0]}{\mathbb{E}[T \mid Z=1] - \mathbb{E}[T \mid Z=0]} \tag{9.31}$$

□

This is exactly the Wald estimand that we saw back in the linear setting (Section 9.3) in Equation 9.7. However, this time, it is the corresponding statistical estimand of the *local* ATE $\mathbb{E}[Y(T = 1) - Y(T = 0) \mid T(1) = 1, T(0) = 0]$, also known as the complier average causal effect (CACE). This LATE/CACE causal estimand is in contrast to the ATE causal estimand that we saw in Section 9.3: $\mathbb{E}[Y(T = 1) - Y(T = 0)]$. The difference is that the *complier* average causal effect is the ATE specifically in the subpopulation of compliers, rather than the total population. It's *local* (LATE) to that subpopulation, rather than being global over the whole population like the ATE is. So we've seen two different assumptions that get us to the Wald estimand with instrumental variables:

1. Linearity (or more generally homogeneity)
2. Monotonicity

Problems with LATE/CACE There are a few problems with the Wald estimand for LATE, though. The first is that monotonicity might not be satisfied in your setting of interest. The second is that, even if monotonicity is satisfied, you might not be interested in the causal effect specifically among the compliers, especially because you can't even identify who the compliers are (see Section 9.5.2). Rather, the regular ATE is often a more useful quantity to know.

9.6 More General Settings for ATE Identification

A common more general setting instrumental variable setting is to consider that the outcome is generated by a complex function of treatment and observed covariates plus some additive unobserved confounders:

$$Y := f(T, W) + U \tag{9.32}$$

See, for example, Hartford et al. [75] and Xu et al. [76] for using deep learning to model f. See references in those papers for using other models such as kernel methods to model f. In those models and given that U enters in the structural equation for Y additively, you can get identification with instrumental variables.

[75]: Hartford et al. (2017), 'Deep IV: A Flexible Approach for Counterfactual Prediction'
[76]: Xu et al. (2020), *Learning Deep Features in Instrumental Variable Regression*

Alternatively, we could give up on point identification of causal effects, instead settle for set identification (partial identification), and use instrumental variables to get bounds on causal effects. For more on that, see Pearl [18, Section 8.2]. Additionally, settling for identifying a set, rather than a point, allows us to relax the additive noise assumption above in Equation 9.32. For example, Kilbertus et al. [77] considers the setting where U doesn't enter the structural equation for Y additively:

[18]: Pearl (2009), *Causality*

[77]: Kilbertus et al. (2020), *A Class of Algorithms for General Instrumental Variable Models*

$$Y := f(T, U) \tag{9.33}$$

10 Difference in Differences

Note: the following chapter is much more rough than usual and currently does not contain as many figures and intuition as the corresponding lecture.

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10.1 Preliminaries

We first introduced the unconfoundedness assumption (Assumption 2.1) in Chapter 2:

$$(Y(1), Y(0)) \perp\!\!\!\perp T \tag{10.1}$$

Recall that this is equivalent to assuming that there are no unblocked backdoor paths from T to Y in the causal graph. When this is the case, we have that association is causation. In other words, it gives us the following (hopefully familiar) identification of the ATE:

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0)] &= \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] && (10.2)\\
&= \mathbb{E}[Y(1) \mid T = 1] - \mathbb{E}[Y(0) \mid T = 0] && (10.3)\\
&= \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0] && (10.4)
\end{aligned}$$

where we used this unconfoundedness in Equation 10.3.

