Econometrics for Social Scientists

Volume I: Causal Inference and Estimation

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Preface

This book is the 1st book in a sequence on **Econometric Methods for Political Analysis**.

- 1. Volume I: Causal Inference and Estimation (this book) introduces causal inference, randomised experiments, the ordinary least squares estimator, the instrumental variables estimator, and quasi-experimental designs.
- 2. Volume II: Further Causal Estimators expands on the first book by discusses other estimators, such as the maximum likelihood estimator, selection on observables estimators, and generalised methods of moments estimator.
- 3. Volume III: Prediction and Forecasting introduces prediction methods such as non-linear prediction, classification, cross-validation, as well as time-series models.
- 4. Volume IV: Multivariate Measurement discusses topics in latent variable measurement, dimensional reduction, and measurement of textual data for statistical analysis.

This series is designed to be both an approachable, but also rigorous, introduction to Econometrics and the use of statistical methods for the analysis of social and political actors and phenomena.

I assume a solid understanding of basic probability and statistics, including the topics of conditional probabilities, random variables and distributions, expectation/mean and variance, and correlation.

I also assume a solid understanding of mathematics, including algebra, single variable calculus, and some linear algebra. While you will be able to still learn from this book without a solid mathematical background, you will gain much more from understanding the mathematics behind the methods.

To see what mathematics is specifically required, or to refresh on the mathematics needed, consult the **Quantitative Methods** sequence (particularly the 1st volume and some topics in the 2nd volume).

I will also add some reference code for the R-programming language and Stata in case you are interested in implementing these methods on your own.

Part I

Introduction to Causal Inference

Causal Inference

1.1 Correlation is not Causation

Econometrics is the field of applying statistical methods to analyse real-world social science data. Econometrics has two primary goals:

- 1. Causal Inference: Establishing how one feature directly causes another feature. This is essential to understanding the world around us and designing better policies. Key point: correlation causation.
- 2. **Predictive Inference/Forecasting:** Given data we have, how can we predict the values of data we do not have? For example, what will sales be next year? GDP? Who will win the next election? What are the likely costs/effects of a policy?

We will focus on causal inference in this book, as it is the most important aspect of econometrics.

Variables are **correlated** when one variable changing is associated with the other variable changing on average. For example, an increase in X causes an increase/decrease in Y would mean X and Y are correlated.

There are three reasons why variables are correlated with each other.

- 1. There could be a causal effect of X on Y, causing the correlation.
- 2. There could be a third variable, W, causing both X and Y to change.
- 3. There is a causal effect of Y on X.

The most important thing about econometrics and causal inference is that <u>correlation does not equal causation</u>. This is because of the 2nd reason listed above - our correlation between X and Y could be caused by a third variable W, called a **confounder**.

For example, *Ice Cream Sales* and *Number of Fatal Shark Attacks* are two highly correlated variables in the United States. Does this mean that selling ice cream causes fatal shark attacks? No!

The reason this relationship exists is because of another variable - the *weather*. The weather, when it is sunny, causes both ice cream sales and more people to go to the beach, which causes more fatal shark attacks. However, there is no direct link between ice cream sales and the number of fatal shark attacks.

The goal of econometrics is to distinguish between correlation and causation. We want to "partial out" the effect of confounding variables and isolate the causal effects.

This book will introduce a series of different **estimators** that will help us isolate and estimate these causal effects.

1.2 Potential Outcomes and Causal Effects

A causal effect is a change in some feature of the world Y, that would directly result from a change in some other feature D. Essentially, change in D causes change in Y.

? Key Definition: Potential Outcomes

Causal effect implies that there are **potential outcomes**. Imagine that there are 2 worlds, that are exactly the same until treatment D occurs. In one world, you get the treatment D, and the other world, you do not get this treatment. Since these 2 worlds are identical besides the treatment D, the difference between the world's Y outcomes are the effect of our treatment D.

In the real world, we only observe one of these realities - either a unit i gets, or does not get, the treatment. The other world that we do not observe is called a **counterfactual**.

Thus, there are two states of the world in the potential outcomes framework:

- The <u>control state</u> D = 0 is the world where a unit does not receive the treatment D. Y_{1i} is the potential outcome for unit i, given it is in the treatment state $D_i = 1$.
- The treatment state D = 1 is identical to the control state, with the only exception that a unit receives the treatment D. Y_{0i} is the potential outcome for unit i, given it is in the control state $D_i = 0$.

The **individual causal effect** τ of the treatment D for any unit i is $\tau_i = Y_{1i} - Y_{0i}$. Since the two states are identical except for treatment D, the resulting difference must be as a result of treatment D. However, in the real world, we do not have parallel worlds (unfortunately) - we only observe one outcome: either unit i gets the treatment $D_i = 1$, or does not get the treatment $D_i = 0$.

? Key Definition: Observed Outcomes

The **observed** Y **outcome** (in the real world) of any unit i is given by the equation:

$$Y_i = D_i \times Y_{1i} + (1 - D_i) \times Y_{0i}$$

This equation might be a little abstract, however, it is easy to understand by plugging D_i in:

$$[Y_i|D_i=0]=0\times Y_{1i}+(1-0)\times Y_{0i}=Y_{0i}$$

$$[Y_i|D_i=1] = 1 \times Y_{1i} + (1-1) \times Y_{0i} = Y_{1i}$$

Intuitively, if the observation is in the control state $D_i = 0$, we observe potential outcome Y_{0i} . When an observation is in the treatment state $D_i = 1$, we observe potential outcome Y_{1i} .

Stable Unit Treatment Value Assumption

Take two units i and j. The Stable Unit Treatment Value Assumption (SUTVA) is the assumption that unit j getting the treatment D, does not affect the outcomes of unit i.

This is important - because if this assumption were to be violated, we would have more than two potential outcomes. If unit i were affected by j's treatment status, we would not only have the potential outcomes of unit i being in treatment or control, but also would have to consider unit j being in treatment or control.

Estimands and Estimator Bias and Consistency 1.3

Causal Estimands

An **estimand** is the quantity we are trying to estimate (i.e. what we are interested in). The "key" estimand is the Average Treatment Effect:

• Key Definition: Average Treatment Effect

Average treatment effect (ATE) is the average of all individual treatment effects:

$$\tau_{ATE} = \mathbb{E}[\tau_i] = \mathbb{E}[Y_{1i} - Y_{0i}] = \frac{1}{n} \left(\sum Y_{1i} - \sum Y_{0i}\right)$$

The conditional average treatment effect (CATE) is the treatment effect of units, given they have some other variable X value. For example, if X is gender, the CATE could be the treatment effect on only females. This is useful if we believe the strength of our causal effect is different in different groups of X

$$\tau_{CATE} = \mathbb{E}[Y_{1i} - Y_{0i}|X = x]$$

Sometimes, our treatment variables are continuous. For example, if we want to estimate how GDP affects democratisation, our treatment variable GDP is not binary. Instead, we can estimate the average causal **effect** - the change in Y resulting from a one unit increase in D.

There are also other treatment effects we can use to estimate. The average treatment effect on the treated (ATT) is the treatment effect of only units who received the treatment $D_i = 1$

$$\tau_{ATT} = \mathbb{E}[Y_{1i} - Y_{0i}|D_i = 1]$$

We can also estimate the average treatment effect on the controls (ATC), which is the same as above but only for units in control $D_i = 0$.

Estimator Bias and Consistency

The above causal estimands are not directly calculable, and we to estimate them with an **estimator**.

Bias is when an estimator consistently and systematically poorly estimates the estimand.

Or in other words, an estimator is biased, if on average, the estimate of our estimand (over many tries of estimation), is not actually the true value of the estimand. That means something is consistently off with our estimator - we might be consistently overestimating by 5%, or underestimating, etc. Mathematically:

 $\mathbb{E}[\hat{\theta}_i] = \theta$, where $\hat{\theta}$ is the estimate, and θ is the true value of the estimand

Consistency or Variance is the difference between our estimations derived from our estimator.

For example, you might have an unbiased estimator, where our average estimate is the actual causal estimand. However, while the average is correct, the variance of our estimates is very wide.

Ideally, we want an unbiased estimator that has low variance. We will explore many different types of estimators for causal effects throughout this book, each with its own bias and variance.

1.4 Naive Estimator of Observed Outcomes

The **naive estimator** is an estimator that only compares our observed outcomes, without any comparison to the counterfactual potential outcomes:

$$\hat{\tau}_{\text{naive}} = \mathbb{E}[Y_{1i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i = 0]$$

Or more intuitively, the average observed outcome Y of those in the treatment group, minus the average observed outcome Y of those in the control group.

This is often what many people initially do when trying to find a causal effect. Essentially, we are comparing units that are assigned to treatment, and the units that are not assigned to treatment, and their observed outcomes.

