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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1.1 Simpson's Paradox

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.

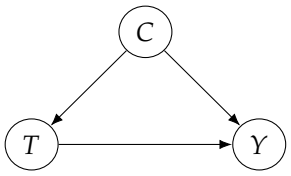


Figure 1.1: Causal structure of scenario 1, where condition C is a common cause of treatment T and mortality Y. Given this causal structure, treatment B is preferable.

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.

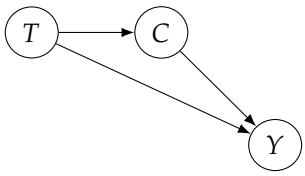


Figure 1.2: Causal structure of scenario 2, where treatment T is a cause of condition C. Given this causal structure, treatment A is preferable.

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*

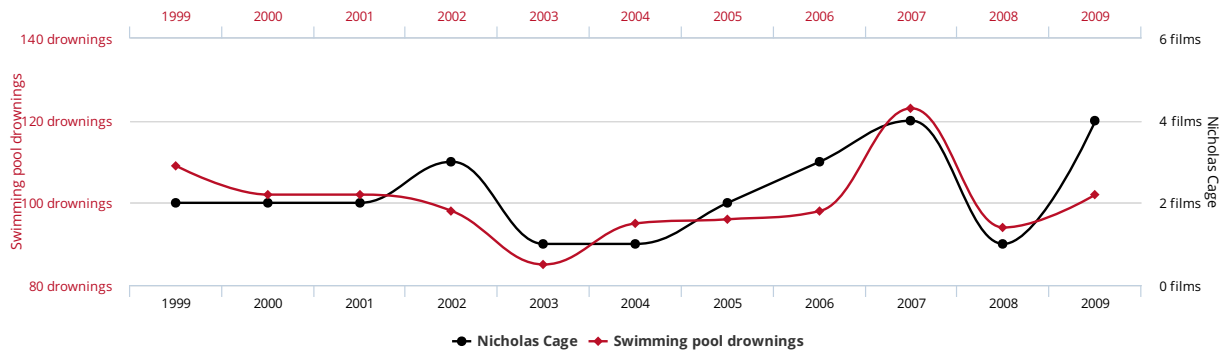


Figure 1.3: The yearly number of movies Nicolas Cage appears in correlates with the yearly number of pool drownings [1].

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 binary. For any given amount of association, it does not need to be "all the association is causation" or "no causation." It is possible to have *some* causation while having a large amount of association. 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.

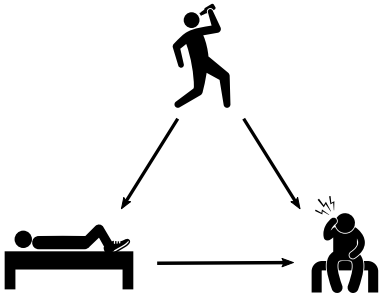


Figure 1.4: Causal structure, where drinking the night before is a common cause of sleeping with shoes on and of waking up with a headaches.

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.

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]

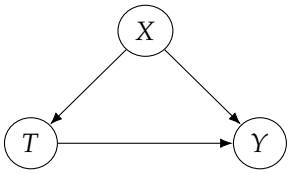


Figure 2.1: Causal structure of X confounding the effect of T on Y.

[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 sum of the $Y(0)$ column, ignoring the question marks, and subtracting that from the sum 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)

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

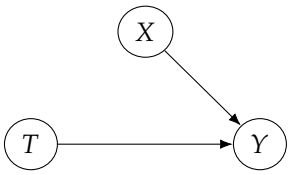


Figure 2.2: Causal structure when the treatment assignment mechanism is ignorable. Notably, this means there's no arrow from X to T, which means there is no confounding.

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.

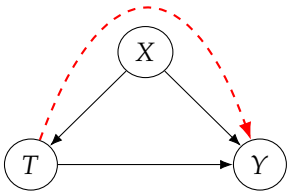


Figure 2.3: Causal structure of X confounding the effect of T on Y. We depict the confounding with a red dashed line.

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.