However, the ATE is not the only average causal effect that we might be interested in. It is often the case that practioners are interested in the ATE specifically in the treated subpopulation. This is known as the *average treatment effect on the treated* (ATT): $\mathbb{E}[Y(1) - Y(0) \mid T = 1]$. We can make a weaker assumption if we are only interested in the ATT, rather than the ATE:

$$Y(0) \perp\!\!\!\perp T \tag{10.5}$$

We only have to assume that $Y(0)$ is unconfounded here, rather than that both $Y(0)$ and $Y(1)$ are unconfounded. We show this in the following proof:

$$\begin{aligned}
\mathbb{E}[Y(1) - Y(0) \mid T = 1] &= \mathbb{E}[Y(1) \mid T = 1] - \mathbb{E}[Y(0) \mid T = 1] && (10.6)\\
&= \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y(0) \mid T = 1] && (10.7)\\
&= \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y(0) \mid T = 0] && (10.8)\\
&= \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0] && (10.9)
\end{aligned}$$

where we used this weaker unconfoundedness in Equation 10.8.

We are generally interested in the ATT estimand with difference-in-differences, but we will use a different identifying assumption.

10.2 Introducing Time

We will now introduce the time dimension. Using information from the time dimension will be key for us to get identification without assuming the usual unconfoundedness. We'll use τ for the variable for time.

Setting As usual, we have a treatment group ($T = 1$) and a control group ($T = 0$). However, now there is also time, and the treatment group only gets the treatment after a certain time. So we have some time $\tau = 1$ that denotes a time after the treatment has been administered to the treatment group and some time $\tau = 0$ that denotes some time before the treatment has been administered to the treatment group. Because the control group never gets the treatment, the control group hasn't received treatment at either of time $\tau = 0$ or at time $\tau = 1$. We will denote the random variable for potential outcome under treatment t at time τ as $Y_\tau(t)$. Then, the causal estimand we're interested in is the average difference in potential outcomes after treatment has been administered (in time period $\tau = 1$) in the treatment group:

$$\mathbb{E}[Y_1(1) - Y_0(1) \mid T = 1] \tag{10.10}$$

In other words, we're interested in the ATT after the treatment has been administered.

10.3 Identification

10.3.1 Assumptions

You can just treat Y_1 and Y_0 as two different random variables. So even though we have a time subscript now, we still have trivial identification via consistency (recall Assumption 2.5) when the value inside of the parenthesis for the potential outcome matches the conditioning value for T:

Assumption 10.1 (Consistency) *If the treatment is T, then the observed outcome Y_τ at time τ is the potential outcome under treatment T. Formally,*

$$\forall \tau, \quad T = t \implies Y_\tau = Y_\tau(t) \tag{10.11}$$

We could write this equivalently as follow:

$$\forall \tau, \quad Y_\tau = Y_\tau(T) \tag{10.12}$$

Consistency is what tells us that the causal estimand $\mathbb{E}[Y_\tau(1) \mid T = 1]$ equals the statistical estimand $\mathbb{E}[Y_\tau \mid T = 1]$, and, similarly, that $\mathbb{E}[Y_\tau(0) \mid T = 0] = \mathbb{E}[Y_\tau \mid T = 0]$. In contrast, $\mathbb{E}[Y_\tau(1) \mid T = 0]$ and $\mathbb{E}[Y_\tau(0) \mid T = 1]$ are counterfactual causal estimands, so consistency does not directly identify these quantities for us. Note: In our derivations in this chapter, we are also implicitly assuming the no interference assumption (Assumption 2.4) extended to this setting where we have a time subscript.

We have now arrived at the defining assumption of difference-in-differences: *parallel trends*. This assumption states that the trend (over time) in the treatment group would match the trend in the control group (over time) if the treatment group were not given treatment.

Assumption 10.2 (Parallel Trends)

$$\mathbb{E}[Y_1(0) - Y_0(0) \mid T = 1] = \mathbb{E}[Y_1(0) - Y_0(0) \mid T = 0] \tag{10.13}$$

This is like an assumption about unconfoundedness between *difference*:

$$(Y_1(0) - Y_0(0)) \perp\!\!\!\perp T \tag{10.14}$$

Regular unconfoundedness:

$$Y(0) \perp\!\!\!\perp T \tag{10.5 revisisted}$$

So you could see this as like the regular unconfoundedness we saw in Equation 10.5, but where treatment is independent of a *difference* of potential outcomes, rather than being independent of the potential outcome themselves.