• In other words, the naive estimator is looking at the correlation between the treatment D and the observed outcomes of Y, without considering the counterfactual.

The <u>naive estimator is a bad idea</u>. Remember, our treatment effects are supposed to be comparing to two potential outcomes of the same unit. We are supposed to compare Y_{1i} to Y_{0i} .

- However, in this scenario, we are not comparing the potential outcomes of the same individual. We are comparing the outcome of some observation A in treatment Y_{1A} and the outcome of some other observation B in control Y_{0B} .
- But what if observation A and B are different? There outcomes may not be due to the treatment D, but because of the differences between A and B. This is why counterfactual comparison is important
 when we compare the potential outcomes of the same unit in control and treatment groups, we can be confident of the affect of the treatment, since it is the same unit of observation for both groups.

We can prove this mathematically. We start with the naive estimator:

$$\mathbb{E}[Y_{1i}|D_i=1] - \mathbb{E}[Y_{0i}|D_i=0]$$

Then, we do a little algebra trick - we add a new term to this equation, and then subtract the same term. The two new terms thus cancel each other out to 0.

$$= \mathbb{E}[Y_{1i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i = 0] + \mathbb{E}[Y_{0i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i - 1]$$

Then, we rearrange the terms, then simplify, getting the result:

$$\begin{split} &= \mathbb{E}[Y_{1i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i = 1] + \mathbb{E}[Y_{0i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i = 0] \\ &= \mathbb{E}[Y_{1i} - Y_{0i}|D_i = 1] + \mathbb{E}[Y_{0i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i = 0] \end{split}$$

If we look at the final result, we can divide it into 2 parts:

- 1. The first part, $\mathbb{E}[Y_{1i}-Y_{0i}|D_i=1]$, is the average treatment effect of the treated τ_{ATT} that we introduced previously.
- 2. The second part $\mathbb{E}[Y_{0i}|D_i=1] \mathbb{E}[Y_{0i}|D_i=0]$ is what we call the **selection bias**. Intuitively, it is the difference between the treatment and control groups prior to the treatment taking place (hence the potential outcome being Y_{0i}).

1.5 Selection Bias and Confounding Variables

The differences between the treatment and control groups prior to treatment is captured in our naive estimator, which is why our results with the naive estimator are biased.

For example, if we are measuring the question does going to the hospital make you more healthy, and we simply measured the outcomes of people who went to the hospital and did not go to the hospital, we might see that in general, people who did not go to the hospital are healthier!

- Does this mean that going to the hospital makes you unhealthier? No! It is because more unhealthy people choose to go to the hospital in the first place. Thus, the hospital has generally more unhealthy individuals in it. The hospital might perform miracles on these people, but they are still not as healthy as the healthy people who did not need to go to the hospital.
- The differences between the people who chose to go to the hospital versus the people who did not go to the hospital explains the differences in our outcome, not the actual treatment that the hospital provided.

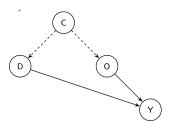
This is selection bias - when our treatment and control groups are fundamentally different and unequal even prior to treatment.

• Key Definition: Confounders

A confounder is a variable that is explaining the differences in the treatment and control groups.

Confounding variables result in selection bias, and are why correlation does not equal causation. In order to accurately calculate causal effects, we need to find some way to eliminate the effect of confounding variables.

For example, look at the figure below:



In the figure above, D is the treatment group, and O is the observed group. C is some confounding variable correlated with D, that affects whether an observation i gets the treatment D or control C.

When we calculate the naive estimate (or correlation), our causal estimate captures both the effect of $D \to Y$, but also the effect of $D \leftrightarrow C \rightarrow O \rightarrow Y$, since D and C are correlated.

- This second effect through the correlation of D and C is called the **backdoor path**.
- Both the direct $D \to Y$ and the backdoor path are included in the naive estimator (and correlation).

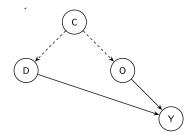
However, the actual causal effect of treatment D on Y is only the section of $D \to Y$, which does not include the backdoor path. So, we need to find some way to only look at $D \to Y$, and eliminate/partial out the effect of the backdoor path.

To make an accurate causal effect estimate, we must get rid of confounding variables, selection bias, and the backdoor path. How do we do this? The best method is randomisation, which we will cover next.

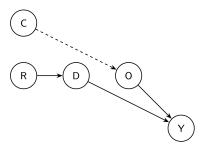
Randomised Controlled Trials

2.1 Random Assignment and Confounders

The assignment mechanism is how we decide which observations receive treatment D. In the last chapter, we discussed how confounder C affects which observations get the treatment, introducing selection bias.



We can address this confounder C and eliminate the backdoor path $D \leftrightarrow C \to O \to Y$ by randomly assigning units into either the treatment or control group:



With random assignment mechanism R, now units are assigned to control randomly, not based on the confounder variable C. Thus, C and D should no longer be correlated, thus removing the backdoor effect, selection bias, and the influence of confounder C.

• Key Definition: Random Assignment

With random assignment, selection bias and the influence of confounder C is eliminated, thus the control group and treatment group should be very similar. Thus, the potential outcomes are independent of treatment/control status:

$$\mathbb{E}[Y_{1i}|D_i=1] \approx \mathbb{E}[Y_{1i}|D_i=0] \quad \text{and} \quad \mathbb{E}[Y_{0i}|D_i=1] \approx \mathbb{E}[Y_{0i}|D_i=0]$$

2.2Difference-in-Means Estimator

If this assumption of random assignment is met, then we can use the naive estimator to estimate the treatment effect. This is because we have eliminated the confounders that cause selection bias.

Mathematically, we can prove this. Recall the naive estimator:

$$\mathbb{E}[Y_{1i} - Y_{0i}|D_i = 1] + \mathbb{E}[Y_{0i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i = 0]$$

The selection bias term $\mathbb{E}[Y_{0i}|D_i=1]-\mathbb{E}[Y_{0i}|D_i=0]=0$ under the above assumptions of randomisation. Thus, there is no longer selection bias, and confounding variables will have been accounted for.

Our observed potential outcomes in our randomised experiments are Y_{1i} and Y_{0i} . We know that the treatment group D_i does not affect our potential outcomes, since units are the same in both treatment and control. Thus we know that:

$$\mathbb{E}[Y_{1i}|D_i=1] = \mathbb{E}[Y_{1i}]$$

$$\mathbb{E}[Y_{0i}|D_i=0] = \mathbb{E}[Y_{0i}]$$

 $\mathbb{E}[Y_{1i}]$ is the expected value of Y in the treatment group. Basically, we just find the mean Y value of all observations of the treatment group.

 $\mathbb{E}[Y_{0i}]$ is the expected value of Y in the control group. Basically, we just find the mean Y value of all observations in the control group.

Thus with these, we can find the average treatment effect:

• Key Definition: ATE Estimate of a Randomised Experiment

The estimate of the average treatment effect of a randomised controlled trial is:

$$\hat{\tau}_{ATE} = \mathbb{E}[\tau_i] = \mathbb{E}[Y_{1i}] - \mathbb{E}[Y_{0i}] = \bar{Y}_t - \bar{Y}_c$$

Where \bar{Y}_t is the average Y value of the treatment group, and \bar{Y}_c is the average Y value of the control group. Thus, the causal effect is simply a difference-in-means of the two groups.

Or in other words, to calculate the average treatment effect of a randomised experiment, we:

- 1. Calculate \bar{Y}_t , the average Y value of all observations in the treatment group.
- 2. Calculate \bar{Y}_c , the average Y value of all observations in the control group.
- 3. Find the difference $\bar{Y}_t \bar{Y}_c$. That is our average treatment effect.

For this estimate of the ATE to be true, there must be random assignment of treatment, and the treatment and control groups must be similar to each other on all confounding variables.

If this assumption is violated (either due to lack of random assignment, or failure of randomisation), we can no longer estimate the causal effects with the difference-in-means estimator. This is because if the control and treatment groups are not similar to each other on all confounders, there is the possibility of selection bias in our difference-in-means estimator.

Finally, note that we can also use a different estimator, the OLS estimator (with simple linear regression), to get the same final estimate. We will discuss this in chapter 3.

2.3 Uncertainty and Standard Errors

Intuition of Uncertainty

Remember how we randomly assigned units to treatment or control? What if we ran the experiment again? The treatment and control groups would very likely not be exactly the same, and thus, we would get a slightly different causal effect.

Thus, we have some uncertainty with our causal estimate - re-running the experiment might result in a different answer. The ATE we have calculated is only our specific sample average treatment effect (SATE), often notated $\hat{\tau}_{ATE}$ or $A\hat{T}E$.

• Why sample? Well, through random assignment, you are basically "randomly sampling" potential outcomes - since randomly choosing one unit to be in treatment/control means not seeing the other counterfactual potential outcome.

