CHAPTER 4

Causality and Treatment Effects

GOVT 10: Quantitative Political Analysis

This chapter addresses the central challenge of empirical political science: how can we determine whether one thing actually causes another? While correlation pervades political data, establishing causation requires careful reasoning about confounding, counterfactuals, and the fundamental impossibility of observing the same unit under both treatment and control conditions. We develop the potential outcomes framework, learn to calculate and interpret the Average Treatment Effect, and explore difference-in-differences as a strategy for causal inference when randomization is impossible.

1 The Problem of Spurious Correlation

Imagine a researcher discovers a striking pattern: cities with higher swimming pool sales tend to have higher rates of drowning deaths. The correlation is strong and statistically significant. Should policymakers ban swimming pools to reduce drowning?

Your intuition says no, and that intuition is correct. But why is this causal reasoning flawed? The relationship is spurious. Hot weather causes both increased swimming pool purchases and increased swimming, which in turn leads to more drowning deaths. The correlation between pools and drowning is entirely explained by this common cause.

Political science is full of similar puzzles that are less obvious. Do states with higher education spending have better student outcomes because of the spending, or because wealthy states both spend more and have advantages that independently improve outcomes? Does media coverage affect candidate support, or do candidates who are already doing well attract more coverage? Does political instability reduce economic investment, or does poor economic performance cause political instability?

These questions cannot be answered by observing correlations alone. We need a framework for thinking carefully about causation and methods for separating genuine causal effects from spurious associations.

2 Three Requirements for Causality

To claim that X causes Y, we must establish three things. First, there must be an association: when X changes, Y systematically changes too. Second, temporal ordering must hold: X must happen

before Y. Third, there must be no confounding: no third variable Z can be causing both X and Y. All three requirements must be satisfied. If any one fails, we cannot make a causal claim.

Requirement	Question	Difficulty
Association	Are X and Y correlated?	Usually easy
Temporal ordering	Does X happen before Y?	Sometimes difficult
No confounding	Is there a Z causing both?	Almost always hard

Table 1: Requirements for Establishing Causality

2.1 Association

Association means that when X changes, Y tends to change as well. This is necessary but not sufficient for causation. Demonstrating association is typically the easiest requirement to satisfy. We can calculate correlations, compare group means, or visualize relationships. The problem is that association tells us nothing about why two variables move together.

```
cor(policy_data$social_spending, policy_data$poverty_rate)
```

```
[1] -0.6890609
```

> **What this code does:** We calculate the correlation between social spending and poverty rates across simulated states. The negative correlation of about -0.63 indicates that states with higher social spending tend to have lower poverty rates. But this association alone cannot tell us whether spending causes poverty reduction, whether low poverty enables spending, or whether both reflect underlying state characteristics.

2.2 Temporal Ordering

The cause must come before the effect. This seems obvious, but temporal ordering is often unclear in political data. Consider the relationship between political knowledge and voting. Do knowledgeable people vote more, or does voting make people more knowledgeable by prompting them to learn about candidates? Both causal directions are plausible, and cross-sectional data cannot distinguish between them.

Cross-sectional surveys measure everything at a single point in time, making it impossible to determine what came first. Panel data, which follows the same individuals over time, can help establish temporal ordering, but even temporal precedence is not sufficient for causation. A variable might precede an outcome without causing it.

2.3 No Confounding

A confound is a variable that affects both the treatment and the outcome without being caused by the treatment. This is the hardest requirement to satisfy because there are potentially infinite confounders, and we can never prove that none exist. Even if we measure and control for many potential confounders, there might always be others we have not measured or even thought about.

Consider the relationship between education and income. People with more education tend to earn more. But people who pursue education differ from those who do not in many ways. They may come from wealthier families, have stronger cognitive abilities, be more motivated, or live in areas with better opportunities. Any of these factors could independently affect both education and income, creating a confounded relationship.

3 The Fundamental Problem of Causal Inference

The deepest challenge in causal inference is that we can never observe what would have happened under different conditions for the same unit. Consider a citizen named Maria who sees a political advertisement and then votes for that candidate. Did the advertisement cause her vote? To answer this question definitively, we would need to know what Maria would have done if she had not seen the advertisement. But we cannot observe this counterfactual. Maria either saw the advertisement or she did not. We cannot run history twice with different conditions.