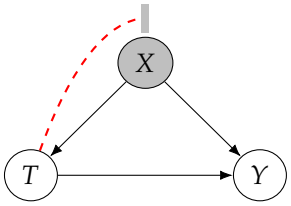


Figure 2.4: Illustration of conditioning on X leading to no confounding.

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 or *do*-operator in it. We will use the phrase *statistical estimand* to denote the complement: any estimand that does not

contain a potential outcome or *do*-operator in it. For an example, recall the adjustment formula (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: $\mathbb{E}_X\left[\mathbb{E}[Y \mid T = 1, X] - \mathbb{E}[Y \mid T = 0, X]\right]$.[14]

[14] 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.

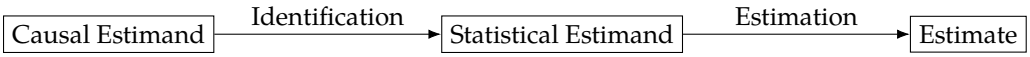


Figure 2.5: The Identification-Estimation Flowchart – a flowchart that illustrates the process of moving from a target causal estimand to a corresponding estimate, through identification and estimation.

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.[15] 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'

[15] 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']]`,[16] 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 $\frac{|.85-1.05|}{1.05} \times 100\% = \mathbf{19}\%$.

[16] 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):