Thus, we need some mechanism to account for sampling variability and how rerunning the experiment might result in slightly different results. We do this with sampling distributions and standard errors.

Sampling Distributions and Standard Error

Imagine that we take a sample from a population (or some random assignment mechanism). Then, we find the average treatment effect of the sample $\hat{\tau}_{ATE}$.

That is a **sample estimate**, which is often notated $\hat{\theta}$. (I use θ , since this idea of uncertainty can be applied to any estimate, not just average treatment effect).

Then, let us take another sample from the same population (or do another random assignment), and find the sample estimate. This will be slightly different than the first sample, since we are randomly sampling. That is another sample estimate.

We keep taking samples from the same population (more random assignments), and getting more and more sample estimates.

Let us plot all our sample estimates $\hat{\theta}$ (different $\hat{\tau}_{ATE}$ values) into a "histogram" or density plot. The x axis labels the possible $\hat{\tau}_{ATE}$ values, and the y axis is how frequently a specific sample estimate occurs.

The result is a distribution, just like a random variable distribution. That distribution is the **sampling** distribution.

According to **central limit theorem**, the sampling distribution approximates that of a **normal distribution** (or t-distribution if our sample size is small). We know that a normal distribution is defined by two parameters - mean and variance.

Standard deviation is the square root of variance. The standard deviation of our sampling distribution is what is called the **standard error** of our estimate.

? Key Definition: Standard Error

A **sampling distribution** is the imaginary distribution of estimates, if we repeated the sampling and estimation process many, many times.

The **standard error** is the standard deviation of the sampling distribution. It is often notated $SE(\hat{\theta})$. The computer/software we use will calculate an estimate for us.

2.4 Hypothesis Testing and Causal Inference

We know there is some uncertainty with our sample estimate, as defined by the standard error. So, how do we know if we actually have a causal effect with this uncertainty?

What we do is hypothesis testing: a way to test, given a certain level of uncertainty, whether or not we believe there is a causal effect.

We start off with the status-quo "old theory", and try to disprove it. This status quo theory, called the **null hypothesis** and notated H_0 , is typically that there is no causal effect of D on Y. The new theory we have come up with, and are trying to prove, is called the **alternate hypothesis**, often notated H_1 or H_a .

Mathematically, our hypothesis are:

$$H_0: \tau_{ATE} = 0$$
 and $H_1: \tau_{ATE} \neq 0$

We assume that the null hypothesis is true, unless we are 95% confident that we can reject the null hypothesis, and only then, can we accept the alternative hypothesis.

How do we actually test these hypotheses? First, we have to calculate a t-test statistic:

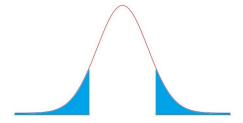
• Key Definition: T-test Statistic

The t-test statistic tells us how far our estimate is from the null θ_0 , in terms of standard errors:

$$t = \frac{\hat{\theta}_j - \theta_0}{\widehat{SE}(\hat{\theta}_j)}$$

Then, we find the corresponding t-distribution based on the degrees of freedom (n-2). Then, we start from the middle of that t distribution, and go the *number of standard errors* away based on the t-statistic. We do this on both sides from the middle of the t-distribution.

Once we have found that point, we find the probability (area under the distribution) of a t-test statistic of ours, or more extreme, could occur. The figure below shows this probability (called the p-value):



? Key Definition: P-value

Essentially, a p-value is how likely we are to get a test statistic at or more extreme than the one we got for our estimated θ_j , given the null hypothesis is true. If this is less than 0.05 (5%), then we reject the null hypothesis as no longer true.

- So if the p-value is above 0.05, there is a high chance that the null hypothesis is true. Thus, we cannot reject that there is no causal effect of D on Y.
- If the p-value is lower than 0.05, then there is a low chance that the null hypothesis is true. Thus, we can reject the null hypothesis, and conclude a causal effect of D on Y.

2.5 Balance Table and Stratified Experiments

Balance Tables

If the treatment and control groups are not similar (in regard to key confounding variable values), our difference-in-means estimator will be biased with selection bias.

To confirm this assumption is met, researchers will often show a **balance** table before or their estimation process. A balance table is essentially a table that shows the average difference in values of confounders in both treatment and control groups. For example, below is a balance table:

	Confounder 1	Confounder 2	Confounder 3	Confounder 4
Control Group Values	11.503***	1.474***	0.405***	81.983***
	(0.196)	(0.060)	(0.015)	(2.980)
Treatment - Control	-0.069	-0.062	0.009	-1.760
	(0.241)	(0.074)	(0.019)	(4.179)
Num. obs.	562	565	560	565

^{***}p < 0.001; **p < 0.01; *p < 0.05

The key row to look at is the *treatment-control* row. The numbers not in parentheses the difference between treatment and control for the corresponding confounder. The numbers in parentheses are the standard errors of the estimated difference. Stars (see legend below the table) show significance of a t-test (we will discuss this in the next section).

If no *treatment-control* for key confounders is significantly different (no stars), then randomisation has suceeded, and indeed, our control and treatment group are similar.

Blocking and Stratified Experiments

Blocking, also called stratified experiments, is an extension of randomised experiments to ensure that randomisation does not fail. Imagine that you have four units in your experiment that you have to assign to treatment/control. Their pre-treatment outcomes are $Y_{0i} = \{2, 2, 8, 8\}$. This means that you have a 1/3 chance to end up with the random assignment of $\{2, 2\}$ in one group and $\{8, 8\}$ in the other group, which would result in selection bias.

With blocking, you can prevent this from happening. The procedure is as following:

- 1. Before randomisation, you separate your sample of N units into J subgroups.
- 2. Within each group, randomly assign units to treatment and control group (essentially, smaller randomised experiments within a bigger experiment).

For example, we could divide our prior example into 2 subgroups: $\{2,2\}$ and $\{8,8\}$. Then, within each group, randomly assign one observation to treatment, and one to control. Thus, we are guaranteed to get units from both subgroups in both our treatment and control groups.

To estimate our effects for blocking experiments, we will have to take the weighted average of each subgroup's average treatment effect (ATE), with the weights being the proportion of units each group accounts for:

$$\tau_{ATE} = \sum_{j=1}^{J} \frac{N_j}{N} \tau_j$$

Where N is the total number of observations, J is the total number of subgroups, j is one of the subgroups, N_j is the number of units within subgroup j, and τ_j is the ATE of the subgroup j.

2.6 Validity and Issues with Experiments

Randomisation, when it works, is magical - it can help us obtain the best estimates of causal effects that are possible.

But, randomised controlled trials do have several issues that can affect our validity of our estimates and conclusions:

- 1. **Failure of randomisation**: if for some reason, randomisation does not result in control and treatment groups being similar, we cannot accurately estimate the average treatment effect. Blocking should help us deal with this, but it is still possible for randomisation to fail.
- 2. Non-Compliance: Sometimes, despite assigning certain units to treatment or control, the units do not comply and do the opposite (i.e. people assigned to control still take the treatment). This is a huge issue since researchers are not gods we cannot force people to take treatment. The biggest threat non-compliance creates is that perhaps, a confounder is causing certain units to be more likely to not comply. We will deal with this issue in the later parts of this book with Instrumental Variables.
- 3. Attrition: sometimes, it is not possible to measure the outcomes of some people in a study, either due to people moving away, passing away, or refusing to answer surveys or have their measurements be taken. This once again is an issue since perhaps a confounding variable is causing this issue.

However, the biggest issue with randomised controlled trials is the impracticality and infeasability of them in many situations, especially in social science research.

- For example, let us say you want to run an experiment on how democracy affects economic growth. It is nearly impossible to randomly assign countries to be a democracy. First, if you assign, for example, Russia to be a democracy, you have no power to make them actually follow through with your study. Second, there are ethical concerns about randomly allocating millions to democracy or autocracy.
- Another major issue is that even when experiments are theoretically feasible, they can be too expensive
 to implement. Randomised Controlled Trials are extremely expensive even when they are possible to
 run.

Thus, we will need to introduce ways to address for confounders and estimate causal effects for the vast majority of cases where we will not be able to run a randomised controlled trial.

The rest of the book focuses on these techniques. A brief overview of them:

- Simple Linear Regression will not be of much use for this, as it typically only is feasible in ideal random situations, however, it is the core that many further methods build on, and can help us explore continuous treatment variables.
- Multiple Linear Regression, in theory, can control for confounding variables. However, the limitation
 of this is that for an accurate estimation, all possible confounding variables (including unobservable or
 unmeasureable) confounders have to be included to prevent bias, which is very infeasible most of the
 time.
- Instrumental Variables Estimator, in theory, can deal with both non-compliance and confounders, however, there are also drawbacks to this method that we will cover later.
- Finally, we will discuss quasi-experimental methods, like regression discontinuity and differences-indifferences, which are the most popular methods of causal inference today.