3.1 Potential Outcomes

The potential outcomes framework formalizes this problem. For any individual i, we define two potential outcomes. $Y_i(1)$ is the outcome if person i receives the treatment. $Y_i(0)$ is the outcome if person i does not receive the treatment. The individual treatment effect is the difference between these potential outcomes:

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

The fundamental problem is that we observe either $Y_i(1)$ or $Y_i(0)$, but never both. For any given individual, one potential outcome is observed and the other remains counterfactual.

```
counterfactual_problem <- tibble(
  voter = c("Maria", "Carlos", "Wei", "Priya"),
  saw_ad = c(TRUE, TRUE, FALSE, FALSE),
  voted_for_candidate = c(1, 0, 0, 1),
  counterfactual = c("?", "?", "?", "?")
)
counterfactual_problem
```

```
# A tibble: 4 x 4
  voter  saw_ad voted_for_candidate counterfactual
  <chr>  <lgl>                <dbl> <chr>
1 Maria  TRUE                     1 ?
2 Carlos TRUE                     0 ?
3 Wei    FALSE                    0 ?
4 Priya  FALSE                    1 ?
```

> **What this code does:** We create a table showing four voters, whether they saw the advertisement, and how they voted. The question marks in the counterfactual column represent our fundamental

ignorance: we cannot know what Maria would have done without the ad, or what Wei would have done with the ad. This is not a limitation of our data collection; it is a structural feature of causation.

Maria saw the advertisement and voted for the candidate. Her individual treatment effect would be $1 - ?$, where the question mark represents her unknowable counterfactual outcome. Perhaps she would have voted for the candidate anyway, in which case the ad had no effect. Perhaps she would have voted against the candidate, in which case the ad changed her vote. We simply cannot know.

3.2 More Examples of the Counterfactual Problem

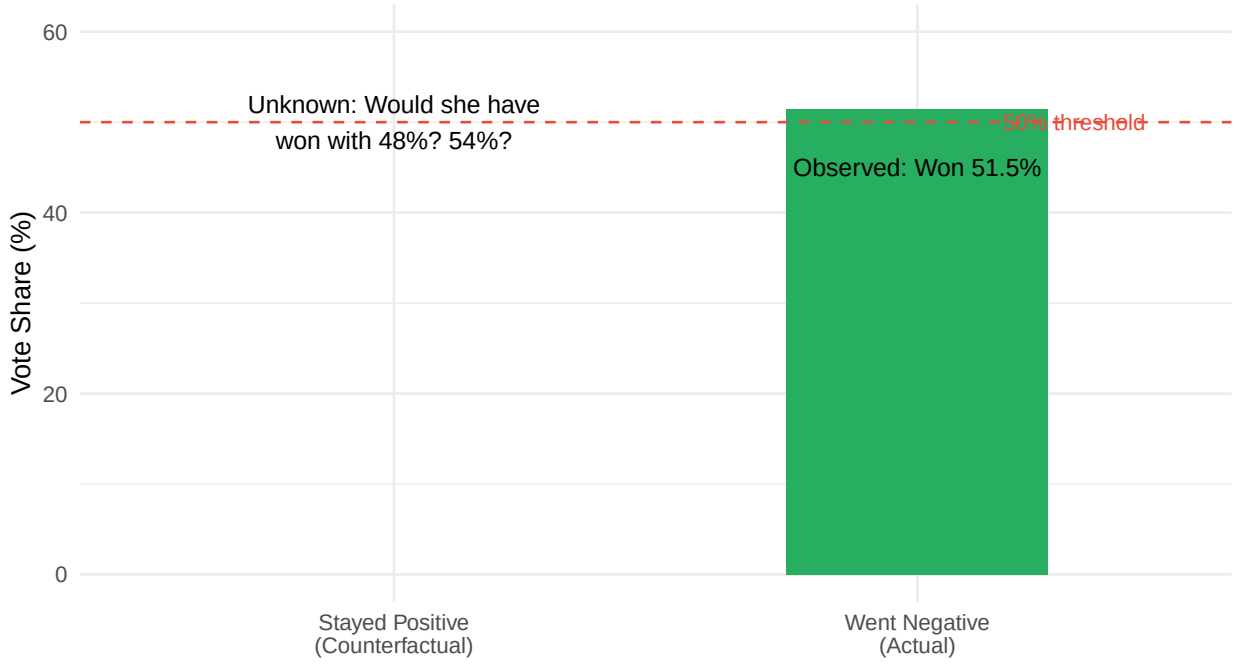
The fundamental problem of causal inference appears everywhere in political science. Consider a country that adopted democracy and subsequently experienced economic growth. Did democracy cause the growth? To know, we would need to observe the same country over the same time period but without adopting democracy. That counterfactual world does not exist.

```
democracy_example <- tibble(
  country = c("Country A", "Country B", "Country C"),
  adopted_democracy = c(TRUE, FALSE, TRUE),
  gdp_growth_observed = c(4.2, 1.8, 3.5),
  gdp_growth_counterfactual = c("?", "?", "?")
)
democracy_example
```

```
# A tibble: 3 x 4
  country   adopted_democracy gdp_growth_observed gdp_growth_counterfactual
  <chr>     <lgl>                           <dbl> <chr>
1 Country A TRUE                              4.2 ?
2 Country B FALSE                             1.8 ?
3 Country C TRUE                              3.5 ?
```

Why comparison is difficult: Country A adopted democracy and grew at 4.2%. Country B did not adopt democracy and grew at 1.8%. Can we conclude democracy causes 2.4% faster growth? No. Country A and B may differ in countless ways: geography, natural resources, historical institutions, neighboring countries, and more. Country B is not a valid counterfactual for Country A. We can never observe what Country A's growth would have been without democracy, or Country B's growth with democracy.