Then, we need one final assumption. This is the assumption that the treatment has no effect on the treatment group before it is administered.

Assumption 10.3 (No Pretreatment Effect)

$$\mathbb{E}[Y_0(1) - Y_0(0) \mid T = 1] = 0 \tag{10.15}$$

This assumption may seem like it's obviously true, but that isn't necessarily the case. For example, if participants anticipate the treatment, then they might be able to

10.3.2 Main Result and Proof

Using the assumptions in the previous section, we can show that the ATT is equal to the difference between the differences across time in each treatment group. We state this mathematically in the following proposition.

Proposition 10.1 (Difference-in-differences Identification) *Given consistency, parallel trends, and no pretreatment effect, we have the following:*

$$\begin{aligned}&\mathbb{E}[Y_1(1) - Y_1(0) \mid T = 1] \\ &\quad = (\mathbb{E}[Y_1 \mid T = 1] - \mathbb{E}[Y_0 \mid T = 1]) - (\mathbb{E}[Y_1 \mid T = 0] - \mathbb{E}[Y_0 \mid T = 0])\end{aligned} \tag{10.16}$$

Active reading exercise: How would you estimate the statistical estimand on the right-hand side of Equation 10.16?

Proof. As usual, we start with linearity of expectation:

$$\mathbb{E}[Y_1(1) - Y_1(0) \mid T = 1] = \mathbb{E}[Y_1(1) \mid T = 1] - \mathbb{E}[Y_1(0) \mid T = 1] \tag{10.17}$$

We can immediately identify the treated potential outcome in the treated group using consistency

$$= \mathbb{E}[Y_1 \mid T = 1] - \mathbb{E}[Y_1(0) \mid T = 1] \tag{10.18}$$

So we've identified the first term, but the second term remains to be identified. To do that, we'll solve for this term in the parallel trends assumption:[1]

$$\mathbb{E}[Y_1(0) \mid T = 1] = \mathbb{E}[Y_0(0) \mid T = 1] + \mathbb{E}[Y_1(0) \mid T = 0] - \mathbb{E}[Y_0(0) \mid T = 0] \tag{10.19}$$

We can use consistency to identify the last two terms:

$$= \mathbb{E}[Y_0(0) \mid T = 1] + \mathbb{E}[Y_1 \mid T = 0] - \mathbb{E}[Y_0 \mid T = 0] \tag{10.20}$$

But the first term is counterfactual. This is where we need the no pretreatment effect assumption:[2]

$$= \mathbb{E}[Y_0(1) \mid T = 1] + \mathbb{E}[Y_1 \mid T = 0] - \mathbb{E}[Y_0 \mid T = 0] \tag{10.21}$$

Now, we can use consistency to complete the identification:

$$= \mathbb{E}[Y_0 \mid T = 1] + \mathbb{E}[Y_1 \mid T = 0] - \mathbb{E}[Y_0 \mid T = 0] \tag{10.22}$$

Now that we've identified $\mathbb{E}[Y_1(0) \mid T = 1]$, we can plug Equation 10.22 back into Equation 10.18 to complete the proof:

$$\begin{aligned}
&\mathbb{E}[Y_1(1) \mid T = 1] - \mathbb{E}[Y_1(0) \mid T = 1] \\
&\quad = \mathbb{E}[Y_1 \mid T = 1] - (\mathbb{E}[Y_0 \mid T = 1] + \mathbb{E}[Y_1 \mid T = 0] - \mathbb{E}[Y_0 \mid T = 0]) && (10.23) \\
&\quad = (\mathbb{E}[Y_1 \mid T = 1] - \mathbb{E}[Y_0 \mid T = 1]) - (\mathbb{E}[Y_1 \mid T = 0] - \mathbb{E}[Y_0 \mid T = 0]) && (10.24)
\end{aligned}$$