Part II

Ordinary Least Squares Estimator

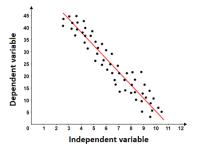
Simple Linear Regression and Causal Inference

3.1 Introduction to Regression

So far, we have looked at only binary treatment variables D, that can either be D=0 control group or D=1 treatment group. However, a lot of the relationships we are interested in are not binary. For example, you might be interested in how someone's income affects their likelihood to vote. Or how someone's age affects their political beliefs. Or how a country's GDP affects the chance a country becomes a democracy. Income, age, and GDP are all treatment variables that are not binary.

How can we explore the causal relationship between a continuous treatment variable D (often instead notated as X in statistics), and a continuous Y variable (we will focus on non-continuous Y later in chapter 5)?

One way is that we can explore this relationship with a linear best-fit line. A best-fit line is useful, since the slope of the line represents how much Y changes for every unit change of D.



The graph here introduces some common terminology:

- The independent variable, also called the explanatory variable or treatment variable, often notated x (or D in casual inference settings) is the variable we believe to be doing the causing.
- The **dependent variable**, also called the **response variable** or **outcome variable** is notated y. This is the variable that is being caused.

With regression, we are interested in how x causes y. Given a set of data with n number of observations, each observation i being a data point (x_i, y_i) , the goal of regression is to fit a best-fit line through this data. With this best-fit line, the slope will tell use the average affect of a one unit change in x on y.

3.2 Simple Linear Regression Model

A linear regression model is the specification of the conditional distribution of y, given x:

- Why a distribution? For example, if x was age and y was income, at age x = 30, not every single 30 year old makes the same amount of money. There is some distribution of incomes y at age x = 30.
- Why conditional? Well, if we believe x causes y, then we should expect the distribution of y values to change based on x values. If y values do not change when x changes, then there is no causal effect.

The linear regression model focuses on the **expected value** of the conditional distribution, notated $\mathbb{E}[y_i|x_i]$. Essentially, as x changes, the expected value of y changes.

• Key Definition: Simple Linear Regression

Take a set of observed data with n number of pairs of (x_i, y_i) observations. The linear model takes the following form:

$$\mathbb{E}[y_i|x_i] = \beta_0 + \beta_1 x_i$$

- Where $\mathbb{E}[y_i|x_i]$ is the expected value of y, given some value of x for observation i, and x_i is the observed value of x for observation i.
- Where the coefficients of the model are β_0 (the intercept) and β_1 (the slope). These are unknown quantities we need to estimate these by finding what intercept and slope make the best best-fit line.

We can also write the linear model for the value of any point Y_i in our data, which is more common:

$$y_i = \beta_0 + \beta_1 x_i + u_i$$

- Where y_i is the observed value of y for observation i, and x_i is the observed value of x for observation i.
- Where coefficients β_0 and β_1 need to be estimated.
- Where u_i is the error term that determines the error for each unit i. This error term essentially reflects the "distribution" that we discussed earlier, where $\mathbb{E}[u_i] = 0$.

Once we have the estimates of β_0 and β_1 (estimation process explained in next section), we will get the fitted values model:

$$\hat{y}_i = \hat{\beta}_0 + \hat{\beta}_1 x_i$$

- The $\hat{}$ on top of the y_i , β_0 , and β_1 indicate estimates. This is because we estimate the coefficients β_0 and β_1 , which are then used to estimate y_i .
- Notice that the error term u_i is no longer here. This is because we assume $\mathbb{E}[u_i] = 0$.

With these fitted values, we can do the following:

- 1. Prediction: since we have estimated the coefficients, we can plug in x_i to see what the predicted \hat{y}_i would be. This book will not focus on prediction (see Volume III of this series).
- 2. Causal Inference: If we assume that treatment x_i is randomly assigned, then β_1 can be used for causal estimation. We will illustrate this in the next few sections.

Simple Linear Regression is also a very important building block for all advanced causal inference and predictive techniques, many that we will introduce later in this book and series.

3.3 Ordinary Least Squares Estimator

We want to fit a best-fit line that is accurate, so we want to minimise errors. An error is the difference between the actual y_i and our fitted model's prediction \hat{y}_i .

• Key Definition: Sum of Squared Errors

The sum of squared errors (SSE) is as follows:

$$SSE = \sum (y_i - \hat{y}_i)^2 = \sum (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i)$$

Or more intuitively, it is exactly as it sounds - find the error of our prediction $y_i - \hat{y}_i$ for every observation i, square that difference, then sum up for all units i in our data.

The Ordinary Least Squares (OLS) Estimator estimates our parameters by finding the values of β_0, β_1 that minimizes the sum of squared errors for our predictions.

• Why squared? this is because squaring the error removes the direction (positive or negative error), and keeps the magnitude. OLS also has some unique properties (see chapter 4).

Let us define the SSE as function S. The OLS estimator wants to find the parameters that **minimise SSE**:

$$(\hat{\beta}_0,\hat{\beta}_1) = \arg\min_{\hat{\beta}_0,\hat{\beta}_1} \sum_{i=1}^n (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i)^2 = \arg\min_{\hat{\alpha},\hat{\beta}} S(\hat{\beta}_0,\hat{\beta}_1)$$

First Order Conditions

Let us first look at the parameter $\hat{\beta}_0$. How do we find what value $\hat{\beta}_0$ minimises the sum of squared errors? We know through calculus, that deriving the function to find the first order condition can accomplish this:

$$\frac{\partial S(\hat{\beta}_0,\hat{\beta}_1)}{\partial \hat{\beta}_0} = \frac{\partial S(\hat{\beta}_0,\hat{\beta}_1)}{\partial \hat{\beta}_0} \left[\sum_{i=1}^n (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i)^2 \right]$$

First, ignore the summation. The partial derivative of the internal section, using chain rule, is the following:

$$\frac{\partial}{\partial \hat{\beta}_0} \left[(y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i)^2 \right] = -2(y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i)$$

But how do we deal with the summation? We know that there is the sum rule of derivatives [f(x) + g(x)]' = f'(x) + g'(x). Thus, we know we just sum up the derivatives to get the derivative:

$$\frac{\partial S(\hat{\beta}_0,\hat{\beta}_1)}{\partial \hat{\beta}_0} = \sum_{-i=1}^n \left[-2(y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i) \right] = -2 \sum_{i=1}^n (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i)$$

Let us do the same for $\hat{\beta}_1$: find the derivative:

$$\frac{\partial S(\hat{\beta}_0,\hat{\beta}_1)}{\partial \hat{\beta}_1} = \sum_{i=1}^n \left[-2x_i(y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i) \right] = -2\sum_{i=1}^n x_i(y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i)$$

We set the two derivatives equal to 0 (first order conditions) We can ignore the -2 out front.

Yey Definition: First Order Conditions of OLS

Thus, our first order conditions are a system of equations:

$$\sum_{i=1}^n (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i) = 0 \quad \text{and} \quad \sum_{i=1}^n x_i (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i) = 0$$

Now we have to solve this system of 2 equations. Let us first solve for $\hat{\alpha}$ in terms of $\hat{\beta}$:

$$\begin{split} \sum_{i=1}^{n} (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_i) &= \sum_{i=1}^{n} y_i - n \hat{\beta}_0 - \hat{\beta}_1 \sum_{i=1}^{n} x_i = 0 \\ &- n \hat{\beta}_0 = - \sum_{i=1}^{n} y_i + \hat{\beta}_1 \sum_{i=1}^{n} x_i \\ &\hat{\beta}_0 = \frac{1}{n} \sum_{i=1}^{n} y_i - \frac{1}{n} \hat{\beta}_1 \sum_{i=1}^{n} x_i = \bar{y} - \hat{\beta}_1 \bar{x} \end{split}$$

Now, let us substitute our calculated $\hat{\beta}_0 = \bar{y} - \hat{\beta}_1 \bar{x}$ into the $\hat{\beta}_1$ condition and solve: for $\hat{\beta}_1$:

$$\begin{split} 0 &= \sum_{i=1}^n \left[x_i (y_i - [\bar{y} - \hat{\beta}_1 \bar{x}] - \hat{\beta}_1 x_i) \right] = \sum_{i=1}^n \left[x_i (y_i - \bar{y} - \hat{\beta}_1 (x_i - \bar{x})) \right] \\ &= \sum_{i=1}^n \left[x_i (y_i - \bar{y}) - x_i \hat{\beta}_1 (x_i - \bar{x}) \right] = \sum_{i=1}^n x_i (y_i - \bar{y}) - \hat{\beta}_1 \sum_{i=1}^n x_i (x_i - \bar{x}) \end{split}$$