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

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

```
y = df['blood_pressure']
model = LinearRegression()
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. In other words, we have compressed all of $\mathbb{E}[Y \mid t]$, 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,[17] 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 [11, Sections 6.2 and 6.3] for a more complete critique of using the coefficient in front of treatment as the ATE estimate.

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

[11]: 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.

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.

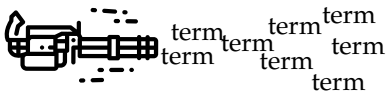


Figure 3.1: Terminology machine gun

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.

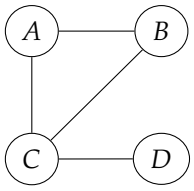


Figure 3.2: Undirected graph

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.

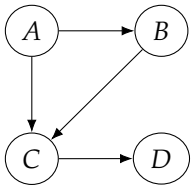


Figure 3.3: Directed graph

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*. If there

are no cycles in a directed graph, the graph is known 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 $X \rightarrow Z \leftarrow Y$ is known as an *immorality*. Seriously; that's a real term in graphical models. For example, if the $A \rightarrow B$ edge did not exist in Figure 3.3, then $A \rightarrow C \leftarrow B$ would be an 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 $P(x_4 \mid x_3, x_2, x_1)$, we could model it as $P(x_4 \mid x_3)$ if we have reason to believe that X_4 only locally depends on X_3. In fact, in the corresponding graph in Figure 3.4, 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.

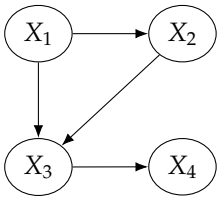


Figure 3.4: Four node DAG where X_4 locally depends on only X_3.

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 of 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

$$P(x_1, x_2, x_3, x_4) = P(x_1)\,P(x_2|x_1)\,P(x_3|x_2, x_1)\,P(x_4|x_3, x_2, x_1)\,. \quad (3.2)$$

If P is Markov with respect to the graph[1] in Figure 3.4, then we can simply the last factor:

$$P(x_1, x_2, x_3, x_4) = P(x_1)\,P(x_2|x_1)\,P(x_3|x_2, x_1)\,P(x_4|x_3)\,. \quad (3.3)$$

If we further remove edges, removing $X_1 \to X_2$ and $X_2 \to X_3$ as in Figure 3.5, we can further simplify the factorization of P:

$$P(x_1, x_2, x_3, x_4) = P(x_1)\,P(x_2)\,P(x_3|x_1)\,P(x_4|x_3)\,. \quad (3.4)$$

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 [12, 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 of 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.

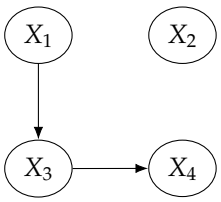


Figure 3.5: Four node DAG with even more independencies.

[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.

[12]: 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., 13, Section 6.5.3].

[13]: 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 3.6, 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 3.6.

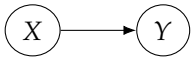


Figure 3.6: Two connected nodes

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 of their 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.

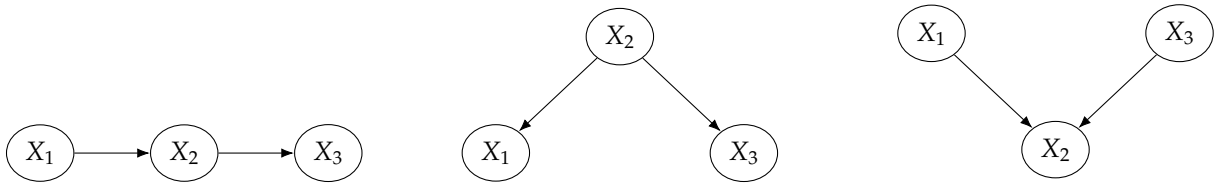
As we will see in the next few sections, the main assumptions that we need for our causal graphical models to tell us how association and causation flow between variables are the following two:

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 3.7a), forks (Figure 3.7b), immoralities (Figure 3.7c), two unconnected nodes (Figure 3.8), and two connected nodes (Figure 3.9).



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

Figure 3.7: 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 3.8, 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 3.8: 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 3.8.

In contrast, if there is an edge between the two nodes (as in Figure 3.9), 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.

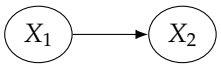


Figure 3.9: Two connected nodes

[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 3.7), starting with chain graphs.

3.5 Chains and Forks

Chains (Figure 3.10) and forks (Figure 3.11) 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,

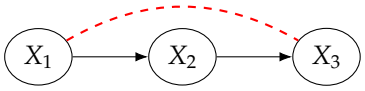


Figure 3.10: Chain with flow of association drawn as a dashed red arc.

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., 14, Section 2.2].

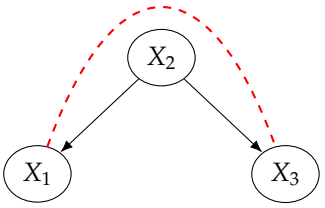


Figure 3.11: Fork with flow of association drawn as a dashed red arc.

[14]: 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 3.10 and Figure 3.11. In the chain graph (Figure 3.10), 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 3.11), 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 only locally depends on 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 3.12 and Figure 3.13. 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.

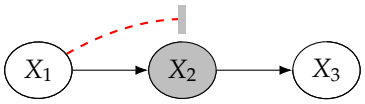


Figure 3.12: Chain with association blocked by conditioning on X_2.

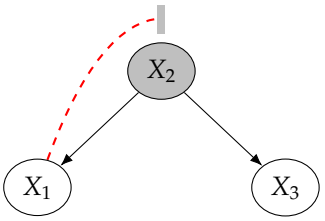


Figure 3.13: Fork with association blocked by conditioning on X_2.

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 3.13).

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 3.14). 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, $X_1 \not\perp\!\!\!\perp X_3$. Look at the graph structure and think about it a bit. Why would X_1 and X_3 be associated? One isn't the descendent 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:

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

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

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

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

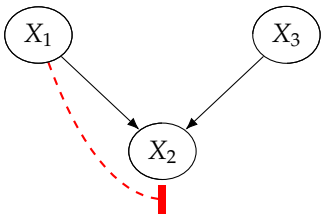


Figure 3.14: Immorality with association blocked by a collider.

We illustrate the independence of X_1 and X_3 in Figure 3.14 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.

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 3.15). 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

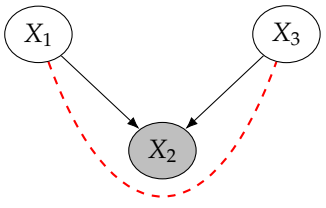


Figure 3.15: Immorality with association unblocked by conditioning on the collider.

a relationship or not)? And what if 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 3.15 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 complexities are outside the scope of this book.

Active reading exercise: Come up with your own example of an immortality 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 3.14 to Figure 3.15.

We can also illustrate this with a scatter plot. In TODO, 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 (top triangle), we are left with a clear negative association. This phenomenon is known as *Berkson's paradox*. The fact that 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*

