

## **Class 8: Cause and effect**

**API-201**

Quantitative Analysis and Empirical Methods I

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### **Agenda**

1. Simpson's paradox
2. A model of causality
3. Randomized experiments

## **A motivating example**

### **Test preparation**

Suppose students who elect to take a test preparation course ultimately end up doing better than those who do not.

What can we conclude about the efficacy of the course?

## Motivating example

Graduate school admissions rates at Berkeley [ 1973 ]

	Applicants	Admitted
Men	8442	44%
Women	4321	35%

Can we conclude that women are admitted at lower rates *because of* their gender? What else might be going on?

[ Discuss with your neighbor. ]

## Simpson's paradox

Trends across groups can disappear or even reverse when groups are disaggregated.

Difficult [ impossible ] to fully identify causal effects from *observational* data.

## Experiments

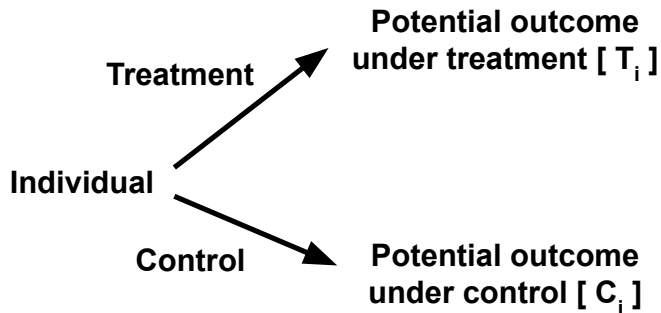
One way to rigorously identify causal effects is through **randomized controlled trials [ RCTs ]**.

We'll return to this idea shortly. But first we need to develop a theory of causality.

## What is causality?

## Neyman-Rubin Causal Model

Potential outcomes



## Neyman-Rubin Causal Model

Unit-level causal effect

Unit-level causal effect is  $T_i - C_i$

- $T_i$  is the potential outcome under treatment. For example, how well a student *would have* done had they taken the test prep.
- $C_i$  is the potential outcome under control. For example, how well a student *would have* done had they not taken the test prep.

## Neyman-Rubin Causal Model

### Fundamental problem of causal inference

It is *impossible* to observe both potential outcomes for a given individual. Cannot *directly* observe unit-level causal effects.

[ Why not? ]

But — amazingly — we can estimate the *average* causal effect across individuals.

## Neyman-Rubin Causal Model

### Average causal effect

$$\bar{T} - \bar{C} = \frac{1}{N} \sum_{i=1}^N T_i - C_i$$

## Neyman-Rubin Causal Model

Average causal effect

$$\begin{aligned}\bar{T} - \bar{C} &= \frac{1}{N} \sum_{i=1}^N T_i - C_i \\ &= \frac{1}{N} \sum_{i=1}^N T_i - \frac{1}{N} \sum_{i=1}^N C_i\end{aligned}$$

Unit-level treatment effect  
can't be observed

Average outcome under  
treatment can be estimated!

Average outcome under  
control can be estimated!

## Neyman-Rubin Causal Model

Estimating the average causal effect

1. Pick a random set of subjects and treat them.
2. Average observed outcome for treatment group is a good estimate of the average treatment potential outcome.
3. Average observed outcome for control group is a good estimate of the average control potential outcome.
4. The difference in these estimates is a good estimate of the average causal effect.

## Estimating the effect of test prep

To estimate the effect of test prep, we can run a **randomized controlled trial**:

1. Offer test prep to a **randomly** selected subset of students.
2. Look at the **difference** in average test performance between those students who were offered test prep and those who were not [ *difference-in-means estimator* ].

## Interventions

Causal effects are inherently tied to *interventions*. We always ask, “what is the effect of this intervention on outcomes?”

The mantra of causal inference is “no causation without manipulation” [ *Holland, 1986* ]

## Interventions

We can ask about the “**effect of a cause**” but not clear how to make statistical sense of the “**cause of an effect.**”

- We can ask, “What is the effect of test prep on academic performance?”
- Harder to make sense of, “Why do some students have better academic performance?”

But policymakers and others are inclined to ask “why” questions, which are inherently hard / impossible to answer!

**With your neighbor come up with “why” questions, and transform them into “intervention” questions.**

## Graduation admissions redux

What would it mean to say gender or race *causes* lower admissions rates? What is the intervention?

[ Discuss with your neighbor. ]



## Implicit assumptions

### Non-interference

The Neyman-Rubin causal model is simple and elegant. But it has several limitations.

A key assumption is **non-interference**: potential outcomes for a unit depend only on the treatment assignment for that unit. We assume no one's treatment assignment affects anyone else's potential outcomes.

**Imagine conducting a randomized controlled trial to measure the effectiveness of a COVID vaccine. Is non-interference violated? [ Discuss with your neighbor. ]**

## Non-compliance

### Assignment $\neq$ treatment

Not everyone who is randomly assigned to the treatment condition will ultimately undergo treatment [ and not everyone who's assigned to control condition will comply ].

## Non-compliance

Assignment  $\neq$  treatment

In our test prep example, *offering* students test prep is not the same as ensuring they *complete* test prep.

The difference in performance between those [ randomly ] offered test prep and those [ randomly ] not offered test prep is called the **intention-to-treat** estimate.

**Come up with another example where compliance would likely be an issue.** [ Discuss with your neighbor. ]

## Key takeaways

The potential outcomes framework is a powerful way to conceptualize causal effects.

Key to this approach is thinking about the **intervention**. *What would have happened had one done something else?*

[ “No causation without manipulation.” ]

We can use **randomized controlled trials [ RCTs ]** to estimate average treatment effects. [ Next time we’ll talk about estimating causal effects with observational data. ]