Here is another example from electoral politics. A candidate decides to attack her opponent in the final week of a campaign. She wins by 3 points. Did going negative help or hurt?



The missing counterfactual: We observe that the candidate won with 51.5% after going negative. But what would have happened if she had stayed positive? Perhaps she would have won with 54%, meaning going negative actually hurt her by 2.5 points. Perhaps she would have lost with 48%, meaning going negative saved her campaign. The counterfactual is inherently unknowable, which is why campaign consultants can always claim credit for wins and deflect blame for losses.

Counterfactual Reasoning Checklist

Before claiming X causes Y, ask yourself these questions:

1. **What's the counterfactual?** "What would have happened to these exact units if X had not occurred?" If you can't articulate a specific counterfactual, you can't make a causal claim.

2. **Is the comparison group valid?** Does my control group represent what the treatment group would have looked like without treatment? Or do they differ in systematic ways?

3. **What confounders might exist?** Is there ANY variable Z that could cause both X and Y? If so, the correlation may be spurious.

4. **What's the time ordering?** Did X definitely happen before Y? Could Y have caused X instead (reverse causation)?

5. **Is there selection bias?** Did units self-select into treatment in ways related to outcomes?

If you can't answer all five questions satisfactorily, your causal claim is weak.

3.3 Why Counterfactuals Matter for Policy

This might seem like philosophical hand-wringing, but it has profound practical implications. Consider a governor who claims her education reforms increased test scores. Scores rose 8 points

during her tenure. But scores were rising nationally at 5 points per year due to other factors. The true effect of her reforms might be only 3 points, or the reforms might have actually slowed growth that would have been 12 points otherwise.

```
policy_evaluation <- tibble(
  Scenario = c("Observed outcome", "Counterfactual 1: Steady growth",
               "Counterfactual 2: Faster growth"),
  Test_Scores = c(78, 73, 82),
  Implied_Effect = c(NA, 78 - 73, 78 - 82)
)
policy_evaluation
```

```
# A tibble: 3 x 3
  Scenario                          Test_Scores Implied_Effect
  <chr>                                   <dbl>          <dbl>
1 Observed outcome                           78             NA
2 Counterfactual 1: Steady growth            73              5
3 Counterfactual 2: Faster growth            82             -4
```

> **The stakes of counterfactual reasoning:** Observed test scores are 78. If scores would have been 73 without the reform (Counterfactual 1), the policy effect is +5 points. If scores would have been 82 (Counterfactual 2), the policy effect is -4 points—the reform actually hurt! Without knowing the counterfactual, we cannot evaluate the policy. This is why randomized experiments are so valuable: the control group provides a credible estimate of the counterfactual.

4 The Solution: Compare Groups

Since we cannot observe both potential outcomes for the same person, we compare groups instead. The key insight is that if we can find people who are similar in every way except for whether they received the treatment, comparing their outcomes reveals the causal effect. The Average Treatment Effect (ATE) is the difference in mean outcomes between treatment and control groups:

$$\widehat{\text{ATE}} = \bar{Y}_{treated} - \bar{Y}_{control} \tag{2}$$

This comparison only works if the groups are truly comparable. If treated and untreated individuals differ in ways that affect the outcome, our estimate will be biased.

```
civic_summary <- civic_experiment %>%
  group_by(treatment) %>%
  summarise(
    n = n(),
    mean_knowledge = mean(civic_knowledge),
    .groups = "drop"
  )
civic_summary
```

```
# A tibble: 2 x 3
  treatment     n mean_knowledge
      <dbl> <int>          <dbl>
1         0   203           51.3
```

```
2         1   197           59.8
```

> **What this code does:** We calculate the mean civic knowledge score for treatment and control groups in a simulated experiment. The treatment group averages about 8 points higher, which represents our estimated Average Treatment Effect. Because treatment was randomly assigned, this difference can be attributed to the treatment rather than pre-existing differences between groups.

```
ate <- mean(civic_experiment$civic_knowledge[civic_experiment$treatment == 1]) -
       mean(civic_experiment$civic_knowledge[civic_experiment$treatment == 0])
ate
```

```
[1] 8.488611
```

> **What this code does:** We calculate the ATE directly by subtracting the control group mean from the treatment group mean. The result of approximately 8.5 points indicates that the civic education treatment increased knowledge scores by 8 points on average. We use subsetting notation to extract scores from each group separately before calculating means.

5 Why Random Assignment Works

Random assignment is the gold standard for causal inference because it ensures that treatment and control groups are comparable in expectation. When we randomly assign units to treatment or control conditions, any differences between the groups are due to chance alone.