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:

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

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 descendent, 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 hold:*

1. *Along the path, there is a chain $\cdots \to W \to \ldots$ or a fork $\cdots \leftarrow W \to \ldots$, 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 [15].*

[15]: 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.

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 equivalent all [see, e.g., 12, 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.

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

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 3.16, we depict the confounding association in red and the causal association in blue.

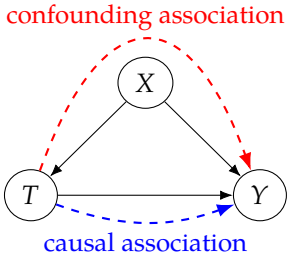


Figure 3.16: Causal graph depicting an example of how confounding association and causal association flow.

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.

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 takes 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.

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 when all of X's causal effect on Y would flow through it's outgoing edges; once those are removed, the only association that remains is purely non-causal association.

In Figure 3.17, 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.

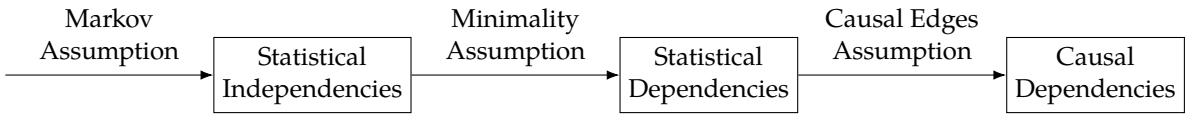


Figure 3.17: A flowchart that illustrates what kind of claims we can make about our data as we add each additional important assumption.

Causal Models 4

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.

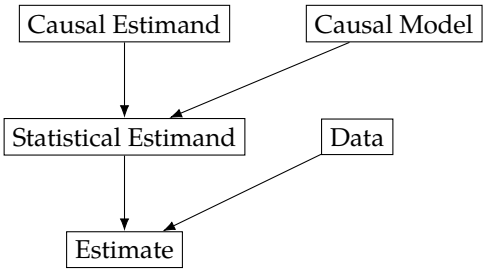


Figure 4.1: The Identification-Estimation Flowchart – a flowchart that illustrates the process of moving from a target causal estimand to a corresponding estimate, through identification and estimation. In contrast to Figure 2.5, this version is augmented with a causal model and data.

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:

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

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:

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

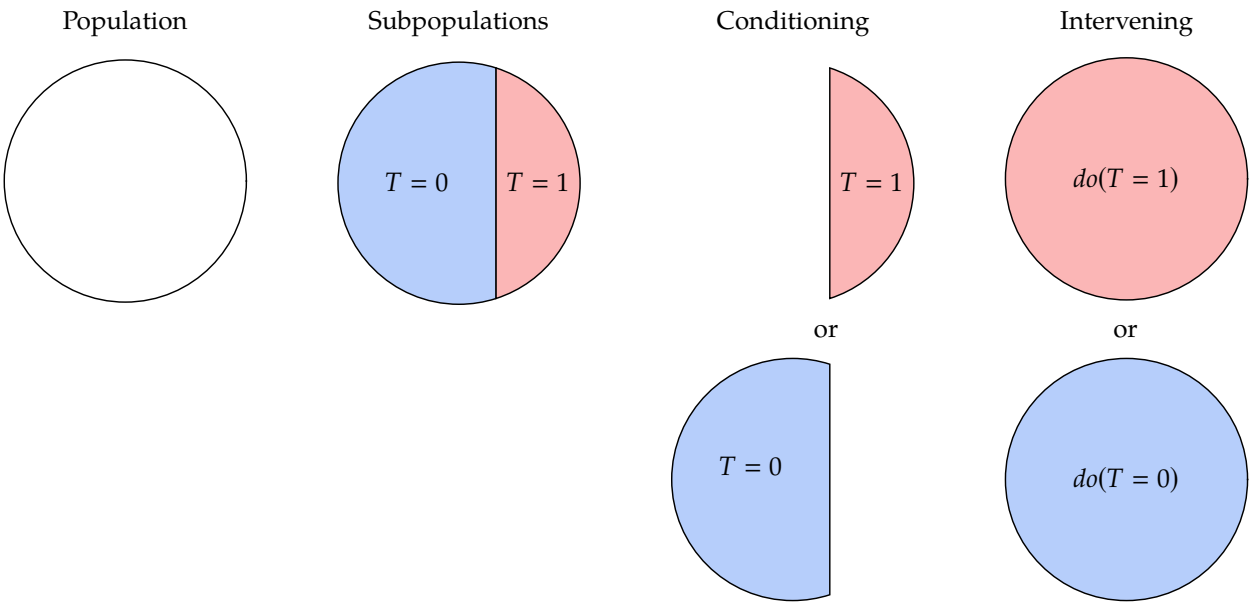


Figure 4.2: Illustration of the difference between conditioning and intervening

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 8.

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.

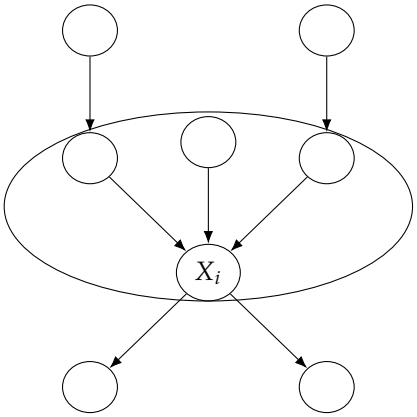


Figure 4.3: A causal graph with the causal mechanism that generates X_i depicted inside an ellipse.

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 $\{1, 2, \ldots, n\}$.

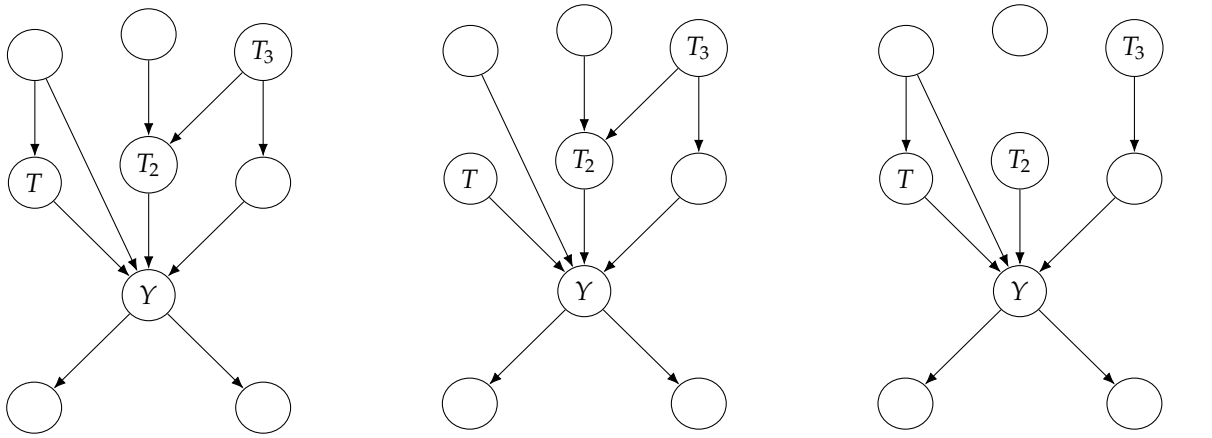
In the second part of the above assumption, we could have alternatively said $P(x_i \mid \text{pa}_i) = 1$ if x_i is *consistent with the intervention*[2] and 0 otherwise. More explicitly, we will say (in the future) that if $i \in S$, a value x_i is consistent with the intervention if x_i equals the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

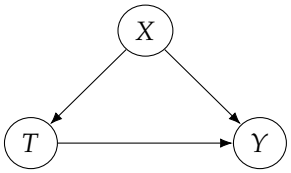


Figure 4.5: Simple causal structure where X counfounds the effect of T on Y and where X is the only confounder.

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