□

10.4 Major Problems

The first major problem with the difference-in-differences methods for causal effect estimation is that the parallel trends assumption is often not satisfied. We can try to fix this by controlling for relevant confounders W and trying to satisfy the controlled parallel trends assumption:

Assumption 10.4 (Controlled Parallel Trends)

$$\mathbb{E}[Y_1(0) - Y_0(0) \mid T = 1, W] = \mathbb{E}[Y_1(0) - Y_0(0) \mid T = 0, W] \tag{10.25}$$

This is commonly done in practice, but it still might not be possible to satisfy this weaker version of the parallel trends assumption. For example, if there is an interaction term between treatment T and time τ in the structural equation for Y, we will never have parallel trends.

Additionally, the parallel trends assumption is scale-specific. For example, if we satisfy parallel trends, this doesn't imply that we satisfy parallel trends under some transformation of Y. The logarithm is one common

[1] Parallel trends assumptions (Assumption 10.2):

$$\mathbb{E}[Y_1(0) \mid T = 1] - \mathbb{E}[Y_0(0) \mid T = 1] = \mathbb{E}[Y_1(0) \mid T = 0] - \mathbb{E}[Y_0(0) \mid T = 0]$$ (10.13 revisited)

[2] No pretreatment effect assumption (Assumption 10.3)

$$\mathbb{E}[Y_0(1) \mid T = 1] - \mathbb{E}[Y_0(0) \mid T = 1] = 0$$ (10.15 revisited)

such transformation. This is because the parallel trends assumption is an assumption about *differences*, which makes it not fully nonparametric. In this sense, the parallel trends assumption is semi-parametric. And, similarly, the difference-in-differences method is a semi-parametric method.

11 Counterfactuals

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12 More Chapters Coming

Appendix

Proofs A

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A.2 Proof of Propensity Score Theorem (7.1) 103
A.3 Proof of IPW Estimand (7.18) 104

A.1 Proof of Equation 6.1 from Section 6.1

Claim Given the causal graph is Figure A.1, $P(m \mid do(t)) = P(m \mid t)$.

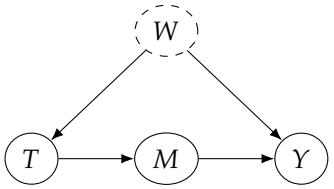

Figure A.1: Causal graph where W is unobserved, so we cannot block the backdoor path $T \leftarrow W \rightarrow Y$.

Proof. We first apply the Bayesian network factorization (Definition 3.1):

$$P(w, t, m, y) = P(w)\,P(t \mid w)\,P(m \mid t)\,P(y \mid w, m) \tag{A.1}$$

Next, we apply the truncated factorization (Proposition 4.1):

$$P(w, m, y \mid do(t)) = P(w)\,P(m \mid t)\,P(y \mid w, m) \tag{A.2}$$

Finally, we marginalize out w and y:

$$\sum_w \sum_y P(w, m, y \mid do(t)) = \sum_w \sum_y P(w)\,P(m \mid t)\,P(y \mid w, m) \tag{A.3}$$

$$P(m \mid do(t)) = \left(\sum_w P(w)\right) P(m \mid t) \left(\sum_y P(y \mid w, m)\right) \tag{A.4}$$

$$= P(m \mid t) \tag{A.5}$$

□

A.2 Proof of Propensity Score Theorem (7.1)

Claim $(Y(1), Y(0)) \perp\!\!\!\perp T \mid W \implies (Y(1), Y(0)) \perp\!\!\!\perp T \mid e(W)$.

Proof. Assuming $(Y(1), Y(0)) \perp\!\!\!\perp T \mid W$, we will prove $(Y(1), Y(0)) \perp\!\!\!\perp T \mid e(W)$ by showing that $P(T = 1, \mid Y(t), e(W))$ does not depend on $Y(t)$, where $Y(t)$ is either potential outcome.