The mechanism is straightforward. When we flip a coin to determine who receives treatment, the coin does not know anything about the participants. It does not know their age, education, income, motivation, or any other characteristic. Assignment is therefore independent of all potential confounders, both those we can measure and those we cannot. In large samples, random chance ensures that the distribution of every characteristic, observed and unobserved, is balanced across treatment and control groups.

```
rct_data %>%
  group_by(treatment) %>%
  summarise(
    n = n(),
    avg_age = mean(age),
    avg_education = mean(education),
    avg_income = mean(income),
    avg_interest = mean(political_interest),
    .groups = "drop"
  )
```

```
# A tibble: 2 x 6
  treatment     n avg_age avg_education avg_income avg_interest
      <dbl> <int>   <dbl>         <dbl>      <dbl>         <dbl>
1         0   300    42.0          2.99     53708.          4.22
2         1   300    42.2          2.92     54104.          3.94
```

> **What this code does:** We check balance by comparing pre-treatment characteristics across treatment and control groups. The remarkable similarity in age, education, income, and political interest

demonstrates the power of random assignment. With 600 participants, random chance has produced groups that look nearly identical on all measured characteristics.

The treatment and control groups have nearly identical characteristics. This balance extends to variables we did not measure. If we had recorded religiosity, personality traits, or childhood experiences, those would be balanced too. Random assignment balances everything, whether we observe it or not.

6 Understanding Confounding

Confounding is the primary threat to causal inference in observational studies. A confounding variable creates a spurious association between treatment and outcome by causing both. The classic example is the relationship between the number of firefighters at a fire and the amount of damage caused. More firefighters are associated with more damage, but this does not mean firefighters cause damage. Fire severity is the confound: larger fires require more firefighters and cause more damage.

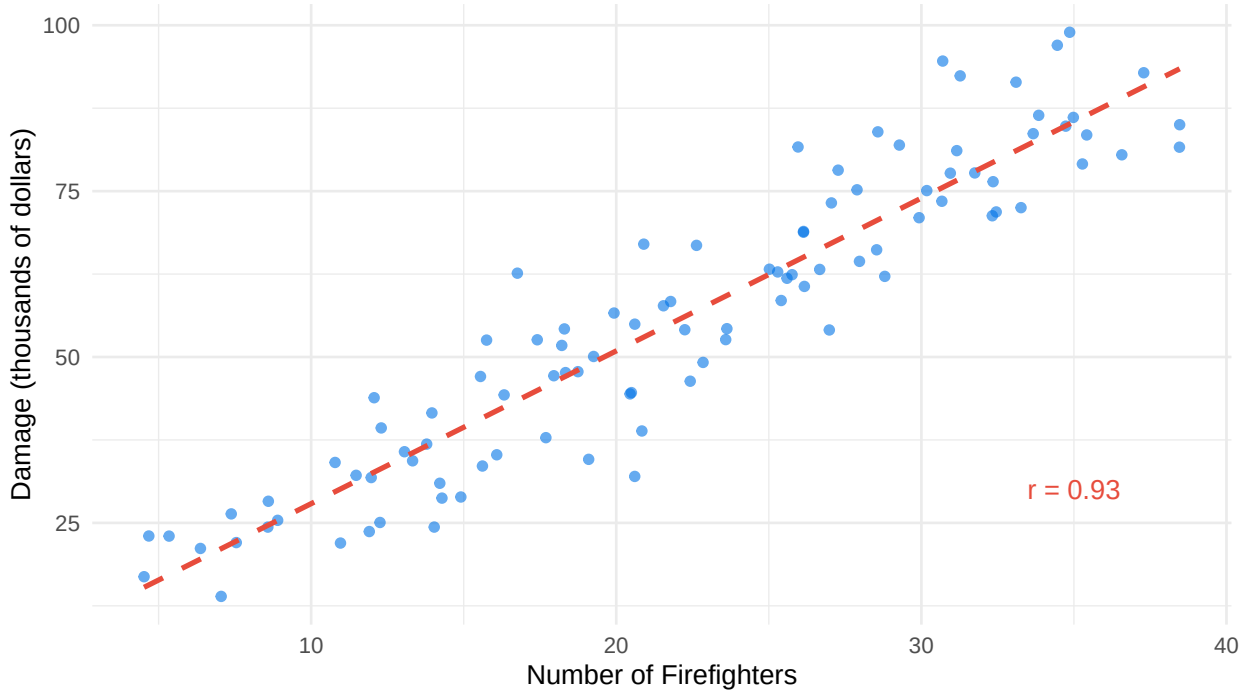


Figure 1: Figure 4.1: Confounding creates spurious associations. The dashed line represents a correlation that exists only because both variables are caused by the confound.