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.

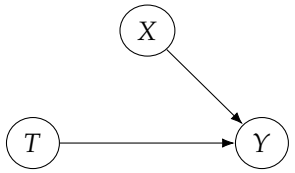


Figure 4.6: Manipulated graph that results from intervening on T, when the original graph is

[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) and that W satisfies the backdoor criterion (Definition 4.1)*

[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.

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.

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

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

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

□

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 x, 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.

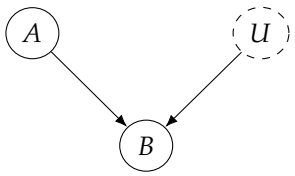


Figure 4.7: Graph for simple structural equation. The dashed node U means that U is unobserved.

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:

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

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 $\{A, U_B, U_C, U_D\}$.

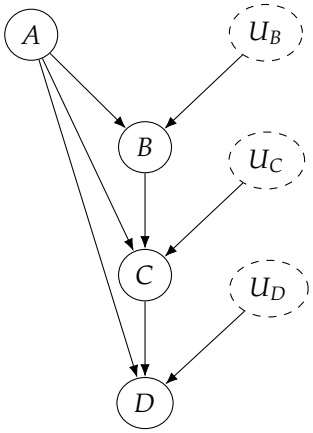


Figure 4.8: Graph for the structural equations in Equation 4.24.

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:

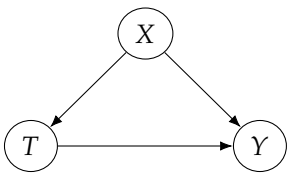


Figure 4.9: Basic causal graph

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

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.

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

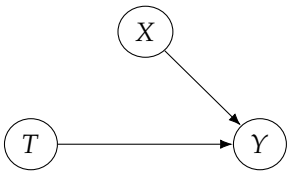


Figure 4.10: Basic causal with the the incoming edges to T removed, due to the intervention $do(T = t)$.

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

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

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

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 8.

[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.

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).

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 [17, Section 11.3.1 and 11.3.3] for more information on this topic.

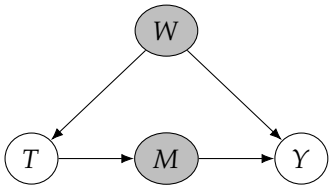


Figure 4.11: Causal graph where all causation is blocked by conditioning on M.

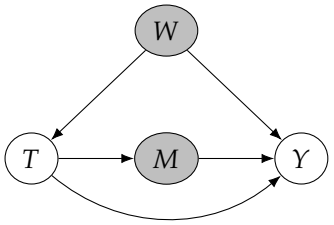


Figure 4.12: Causal graph where part of the causation is blocked by conditioning on M.

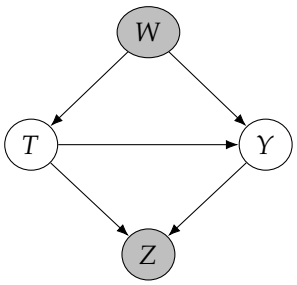


Figure 4.13: Causal graph where conditioning on the collider Z induces bias.

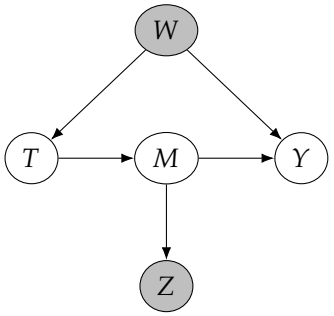


Figure 4.14: Causal graph where the child of a mediator is conditioned on.

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 *pretreatment covariates*.

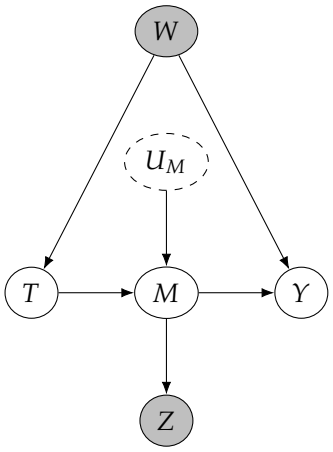


Figure 4.15: Magnified causal graph where the child of a mediator is conditioned on.

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 [18].

4.6 Example Applications of the Backdoor Adjustment

4.6.1 Association vs. Causation in a Toy Example

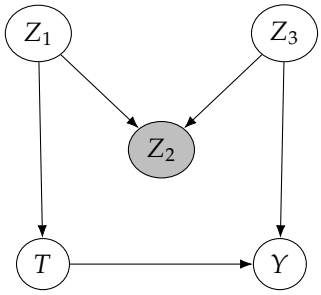


Figure 4.16: Causal graph depicting M-Bias.

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:

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

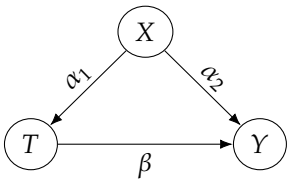


Figure 4.17: Causal graph for toy example

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:

$$\begin{aligned}\mathbb{E}[Y \mid T = t] &= \mathbb{E}[\beta T + \alpha_2 X \mid T = t] && (4.34)\\ &= \beta t + \alpha_2 \mathbb{E}[X \mid T = t] && (4.35)\\ &= \beta t + \frac{\alpha_2}{\alpha_1} t && (4.36)\end{aligned}$$