Here are a few properties on summation that will help us solve this equation:

$$\begin{split} &\sum_{i=1}^{n}(x_{i}-\bar{x})=0\\ &\sum_{i=1}^{n}x_{i}(y_{i}-\bar{y})=\sum_{i=1}^{n}(x_{i}-\bar{x})(y_{i}-\bar{y})\\ &\sum_{i=1}^{n}x_{i}(x_{i}-\bar{x})=\sum_{i=1}^{n}(x_{i}-\bar{x})^{2} \end{split}$$

With these rules, we can transform what we had before into:

$$0 = \sum_{i=1}^{n} (x_i - \bar{x})(y_i - \bar{y}) - \hat{\beta}_1 \sum_{i=1}^{n} (x_i - \bar{x})^2$$

• Key Definition: OLS Estimate

Then solve for $\hat{\beta}_1$ to get the **OLS estimator** for bivariate regression:

$$\hat{\beta}_1 = \frac{\sum\limits_{i=1}^n (x_i - \bar{x})(y_i - \bar{y})}{\sum\limits_{i=1}^n (x_i - \bar{x})^2} = \frac{Cov(x,y)}{Var(x)} = \frac{\sigma_{xy}}{\sigma_x^2}$$

If treatment was randomly assigned in our data, $\hat{\beta}_1$ is an estimate of the average causal effect (we will discuss if this is a good estimate in section 3.6 and chapter 4).

3.4 Interpretations of Coefficients

Coefficient $\hat{\beta}_1$

We know that in a linear model $\mathbb{E}[y_i|x_i] = \beta_0 + \beta_1 x_i$, the coefficient β_1 is the slope, or the rate of change of y given a one unit increase in x. Thus:

i Interpretation of $\hat{\beta}_1$

When x increases by one unit, there is an expected $\hat{\beta}_1$ unit change in y.

If random assignment is present, $\hat{\beta}_1$ is also the estimate of the average causal effect of x on y. However, we still need to conduct hypothesis tests for causal inference.

• If random assignment did not occur, we cannot interpret single linear regression coefficients causally. We will need further methods.

Coefficient $\hat{\beta}_0$

What does intercept $\hat{\beta}_0$ mean? Let us take a regression equation, and input x = 0.

$$\begin{split} \mathbb{E}[y_i|x_i] &= \beta_0 + \beta_1 x_i \\ \mathbb{E}[y_i|x_i = 0] &= \beta_0 + \beta_1(0) = \beta_0 \end{split}$$

i Interpretation of $\hat{\beta}_0$

When x = 0, the expected value of y is $\hat{\beta}_0$

Interpreting in Terms of Standard Deviation

Sometimes, it is hard to understand what changes in y and x mean in terms of units. For example, if we are measuring "democracy", what does a 5 unit change in democracy mean? Is that a lot?

We can add more relevant detail by expressing the change of y and x in standard deviations.

How do we calculate this? Well, let us solve for the change in \hat{y} given x = x and $x = x + \sigma_X$. This will tell us how much \hat{y} changes by given a increase of one standard deviation in x.

$$\begin{split} \mathbb{E}[\hat{y}_i|x=x+\sigma_x] - \mathbb{E}[\hat{y}_i|x=x] &= [\hat{\beta}_0 + \hat{\beta}_1(x+\sigma_x)] - [\hat{\beta}_0 + \hat{\beta}_1(x)] \\ &= \hat{\beta}_0 + \hat{\beta}_1x + \hat{\beta}_1\sigma_x - \hat{\beta}_0 - \hat{\beta}_1x \\ &= \hat{\beta}_1\sigma_x \end{split}$$

To get the change in \hat{y} in terms of standard deviations of y, we just divide $\hat{\beta}_1 \sigma_x$ by σ_y .

i Interpretation in Terms of Standard Deviation

For a one-std. deviation increase in x, there is an expected $\hat{\beta}_1 \sigma_x / \sigma_y$ -std. deviation change in Y.

3.5 Categorical Explanatory Variable Interpretation

Binary Explanatory Variables

Binary explanatory variables are variables with 2 values, 0 and 1.

• These are extremely common in econometrics - as our treatment variable D often takes two states: D = 1 is the treatment group, and D = 0 is the control group. We know that in a regression, D is included as an explanatory variable x.

Binary explanatory variables will change the interpretations of our coefficients. We can "solve" for these interpretations given x has two categories x = 0, x = 1:

$$\begin{split} \mathbb{E}[y|x=0] &= \beta_0 + \beta_1(0) = \beta_0 \\ \mathbb{E}[y|x=1] &= \beta_0 + \beta_1(1) = \beta_0 + \beta_1 \\ \mathbb{E}[y|x=1] - \mathbb{E}[y|x=0] &= (\beta_0 + \beta_1) - \beta_0 = \beta_1 \end{split}$$

Thus, we can interpret the coefficients as follows:

Interpretation of Binary Explanatory Variables

When x is a binary explanatory variable:

- β_0 is the expected value of y given an observation in category x=0
- $\beta_0 + \beta_1$ is the expected value of y given an observation in category x = 1
- β_1 is the expected difference in y between the categories x=1 and x=0.

We can see that β_1 is measuring the difference between the two categories.

- In fact, β_1 actually becomes a **difference-in-means** test, meaning that if β is statistically significant, we can conclude a significant difference in the mean y between the two categories.
- If our treatment is randomly assigned (such as in a randomised controlled trial), β_1 is the estimate of the average treatment effect. Thus, we can use regression to estimate randomised controlled trials.

Once again, if random assignment did not occur, we cannot interpret single linear regression coefficients causally. We will need further methods.

Categorical Explanatory Variable

A categorical polytomous variable is one with 3 or more categories. A classic example is the variable country, which has categories USA, Germany, UK, Australia, etc.

How do we run a regression with polytomous explanatory variables? What happens is that we divide the variables into a set of binary variables (also called dummy variables).

- Dummy binary variables are created for all <u>except one</u> of the categories in our variable. Each dummy variable has two values 1 meaning the observation is in the category, and 0 meaning the observation is not in that category.
- The category without a dummy variable is the **reference/baseline** category. Essentially, when all other dummy variables are equal to 0, that is referring to the reference/baseline category (the intercept)

• Key Definition

Thus, a **polytomous explanatory variable** with n number of categories in x, we would create n-1 dummy variables, and input it into a regression equation as follows:

$$\mathbb{E}[y] = \beta_0 + \beta_{x=1} x_{x=1} + \ldots + \beta_{x=n-1} x_{x=n-1}$$

Where β_0 is the mean of the reference category n, and the other categories 1, ..., n-1 get their own dummy variable.

For example, take the following polytomous variable: *company*, which contains the categories *microsoft*, *google*, and *apple*. Let us create dummy variables for 2 of the 3 categories:

- Google will become the first dummy variable x_g . When $x_g = 1$, that observation is part of the google category. When $x_g = 0$, that observation is NOT a part of the google category.
- Apple will become the second dummy variable x_a . When $x_a = 1$, that observation is part of the apple category. When $x_a = 0$, that observation is NOT a part of the apple category.
- Microsoft will not get its own dummy variable. This is because when both apple and microsoft $x_g = x_a = 0$ that is referring to the microsoft category (observations not a part of either previous category).

Mathematically, this is how it would be represented in a regression equation:

$$\mathbb{E}[y] = \beta_0 + \beta_a x_a + \beta_a x_a$$

To find the expected value of each category, we would do the following:

$$\begin{split} &\mathbb{E}[y|x = \text{Google}] = \mathbb{E}[y|x_g = 1, x_a = 0] = \beta_0 + \beta_g(1) + \beta_a(0) = \beta_0 + \beta_g\\ &\mathbb{E}[y|x = \text{Apple}] = \mathbb{E}[y|x_g = 0, x_a = 1] = \beta_0 + \beta_g(0) + \beta_a(1) = \beta_0 + \beta_a\\ &\mathbb{E}[y|x = \text{Microsoft}] = \mathbb{E}[y|x_g = 0, x_a = 0] = \beta_0 + \beta_g(0) + \beta_a(0) = \beta_0 \end{split}$$

Thus, from these above equations, we can see the interpretation of the coefficients:

- β_0 is the expected value of the reference category, in this case, *microsoft*.
- β_g is the expected y difference between the google category and the reference category microsoft. The statistical significance of this coefficient would be a difference of means test between the two categories.
- β_a is the expected y difference between the apple category and the reference category microsoft. The statistical significance of this coefficient would be a difference of means test between the two categories.

Interpretation of Polytomous Explanatory Variables

 β_j is the expected difference in y values between category j and the baseline category. β_0 is the expected value of y of the baseline category.

Additional note: The coefficient p-values of β_j (discussed in the next section) are a difference-of-means test between two categories, and not a statistical significance test of the entire categorical variable.