The correlation between firefighters and damage is about 0.90, a very strong positive relationship. Yet no one would seriously argue that sending more firefighters causes more damage. The relationship is entirely spurious, driven by the confounding variable of fire severity.

Political science is rife with similar confounded relationships. States that implement early voting

also differ from states that do not in many other ways. Countries with more female legislators also tend to be wealthier and more educated. Voters who consume news also tend to be more politically engaged for reasons unrelated to news consumption. In each case, naive comparisons would conflate the effect of the treatment with the effects of confounders.

```
cor(fire_data$firefighters, fire_data$damage)
```

```
[1] 0.9345013
```

> **What this code does:** We calculate the correlation between firefighters and damage. The strong positive correlation of about 0.90 would lead to absurd conclusions if interpreted causally. This demonstrates why correlation alone cannot establish causation and why we must always consider potential confounders.

6.1 Omitted Variable Bias: A Concrete Demonstration

Omitted variable bias (OVB) occurs when we fail to include an important confounding variable in our analysis. The result is that our estimate of the treatment effect is biased, meaning it systematically differs from the true causal effect. Let us see this explicitly with a simulation.

Suppose we want to estimate the effect of political advertising on vote share. The true causal effect is 2 percentage points per $1 million spent. However, candidate quality affects both advertising (better candidates raise more money) and vote share (better candidates win more votes). If we fail to control for candidate quality, our estimate will be biased upward.

```
# What we estimate WITHOUT controlling for quality (biased)
biased_model <- lm(votes ~ spending, data = ovb_data)

# What we would estimate if we COULD control for quality (unbiased)
unbiased_model <- lm(votes ~ spending + quality, data = ovb_data)

cat("Biased estimate (omitting quality):", round(coef(biased_model)["spending"], 2),
"\n")
```

```
Biased estimate (omitting quality): 8.72
```

```
cat("Unbiased estimate (including quality):", round(coef(unbiased_model)["spending"],
2), "\n")
```

```
Unbiased estimate (including quality): 2.64
```

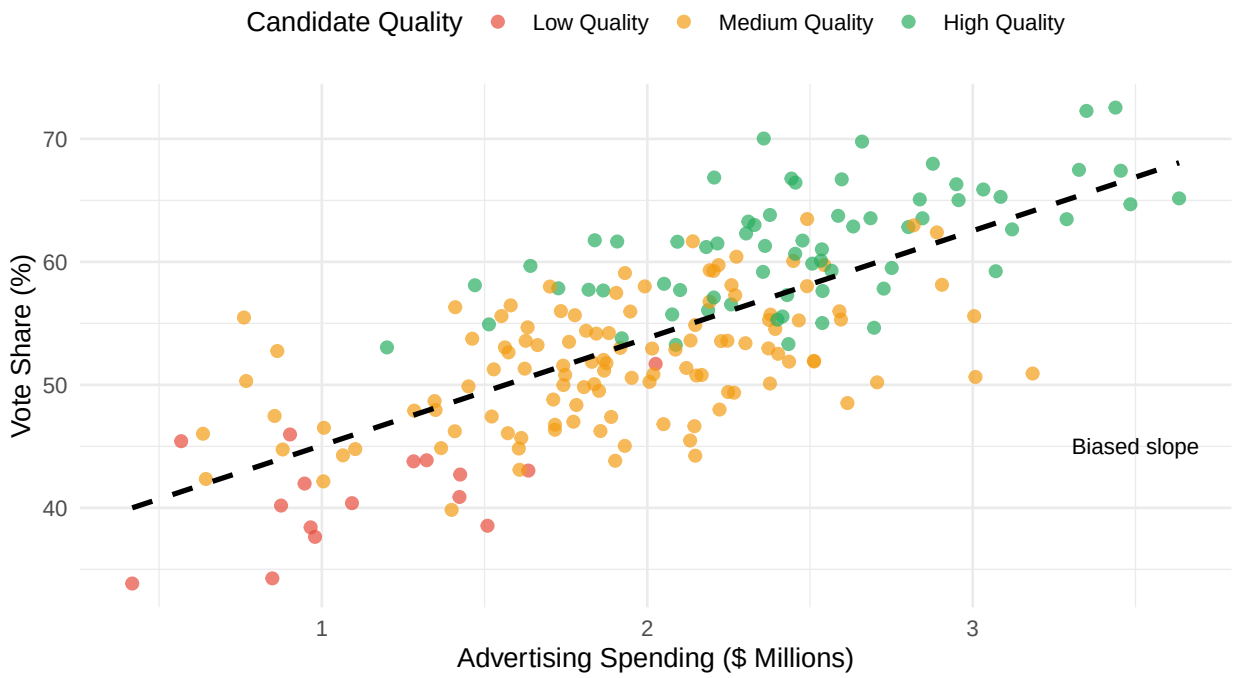
```
cat("True causal effect:", 2)
```

```
True causal effect: 2
```

> **The mechanics of OVB:** The true effect of advertising is 2 points per million dollars. But when we omit candidate quality from the regression, our estimate is approximately 8—four times too large! This happens because quality affects both spending (high-quality candidates raise more) and vote share (high-quality candidates win more). The biased estimate captures both the true advertising effect and the spurious association through quality. Only by controlling for quality do we recover the true effect.