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 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.

[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'

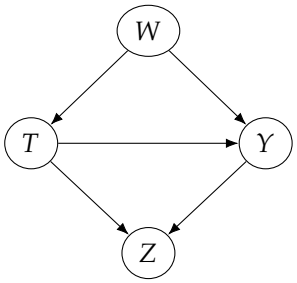


Figure 4.18: Causal graph for the blood pressure example. T is sodium intake. Y is blood pressure. W is age. And, importantly, the amount of protein excreted in urine Z is a collider.

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

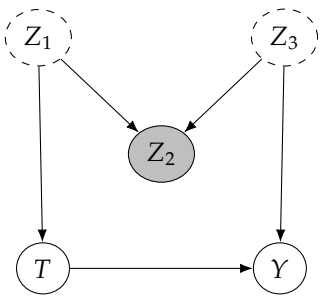


Figure 4.19: Causal graph depicting M-Bias that can only be avoided by not conditioning on the collider Z_2. This is due to the fact that the dashed nodes Z_1 and Z_3 are unobserved.

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.

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 [19, 20], or if you want a textbook, you can find them in [17, Chapter 7.3]. To see proofs of the equivalence of all three forms of the Markov assumption, see, for example, [12, Chapter 3].

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

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

[12]: 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 [21]. Consistency (Assumption 2.5) follows from the axioms of SCMs (see [17, Corollary 7.3.2] and [22]). Positivity (Assumption 2.3) is still a very important assumption that we must make, though it is sometimes neglected in the graphical models literature.

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

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

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

5 Randomized Experiments

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,*

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

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

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

Although we have proven that randomization implies covariate balance, we have not proven that that covariate balance implies identifiability. The intuition is that covariance 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. We'll now prove that $P(y \mid do(T = t)) = P(y \mid T = t)$. For the proof, the main property we utilize is that covariate balance implies X and T are independent.

Proof. First, let X be a sufficient adjustment set. This is the case with randomization since we know that randomization balances everything, not just the observed covariates. Then, we have the following from the backdoor adjustment (Theorem 4.2):

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

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 5.8).

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 in 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 $T \leftarrow X \rightarrow Y$.

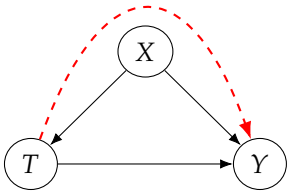


Figure 5.1: Causal structure of X confounding the effect of T on Y.

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:

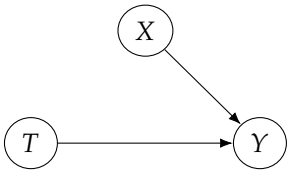


Figure 5.2: Causal structure when we randomize treatment.

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

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.

General Identification 6

6.1 Coming Soon

7 Estimation

7.1 Coming Soon

Counterfactuals 8

8.1 Coming Soon

More Chapters Coming 9

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