3.6 Hypothesis Testing and Causal Inference

Hypothesis Testing of Parameters

We previously discussed hypothesis testing in section 2.4. The mechanics are practically the same, but we replace the sample average treatment effect with $\hat{\beta}_1$ as our sample estimate. Generally, for regressions, our hypotheses that we test are:

- $H_0:\beta_1=0$ i.e. there is no relationship between x and y
- $H_1: \beta_1 \neq 0$ i.e. there is a relationship between x and y

Just like previously discussed in section 2.5, we calculate a t-statistic (minus 0 since that is null hypothesis):

$$t = \frac{\hat{\beta}_1 - 0}{\hat{se}(\hat{\beta}_i)}$$

The t-test statistic tells us how far the estimate is from 0, in terms of standard errors of the estimate.

Then, we have to consult a t-distribution (see section 2.4). We find the probability (area under the distribution) of a t-test statistic of ours, or more extreme, could occur. This is the p-value: how likely we are to get a test statistic at or more extreme than the one we got for our estimated β_1 , given the null hypothesis is true.

- So if the p-value is very high, there is a high chance that the null hypothesis is true.
- If the p-value is very low, then there is a low chance that the null hypothesis is true

Generally, in the social sciences, if the p-value is less that 0.05 (5%), we can **reject the null hypothesis**, and conclude the alternate hypothesis.

Causl Inference in Simple Linear Regression

Remember, correlation does not mean causation. Simple linear regression by itself only measures correlation between variables, not causation.

This is why causal inference, and claiming β_1 is the average causal effect of x on y (even if it is statistically significant and we reject the null) is only possible when random assignment of treatment has occurred (as I have mentioned multiple times).

• Thus, we can use regression to estimate the average causal effect of a randomised controlled trial.

The reason we cannot make causal claims when randomisation (even if our hypothesis test is significant) is because of two factors:

- 1. Without randomisation, we might have confounders and selection bias. Clearly, our one variable linear regression does not account for any confounders. In the next chapter, we will introduce Multiple Linear Regression, which will allow us to control for confounders.
- 2. Without randomisation, we might have reverse causality. It could be that y is causing x. Randomisation prevents this because the random selection is causing x, so y has no independent effect on x. We will introduce further techniques like instrumental variables and quasi-experimental techniques to deal with this.

This chapter has also left out any discussion of the OLS estimator itself - how good is the actual estimator (answer: it is very good under certain conditions). We will cover this in the next chapter as well.

Multiple Linear Regression and Causal Inference

4.1 The Multiple Linear Regression Model

In the social sciences, randomisation is often not possible. Linear regression allows us to estimate a model with both our treatment and outcome variables, as well as a series of **control** variables.

• We will explore the idea of "controlling" in later in the chapter.

In theory, by including every single confounding variable as a control variable in our regression model, we can partial out the effect of confounders and find the average treatment effect. In reality, as we will cover later, it is often impossible to account for all confounding variables, so we may need additional techniques to account for those situations.

The **response variable** (outcome variable) is notated y, just like in single linear regression. The **explanatory variables** are $x_1, x_2, ..., x_k$. We sometimes also denote all explanatory variables as the vector \vec{x} . Our treatment variable D is considered one of the explanatory variables \vec{x} (most often x_1).

A linear regression model is the specification of the conditional distribution of Y, given \vec{x} . The linear regression model focuses on the **expected value** of the conditional distribution, notated $\mathbb{E}[y_i|\vec{x}_i]$.

Key Definition: Linear Regression Model

Take a set of observed data with n number of pairs of (\vec{x}_i, y_i) observations. The linear model takes the following form:

$$\mathbb{E}[y_i|\overrightarrow{x}_i] = \beta_0 + \beta_1 x_{1i} + \ldots + \beta_k x_{ki}$$

• Where the coefficients (that need to be estimated) are $\operatorname{vector} \vec{\beta} = \beta_0, \beta_1, ..., \beta_k$.

We can also write the linear model for the value of any point Y_i in our data:

$$y_i = \beta_0 + \beta_1 x_{1i} + \dots + \beta_k x_{ki} + u_i$$

• Where u_i is the error term function - that determines the error for each unit *i*. Error u_i has a variance of σ^2 , and expectation $\mathbb{E}[u_i] = 0$.

4.2 Ordinary Least Squares Estimator

Similar to our bivariate regression (but with additional variables), our minimisation condition is:

$$(\hat{\beta}_0, \hat{\beta}_1, \hat{\beta}_2, \ldots) = \arg\min_{(\hat{\beta}_0, \hat{\beta}_1, \hat{\beta}_2, \ldots)} (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_{1i} - \hat{\beta}_2 x_{2i} \ldots)^2 = \arg\min_{(\hat{\beta}_0, \hat{\beta}_1, \hat{\beta}_2, \ldots)} S(\hat{\beta}_0, \hat{\beta}_1, \hat{\beta}_2, \ldots)$$

Taking the partial derivatives of each parameter like in simple linear regression, we get first order conditions:

$$\begin{split} &\sum_{i=1}^n (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_{1i} - \hat{\beta}_2 x_{2i}...) = 0 \\ &\sum_{i=1}^n X_{1i} (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_{1i} - \hat{\beta}_2 x_{2i}...) = 0 \\ &\sum_{i=1}^n X_{2i} (y_i - \hat{\beta}_0 - \hat{\beta}_1 x_{1i} - \hat{\beta}_2 x_{2i}...) = 0 \\ &\text{and so on for } X_{3i}, ..., X_{ki} \end{split}$$

This system of equations includes k + 1 variables and k + 1 equations, which is way too difficult to solve. Instead, we can use linear algebra. We start with the linear model:

$$y_i = \beta_0 + \beta_1 x_{1i} + \beta_2 x_{2i} + \dots + \beta_k x_{ki} + u_i$$

The ith observation can be written in vector form as following:

$$y_i = x_i'\beta + u_i, \text{ where } \beta = \begin{bmatrix} \beta_0 \\ \beta_1 \\ \vdots \\ \beta_k \end{bmatrix} \text{ and } x_i = \begin{bmatrix} 1 \\ x_{1i} \\ \vdots \\ x_{ki} \end{bmatrix}$$

- The x_i' in the equation is the transpose of x_i , to make matrix multiplication possible.
- The first element of the x_i matrix is 1, since $1 \times \beta_0$ gives us the first parameter (intercept) in the linear model.

Since our model has n different observations of i, we can express this into vector form, with the X'_i and β being vectors within a vector.

$$\begin{pmatrix} y_1 \\ y_2 \\ \vdots \\ y_n \end{pmatrix} = \begin{pmatrix} x_1'\beta + u_1 \\ x_2'\beta + u_2 \\ \vdots \\ x_n'\beta + u_n \end{pmatrix} = \begin{pmatrix} x_1'\beta \\ x_2'\beta \\ \vdots \\ x_n'\beta \end{pmatrix} + \begin{pmatrix} u_1 \\ u_2 \\ \vdots \\ u_n \end{pmatrix}$$

Since β vector appears as a common factor for all observations i = 1, ..., n, we can factor it out and have an equation:

$$\begin{pmatrix} y_1 \\ y_2 \\ \vdots \\ y_n \end{pmatrix} = \begin{pmatrix} x_1' \\ x_2' \\ \vdots \\ x_n' \end{pmatrix} \beta + \begin{pmatrix} u_1 \\ u_2 \\ \vdots \\ u_n \end{pmatrix}$$

We can expand the $x'_1, ..., x'_n$ vector into a matrix. Remember that each $x'_1, ..., x'_n$ is already a vector of different explanatory variables. Thus, we have a model in the form:

$$y = X\beta + u, \text{ where } X = \begin{bmatrix} 1 & x_{21} & \dots & x_{k1} \\ 1 & x_{22} & \dots & x_{k2} \\ \vdots & \vdots & \vdots & \vdots \\ 1 & x_{2n} & \dots & x_{kn} \end{bmatrix}$$

- Where the notation for elements of X is x_{ki} , with i being the unit of observation i = 1, ... n, and k being the explanatory variables index.
- Where y and u are $n \times 1$ vectors (as seen above), and β is a $k \times 1$ vector.
- The first row of X is a vector of 1, which exists because these 1's are multiplied with β_0 in our model.