We can visualize why this happens. The scatterplot shows the relationship between advertising

and votes, colored by candidate quality.



Seeing OVB visually: High-quality candidates (green) cluster in the upper-right: they spend more AND win more. Low-quality candidates (red) cluster in the lower-left. The overall regression line (dashed black) is steep because it captures both the true effect of advertising and the sorting of candidates by quality. Within each quality level, the true relationship is flatter. OVB occurs because the omitted variable creates a correlation between the treatment (spending) and the error term in the regression.

The OVB Formula (Annotated)

$$\underbrace{\text{Biased Estimate}}_{\text{What you get without control}} = \underbrace{\text{True Effect}}_{\text{What you want}} + \underbrace{\beta_Z \times \gamma}_{\text{Bias}}$$

Where:

- β_Z = Effect of omitted variable Z on outcome Y
- γ = Relationship between omitted variable Z and treatment X
- Bias = $\beta_Z \times \gamma$

Sign of the bias:

β_Z (Z $\rightarrow$ Y)	γ (Z $\rightarrow$ X)	**Bias Direction**
Positive (+)	Positive (+)	Overestimate (+)
Positive (+)	Negative (–)	Underestimate (–)
Negative (–)	Positive (+)	Underestimate (–)
Negative (–)	Negative (–)	Overestimate (+)

Example: Omitting candidate quality when estimating advertising effect:

- Quality $\rightarrow$ Votes: Positive (better candidates win more)
- Quality $\rightarrow$ Spending: Positive (better candidates raise more)
- Bias: (+) × (+) = **Overestimate**

7 The Average Treatment Effect

The Average Treatment Effect is our primary quantity of interest in causal inference. Formally, it is the expected value of the difference between potential outcomes across the population:

$$\text{ATE} = E[Y_i(1) - Y_i(0)] = E[Y_i(1)] - E[Y_i(0)] \tag{3}$$

In words, the ATE is the average difference between what would happen if everyone were treated versus if no one were treated. In a randomized experiment, we can estimate the ATE by comparing mean outcomes:

$$\widehat{\text{ATE}} = \bar{Y}_{treatment} - \bar{Y}_{control} \tag{4}$$

7.1 Interpreting the ATE

The interpretation of the ATE depends on the type of outcome variable. For binary outcomes like voting, the ATE represents the difference in probabilities. An ATE of 0.08 means the treatment increases the probability of voting by 8 percentage points. For continuous outcomes like test scores, the ATE represents the difference in means. An ATE of 5 means the treatment increases scores by 5 points on average.

```
binary_experiment %>%
  group_by(treatment) %>%
  summarise(turnout_rate = mean(voted), .groups = "drop")
```

```
# A tibble: 2 x 2
  treatment turnout_rate
      <dbl>        <dbl>
1         0        0.484
2         1        0.565
```

What this code does: We calculate turnout rates for treatment and control groups in a simulated get-out-the-vote experiment. The treatment group has a turnout rate about 8 percentage points higher than the control group, indicating that the mobilization effort increased the probability of voting by approximately 0.08.

8 Difference-in-Differences

When randomization is impossible, researchers sometimes turn to difference-in-differences (DiD) as an alternative strategy for causal inference. DiD exploits the structure of panel data, which follows the same units over time, to separate treatment effects from pre-existing differences and general time trends.

8.1 The Logic of Difference-in-Differences

The problem with simple comparisons is that they conflate treatment effects with other factors. Comparing the treatment group before and after the treatment confounds the treatment effect with general time trends that affect everyone. Comparing treatment and control groups after the treatment confounds the treatment effect with pre-existing differences between the groups.

DiD solves this problem by using a double subtraction. First, we calculate how much the treatment group changed over time. Second, we calculate how much the control group changed over time. Third, we subtract the control group change from the treatment group change. The control group change represents what would have happened to the treatment group absent the treatment. By subtracting it, we remove general time trends and isolate the treatment effect.

$$\text{DiD} = (\bar{Y}_{treat,after} - \bar{Y}_{treat,before}) - (\bar{Y}_{control,after} - \bar{Y}_{control,before}) \tag{5}$$