First, because T is binary, can turn this probability into an expectation:

$$P(T = 1, \mid Y(t), e(W)) = \mathbb{E}[T \mid Y(t), e(W)] \tag{A.6}$$

Then, using the law of iterated expectations, we can introduce W:

$$= \mathbb{E}\left[\mathbb{E}[T \mid Y(t), e(W), W] \mid Y(t), e(W)\right] \tag{A.7}$$

Because we have now conditioned on all of W and $e(W)$ is a function of W, it is redundant, so we can remove $e(W)$ from the inner expectation:

$$= \mathbb{E}\left[\mathbb{E}[T \mid Y(t), W] \mid Y(t), e(W)\right] \tag{A.8}$$

Now, we apply the unconfoundedness assumption we started with to remove $Y(t)$ from the inner expectation:

$$= \mathbb{E}\left[\mathbb{E}[T \mid W] \mid Y(t), e(W)\right] \tag{A.9}$$

Again, using the fact that T is binary, we can reduce the inner expectation to $P(T = 1 \mid W) \triangleq e(W)$, something that is already conditioned on:

$$= \mathbb{E}\left[P(T = 1 \mid W) \mid Y(t), e(W)\right] \tag{A.10}$$
$$= \mathbb{E}\left[e(W) \mid Y(t), e(W)\right] \tag{A.11}$$
$$= e(W) \tag{A.12}$$

Because this does not depend on $Y(t)$, we've proven that T is independent of $Y(t)$ given $e(W)$. □

A.3 Proof of IPW Estimand (7.18)

Claim Under unconfoundedness and positivity, $\mathbb{E}[Y(t)] = \mathbb{E}\left[\frac{\mathbb{1}(T=t)Y}{P(t|W)}\right]$.

Proof. We will start with the statistical estimand that we get from the adjustment formula (Theorem 2.1). Given unconfoundedness and positivity, the adjustment formula tells us

$$\mathbb{E}[Y(t)] = \mathbb{E}[\mathbb{E}[Y \mid t, W]] \tag{A.13}$$

We'll assume the variable are discrete to break these expectations into sums (replace with integrals if continuous):

$$= \sum_w \left(\sum_y y\, P(y \mid t, w) \right) P(w) \tag{A.14}$$

To get $P(t \mid w)$ in there, we multiply by $\frac{P(t|w)}{P(t|w)}$:

$$= \sum_w \sum_y y\, P(y \mid t, w)\, P(w)\, \frac{P(t \mid w)}{P(t \mid w)} \tag{A.15}$$

Then, noticing that $P(y \mid t, w)\, P(t \mid w)\, P(w)$ is the joint distribution:

$$= \sum_w \sum_y y\, P(y, t, w)\, \frac{1}{P(t \mid w)} \tag{A.16}$$

$\sum_y y\, P(y, t, w)$ is nearly $\sum_y y\, P(y) = \mathbb{E}[Y]$, but because of $T = t$ and $W = w$ are in the probability, the terms of this sum are only non-zero if $T = t$ and $W = w$. Therefore, we get the indicator random variable for this event in the expectation that is over all three random variables (T, W, and Y):

$$= \sum_w \mathbb{E}\left[\mathbb{1}(T = t, W = w)\, Y\right] \frac{1}{P(t \mid w)} \tag{A.17}$$

Now, the $\sum_w \frac{1}{P(t|w)}$ that remains is a weighted expectation over W. Integrating this means that because we are now marginalizing over W, w becomes a random variable (W) and the the $W = w$ inside the indicator becomes redundant. This gives us the following:

$$= \mathbb{E}\left[\frac{\mathbb{1}(T = t)\, Y}{P(t \mid W)}\right] \tag{A.18}$$

□

Note: For some people, it might be more natural to skip straight from Equation A.16 to Equation A.18.

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