Now, let us estimate our coefficients. Let us define our estimation vector $\hat{\beta}$ as:

$$\hat{\beta} = \arg\min_{b} (y - Xb)'(y - Xb) = \arg\min_{b} S(b)$$

We can expand S(b) as follows:

$$S(b) = y'y - b'X'y - y'Xb + b'X'Xb = y'y - 2b'X'y + b'X'Xb$$

Taking the partial derivative in respect to b, then setting equal to 0, we get:

$$\left. \frac{\partial S(b)}{\partial b} \right|_{\hat{\beta}} = \left(\frac{\frac{\partial S(b)}{\partial b_1}}{\vdots \frac{\partial S(b)}{\partial b_k}} \right) \right|_{\hat{\beta}} = 0$$

Differentiating with the vector b yields:

$$\frac{\partial S(b)}{\partial b} = -2X'y + 2X'Xb$$

Evaluted at $\hat{\beta}$, the derivatives should equal zero (since first order condition of finding minimums):

$$\left. \frac{\partial S(b)}{\partial b} \right|_{\hat{\beta}} = -2X'y + DX'X\hat{\beta} = 0$$

• Key Definition

When assuming X'X is invertable, our OLS estimator solution for $\hat{\beta}$ is:

$$\hat{\beta} = (X'X)^{-1}X'y$$

Once we have estimates of $\hat{\beta}$, we can plug them into our linear model to obtain fitted values:

$$\hat{y} = X\hat{\beta} = X(X'X)^{-1}X'y$$

4.3 Regression Anatomy and Coefficient Interpretation

We talked about how multiple linear regression allows us to control for confounders. But what does that mean? How does it affect our interpretations of coefficients? Take our standard multivariate regression:

$$y_i = \beta_0 + \beta_1 x_{1i} + \beta_2 x_{2i} + \dots + \beta_k x_{ki} + u_i$$

Let us say we are interested in x_1 (this can be generalised to any explanatory variable). Let us make x_1 the outcome variable of a regression with $x_2, ..., x_k$:

$$x_{1i}=\gamma_0+\gamma_1x_{2i}+\ldots+\gamma_{k-1}x_{ki}+\widetilde{x_{1i}}$$

 $\gamma_0, ..., \gamma_{k-1}$ are coefficients. The error term is $\widetilde{x_{1i}}$, which represents the part of x_{1i} that are uncorrelated to $x_2, ..., x_k$. In other words, $\widetilde{x_{1i}}$ is the part of x_1 that cannot be explained by any other explanatory variable.

Now, take the regression of with outcome variable y, with all explanatory variables except x_1 :

$$y_i = \delta_0 + \delta_1 x_{2i} + \ldots + \delta_{k-1} x_{ki} + \widetilde{y_i}$$

 $\delta_0,...,\delta_{k-1}$ are coefficients. The error term is $\widetilde{y_i}$, which is the part of y_i that cannot be explained by $x_2,...,x_k$. Since $\widetilde{y_i}$ is not explained by $x_2,...,x_k$, variable x_1 must be the one explaining $\widetilde{y_i}$. But, it is not the whole of x_1 explaining $\widetilde{y_i}$ - since x_1 may also correlated with $x_2,...,x_k$, and the correlated parts of x_1 with $x_2,...,x_k$ are already picked up in the regression by the coefficients of $x_2,...,x_k$.

Thus, $\widetilde{y_i}$ must be explained by the part of x_1 that is uncorrelated and not explained by $x_2, ..., x_k$, which we derived earlier as $\widetilde{x_{1i}}$. Thus, we can create another regression with explanatory variable $\widetilde{x_{1i}}$ and outcome variable $\widetilde{y_i}$.

$$\widetilde{y_i} = \alpha_0 + \alpha_1 \widetilde{x_{1i}} + u_i$$

We can plug $\widetilde{y_i}$ back into our regression of y_i with explanatory variables $x_2...,x_k$:

$$y_i = \delta_0 + \alpha_0 + \alpha_1 \widetilde{x_{1i}} + \delta_1 x_{2i} + \dots + \delta_{k-1} x_{ki} + u_i$$

As we can see, this mirrors the original standard multiple linear regression. The estimate of α_1 will be the same as β_1 in the original regression.

The coefficient α_1 (which is equal to β_1) explains the expected change in y, given an increase in the part of x_1 uncorrelated with $x_2, ..., x_k$. So essentially, we have **partialed out** the effect of the other explanatory variables, and only focus on the effect on y of the uncorrelated part of x_1 . This eliminates the effect of confounders on our estimates, and estimates of the effect of x_1 alone on y.

Interpretation of Coefficients in Multiple Linear Regression

Thus, the interpretation of β_1 , or any β_j multiplied to x_j , is: for every one unit increase in x_j , there is an expected β_j increase in y, when controlling for all other explanatory variables.

• Intecept β_0 is the expected value of y when all $\vec{x} = 0$.

Using the same procedure as in simple linear regression, we can run hypothesis tests on each coefficient for inference purposes (we will discuss causal inference with OLS in the next sections).

Unbiasedness and Consistency of OLS 4.4

An unbiased estimator, if we recall from section 1.3, means that over many different estimates, the expected value of all the estimates is the true parameter value: $\mathbb{E}[\hat{\theta}_i] = \theta$. Unbiasedness is desirable property of causal estimators.

Yey Definition: Gauss-Markov Theorem for Unbiasedness

The Gauss-Markov Theorem states that the ordinary least squares estimator is unbiased under 3 conditions:

- 1. Linearity of the equation (no coefficients $\beta_0, ..., \beta_k$ can be multiplied together. This does not apply to explanatory variables, only the coefficients.
- 2. In a simple linear regression, x has variation (so not all values of x are the same). In a multiple linear regression, this condition becomes that there is no perfect correlation (no perfect multicollinearity) between any two explanatory variables.
- 3. And the most important condition Zero conditional mean: meaning that no matter the value of x, the expected value of the residual u_i is always 0. $\mathbb{E}[u|x] = 0$ for all x (and all explanatory variables in multiple regression).

Proof of Unbiasdness

We want to show $\mathbb{E}[\hat{\beta}_1] = \beta_1$. Before we start, here are a few key properties of summation:

$$\begin{split} \sum_{i=1}^{n}(x_{i}-\bar{x}) &= 0\\ \sum_{i=1}^{n}x_{i}(y_{i}-\bar{y}) &= \sum_{i=1}^{n}(x_{i}-\bar{x})(y_{i}-\bar{y})\\ \sum_{i=1}^{n}x_{i}(x_{i}-\bar{x}) &= \sum_{i=1}^{n}(x_{i}-\bar{x})^{2} \end{split}$$

Let us start off with the OLS estimator (we will use simple linear regression for simplicity). We can expand the numerator, and since $\sum (x_i - \bar{x})\bar{y} = \bar{y}\sum (x_i - \bar{x}) = 0$, we can get:

$$\hat{\beta}_1 = \frac{\sum\limits_{i=1}^n (x_i - \bar{x})(y_i - \bar{y})}{\sum\limits_{i=1}^n (x_i - \bar{x})^2} = \frac{\sum\limits_{i=1}^n (x_i - \bar{x})y_i}{\sum\limits_{i=1}^n (x_i - \bar{x})^2}$$

The existence of $\hat{\beta}_1$ is guaranteed by condition 2 from above, since the denominator is equal to Var(x), so that must not be 0.

Now, let us play with the numerator (note the properties of summation introduced earlier):

$$\begin{split} \sum_{i=1}^n (x_i - \bar{x}) y_i &= \sum_{i=1}^n (x_i - \bar{x}) (\beta_0 + \beta_1 x_i + u_i) \\ &= \beta_0 \sum_{i=1}^n (x_i - \bar{x}) + \beta_1 \sum_{i=1}^n (x_i - \bar{x}) x_i + \sum_{i=1}^n (x_i - \bar{x}) u_i \\ &= 0 + \beta_1 \sum_{i=1}^n (x_i - \bar{x})^2 + \sum_{i=1}^n (x_i - \bar{x}) u_i \end{split}$$

Now, putting the numerator back into the equation, we simplify:

$$\hat{\beta}_1 = \frac{\beta_1 \sum\limits_{i=1}^n (x_i - \bar{x})^2 + \sum\limits_{i=1}^n (x_i - \bar{x}) u_i}{\sum\limits_{i=1}^n (x_i - \bar{x})^2} \ = \ \beta_1 + \frac{\sum\limits_{i=1}^n (x_i - \bar{x}) u_i}{\sum\limits_{i=1}^n (x_i - \bar{x})^2} \ = \ \beta_1 + \sum\limits_{i=1}^n d_i u_i$$

Where $d_i = \frac{x_i - \bar{x}}{\sum (x_i - \bar{x})^2}$, which is a function of random variable x.

Now we need to find the expectation $\mathbb{E}[\hat{\beta}_1]$. First, do not worry about d_i . What should u_i be equal to? Naturally, the best estimate of u_i is its expected utility.

$$\mathbb{E}(\hat{\beta}_1|x) = \beta_1 + \sum_{i=1}^n d_i \ \mathbb{E}(u_i|x)$$

We know by the third Gauss-Markov condition (Zero conditional mean), that $\mathbb{E}[u_i|x_i] = 0$. Thus, that makes the entire summation equal to 0:.

$$\mathbb{E}(\hat{\beta}_1|x) = \mathbb{E}(\hat{\beta}_1) = \beta_1$$

Thus, the ordinary least squares estimator is unbiased, given 3 conditions (linearity, variation in x, zero-conditional mean) are met. If conditions are met, we can conduct accurate causal estimation with no bias.