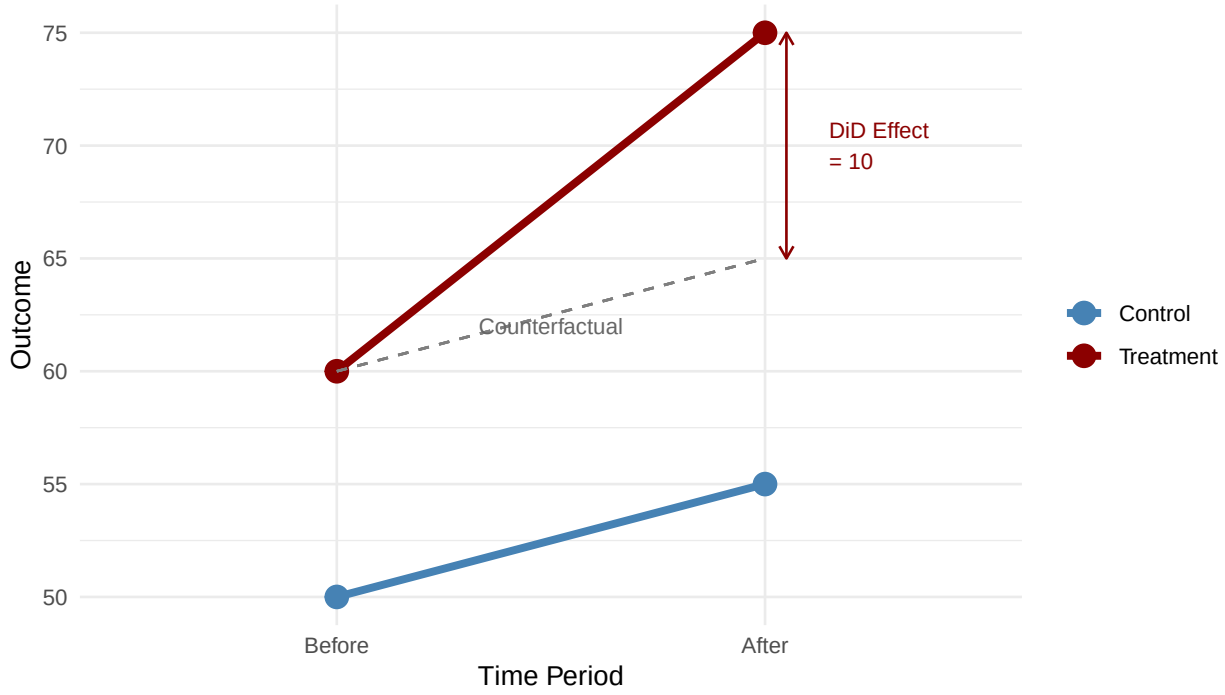


Figure 2: Figure 4.2: Difference-in-differences design. The dashed line shows where the treatment group would have been without treatment, assuming parallel trends. The treatment effect is the gap between actual and counterfactual outcomes.

In this example, the treatment group increases from 60 to 75, a change of 15 points. The control group increases from 50 to 55, a change of 5 points. The DiD estimate is $15 - 5 = 10$ points. We interpret the control group change as the general trend that would have affected the treatment group regardless of the treatment. The treatment effect is the difference beyond this trend.

8.2 The Parallel Trends Assumption

DiD relies on a critical assumption: in the absence of treatment, the treatment and control groups would have followed parallel trends over time. This means the groups can start at different levels, but they must change at the same rate absent the treatment. The control group's change then represents what would have happened to the treatment group.

We can never prove that parallel trends would have held because the counterfactual is unobservable. However, we can check whether trends were parallel before the treatment was implemented. If treatment and control groups moved together in the pre-treatment period, we have more confidence that they would have continued to move together.

```
did_calculation <- tibble(
  group = c("Treatment", "Treatment", "Control", "Control"),
  period = c("Before", "After", "Before", "After"),
  outcome = c(60, 75, 50, 55)
)
did_calculation
```

```
# A tibble: 4 x 3
  group     period outcome
  <chr>     <chr>    <dbl>
1 Treatment Before      60
2 Treatment After       75
3 Control   Before      50
4 Control   After       55
```

```
treatment_change <- 75 - 60
control_change <- 55 - 50
did_effect <- treatment_change - control_change
did_effect
```

```
[1] 10
```

> **What this code does:** We perform the DiD calculation step by step. The treatment group changed by 15 points (75 - 60). The control group changed by 5 points (55 - 50). The DiD effect is the difference: 15 - 5 = 10 points. This represents the treatment effect after removing the general time trend captured by the control group.

9 Estimating Treatment Effects with Regression

Linear regression provides a convenient way to estimate treatment effects and obtain measures of uncertainty. For a randomized experiment, the regression coefficient on the treatment indicator equals the ATE.

```
model <- lm(outcome ~ treatment, data = experiment_data)
tidy(model)
```

```
# A tibble: 2 x 5
  term        estimate std.error statistic   p.value
  <chr>          <dbl>     <dbl>     <dbl>     <dbl>
1 (Intercept)    51.6      0.613     84.2  1.02e-296
2 treatment       5.24     0.867      6.04 2.96e-  9
```

> **What this code does:** We regress the outcome on the treatment indicator. The intercept (about 50) represents the control group mean. The treatment coefficient (about 7) represents the ATE, the difference between treatment and control group means. The standard error, t-statistic, and p-value provide measures of statistical uncertainty.