The key issue is assumption 3: Zero conditional mean $\mathbb{E}[u|x] = 0$, which is the most easily violated of these assumptions. We will explore this in detail in the next section.

Consistency and Efficiency of OLS

There is an additional assumption, that when added to the previous assumptions, makes OLS the best linear unbiased estimator in terms of **consistency** (lowest variation in estimates):

• Key Definition: Full Gauss-Markov Theorem

The Gauss-Markov Theorem states that under 4 conditions, OLS is not only unbiased, but also the most consistent linear estimator. Thus, OLS is deemed BLUE (best linear unbiased estimator):

- 1. Linearity of Parameters.
- 2. Variation in x (for simple regression) and non-perfect multicollinearity (for multiple regression).
- 3. Zero Conditional Mean
- 4. Homoscedasticity: variance of errors constant throughout the model: $Var(u|x) = \sigma^2$.

In other words, the additional assumption **homoscedasticity** is that the variance of the error term u_i is constant, no matter the value of x.

To evaluate this assumption, look at the plot of residuals/errors. If errors show a pattern as x changes (such as becoming larger or smaller), then homoscedasticity is violated, and we have **heteroscedasticity**.

When we do not meet the assumption of homoscedasticity, we have to use heteroscedasticity-**robust** standard errors (which account for our estimates less consistent, making the standard errors larger, thus harder to reject the null hypothesis).

• These days in econometrics, we assume that homoscedasticity is violated, and almost always use robust standard errors by default (which we have been doing so far).

4.5 Omitted Variable Bias and Endogeneity

In the last section, we discussed how the assumption of **zero-conditional mean** $\mathbb{E}[u|x] = 0$ is critical to the unbiasdness of OLS. This assumption is also called **exogeneity**. However, this assumption is frequently violated. The most common reason for this is because of **omitted variable bias**.

Consider two regressions, one with only our treatment variable of interest D, and, and one with an extra control variable x that is omitted from the first regression.:

$$y_i = \beta_0^S + \beta_1^S D_i + u_i^S$$
 "short" regression
 $y_i = \beta_0 + \beta_1 D_i + \beta_2 x_i + u_i$ "long" regression

Now consider an auxiliary regression, where the omitted variable X is the outcome variable, and D_i is the explanatory variable:

$$x_i = \delta_0 + \delta_1 D_i + v_i$$
 where δ_0, δ_1 are coefficients and v_i is the error term

Now we have x_i in terms of D_i , let us plug x_i into our long regression to "recreate" the short regression:

$$\begin{split} y_i &= \beta_0 + \beta_1 D_i + \beta_2 x_i + u_i \\ y_i &= \beta_0 + \beta_1 D_i + \beta_2 (\delta_0 + \delta_1 D_i + v_i) + u_i \\ y_i &= \beta_0 + \beta_1 D_i + \beta_2 \delta_0 + \beta_2 \delta_1 D_i + \beta_2 v_i + u_i \\ y_i &= \beta_0 + \beta_2 \delta_0 + (\beta_1 + \beta_2 \delta_1) D_i + \beta_2 v_i + u_i \end{split}$$

We have "recreated" the short regression with one variable D. That means coefficient $\beta_1^S = \beta_1 + \beta_2 \delta_1$.

The difference between the short regression coefficient β_1^S , and the original long regression coefficient β_1 , is $\beta_2\delta_1$. Or in other words, $\beta_2\delta_1$, which is actually the effect of omitted x on y, is being "tangled up" into the coefficient of the short regression β_1^S .

• If $\beta_2 = 0$ (meaning no relationship between omitted x_i and y), or $\delta_1 = 0$ (meaning no relationship between omitted x_i and D_i), then difference $\beta_2 \delta_1 = 0$, thus there is no issue. But if either of those facts are not true, then $\beta_2 \delta_1 \neq 0$.

Why is this an issue? The omitted variable x's effect is mostly subsumed into the error term u_i^S of the short regression. But some bit of x (that is correlated with D) is included in our coefficient (specifically, $\beta_2 \delta_1$). That means our treatment variable D will be correlated with the error term, violating the Gauss-Markov assumption of zero-conditional mean, and producing biased OLS results.

Key Definition: Endogeneity

Endogeneity is when a regressor x is correlated with the error term u_i . This frequently occurs due to **omitted variable bias**. The explanatory variable that is correlated with the error term is called an **endogenous variable**.

When endogeneity exists, the assumption of zero-conditional mean/exogeneity $\mathbb{E}[u|x] = 0$ is violated. This means that OLS estimates are no longer unbiased. That means, when endoeneity is present, accurate causal estimation with OLS is not possible.

4.6 Model Selection for Causal Inference

In the last section, we established that omitted variable bias will often cause endogeneity, thus causing OLS to be biased. How do we prevent this from happening?

Recall, that omitted variable bias $\beta_2 \delta_1$ is only an issue when $\beta_2 \delta_1 \neq 0$ (since 0 means no bias!). So when does this occur? First, let us figure out when there is no omitted variable bias:

- When $\beta_2 = 0$ (meaning no relationship between omitted x and y), then $\beta_2 \delta_1 = 0$.
- When $\delta_1=0$ (meaning no relationship between omitted x and D_i), then $\beta_2\delta_1=0$

Thus, omitted variable bias is only a problem when their is **both** a correlation between omitted x and y, and a correlation between omitted x and treatment D.

• This is the definition of a confounder variable as well! One that affects selection into treatment $x \to D$ and has a backdoor path to outcome $x \to y$.

So, omitted variable bias only occurs when we fail to control for confounders!

Causal Interpretation of Regression

Thus, for a causal interpretation of regression and OLS estimates, we must be confident that all confounders are controlled for.

- Often times, this is impossible, since many causal effects have unobservable or impossible to measure confounders.
- Thus, to deal with omitted variables and endogeneity when controlling for all confounders is not possible, we will need further techniques (such as instrumental variables).

When this is met, we can interpret β_j of variable x_j as the average causal effect of increasing x_j by one unit on y.

• And we can conduct hypothesis tests to confirm our causal inference.

Note: this assumes that we do not have any measurement issues with our variables (everything is accurately measured). If we have measurement error, endogeneity may also be introduced, and we will be unable to do causal inference.

Another important thing is that non-confounders (variables that do not correlate with both D and y) do not introduce omitted variable bias or endogeneity.

- However, including non-confounders as controls increases our standard error, which decreases the precision of our estimates (and makes it harder to reject the null hypothesis).
- Thus, we should avoid including non-confounders in regression models, as they only make our estimates less consistent, without any benefit.

Model selection (selecting what variables to include in our regression) is one of the most difficult things in causal inference. We must be confident in selecting confounding variables as controls, without selecting non-confounding variables. This is where domain knowledge - expertise of the field you are doing research in - becomes so important. Only through domain knowledge, literature reviews, and intuition, will you be able to create good causal regression models.

The remainder of this book will introduce variations on the regression models we have seen, in order to deal with some of the limitations of causal inference with multiple linear regression.

Additional Topics in Linear Regression

5.1 Linear Probability Model

If our response variable is binary, (i.e. Y only has two values, 0 and 1), then our interpretation differs slightly.

We treat Y as a variable of two categories, Y = 0 and Y = 1. The output \hat{Y}_i indicates the probability of a specific observation i of being in the Y = 1 category - we can also interpret probability in percentages by multiplying by 100.

i Interpretation with Binary Y Variable

For a one-unit increase in X_j , there is an expected $\hat{\beta}_j \times 100$ percentage point change in the probability of being in category Y = 1.

 $\hat{\alpha}$ is the expected probability of being in category Y=1 when all explanatory variables equal 0.

This model with binary Y is also called the **linear probability model**.

- 5.2 Fixed Effects Estimator for Panel Data
- 5.3 Interactions and Conditional Average Treatment Effect
- 5.4 Polynomial Transformations and Estimation
- 5.5 Logarithmic Transformations and Estimation

Part III

Instrumental Variables Estimator

Instrumental Variable Design

- 6.1 Non-Compliance and Reverse Causality
- 6.2 Instrumental Variables
- 6.3 Assumptions for Identification
- 6.4 Causal Estimands and Wald Estimator
- 6.5 Two Stage Least Squares
- 6.6 Weaknesses and Falsification Tests

Continuous Instrumental Variables

Part IV

Quasi Experimental Designs

Differences-in-Differences Design

Expanded Differences-in-Differences

Regression Discontinuity