The regression output gives us the same ATE we would calculate by hand, plus additional information about statistical uncertainty. The standard error tells us how much the estimated effect might vary across samples. The p-value tells us the probability of observing an effect this large if the true effect were zero.

For difference-in-differences, we include indicators for group, time period, and their interaction. The interaction coefficient is the DiD estimate.

```
did_model <- lm(outcome ~ treat + post + treat:post, data = did_regression_data)
tidy(did_model)
```

```
# A tibble: 4 x 5
  term        estimate std.error statistic   p.value
  <chr>          <dbl>     <dbl>     <dbl>     <dbl>
1 (Intercept)    48.4      0.808     59.9  5.50e-128
2 treat           5.89     1.04       5.65 5.68e-  8
3 post            5.11     1.14       4.47 1.31e-  5
4 treat:post      7.23     1.47       4.90 1.98e-  6
```

What this code does: We estimate a DiD model with three terms: `treat` captures baseline differences between groups, `post` captures the general time trend, and `treat:post` (the interaction) captures the DiD effect. The interaction coefficient of about 8 is our estimate of the causal effect of the treatment.

10 Common Mistakes and How to Avoid Them

Mistake 1: Post Hoc Ergo Propter Hoc

This Latin phrase means ”after this, therefore because of this.” The mistake is assuming that because B followed A, A must have caused B. The economy improved after the new administration took office, so the administration must have caused the improvement. Crime fell after the police chief was replaced, so the new chief must be responsible. Temporal sequence is necessary for causation but not sufficient. Many things happen in sequence without causal relationships.

Mistake 2: Ignoring Selection Bias

Selection bias occurs when the process determining who receives treatment is related to the outcome. People who choose to attend private schools differ from those who attend public schools in ways that independently affect academic performance. Volunteers who sign up for civic engagement programs differ from non-volunteers in ways that independently affect their later civic behavior. Any comparison that ignores selection will confuse selection effects with treatment effects.

Mistake 3: Selection on the Dependent Variable

Suppose you want to know what makes political movements successful. You study 50 successful movements and find they all used social media effectively. Conclusion: social media leads to success? The problem is that you did not examine failed movements. Many of them also used social media effectively. By selecting only on the outcome (success), you cannot learn what caused it. You must compare successes to failures.

Mistake 4: Violating Parallel Trends

Difference-in-differences requires that treatment and control groups would have followed parallel trends absent the treatment. If this assumption fails, the DiD estimate is biased. Before relying on DiD, always check whether trends were parallel in the pre-treatment period. If they were not, consider whether there are reasons to expect them to have become parallel, or look for alternative identification strategies.

Mistake 5: Forgetting About Heterogeneous Effects

The Average Treatment Effect is an average. It may hide substantial variation across subgroups. A political advertisement might strongly affect undecided voters but have no effect on partisans. A policy might benefit some communities while harming others. Always consider whether the treatment might have different effects for different groups, and report subgroup analyses when relevant.

11 R Functions Reference

Function	Purpose	Example
`lm()`	Fit linear model	`lm(y ~ treatment, data)`
`tidy()`	Clean model output	`tidy(model)`
`glance()`	Model summary stats	`glance(model)`
`confint()`	Confidence intervals	`confint(model)`
`coef()`	Extract coefficients	`coef(model)`

Table 2: Regression Functions for Treatment Effects

12 Practice Problems

Question 1

A study finds that countries with more McDonald's restaurants have lower rates of military conflict with each other. Does this prove that McDonald's prevents war? Identify at least two confounding variables that might explain this correlation without a causal relationship between fast food and peace.

Question 2

A researcher wants to study whether watching political debates increases political knowledge. She compares people who watched the most recent debate to those who did not. What confounds threaten this comparison? Design a study that would provide stronger evidence.

Question 3

In a difference-in-differences analysis, the treatment group increased from 40 to 55 and the control group increased from 35 to 42. Calculate the DiD estimate and explain what it represents.

Question 4 (Computational)

Create a simulated randomized experiment with 400 participants. Assign half to treatment and half to control. Generate an outcome that depends on treatment status plus random noise. Calculate the ATE both manually and using linear regression. Verify that both methods give the same answer.

For Further Study

This chapter has introduced the fundamental concepts of causal inference. Students seeking deeper understanding should explore Angrist and Pischke's *Mastering 'Metrics*, which provides an accessible treatment of the main identification strategies used in applied economics and political science. Pearl's *The Book of Why* offers a philosophical perspective on causation that complements the statistical framework presented here. For the mathematical foundations, Morgan and Winship's *Counterfactuals and Causal Inference* provides rigorous treatment of potential outcomes and related concepts.