1. Potential Outcomes and Randomization

PhD Applied Methods

Duncan Webb
NovaSBE

Spring 2026

Intros

- **Me**: Duncan Webb
  - Development economist (field experiments in India, Madagascar, Colombia)
  - Email: dmbwebb@gmail.com — Office: B115B
  - Office hours: Wednesdays 1–2pm

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- **You**: Name, research interests, what you hope to get from this course, what you did before starting the PhD

Course Structure

- **Goal**: Deep understanding of modern causal inference methods
- **Format**: One 3-hour class per week
- **Topics**:
  1. Potential Outcomes and Randomization (today)
  2. Instrumental Variables
  3. Difference-in-Differences
  4. Regression Discontinuity Design
  5. Empirical Tools

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  5. Empirical Tools
- **Approach**: Theory + applications + implementation

Assessment

- **Problem sets** (20%): 4 problem sets throughout the course
  - Applied/empirical questions (Stata, R, or Python)
  - Mathematical/theoretical questions
  - Individual work
  - Due Tuesdays at 12pm, starting next week
  - Submit to `dmbwebb@gmail.com`: **.tex**, **.pdf**, and **code files**
  - Include your name in the output

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- **Final exam** (80%): 1.5-hour closed-book exam
  - Theoretical derivations and proofs
  - Applied reasoning and interpretation

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- **Questions?**

Causality and understanding the world

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Causality and understanding the world

- "We do not have knowledge of a thing until we have grasped its why, that is to say, its cause." $\sim$ Aristotle
- Not all research estimates a causal relationship, but the implication or takeaway of a paper is **almost always** a causal one
- Particularly important for **policy evaluation**:
  - What is the effect of microfinance on consumption?
  - What is the effect of reducing class size on education outcomes?
  - What is the effect of reducing the price of a good on its consumption?

Causality and correlation

- For a very long time, economists made causal claims based on **correlations** and very shaky assumptions
- Until the **credibility revolution** (Angrist and Pischke, 2010) which formalized the conditions under which we could claim causality
  - And the use of **randomization** (or quasi-random events) to make those claims
- **Goal for this class**: Deep understanding of the tools we can use to make causal claims

Rubin's potential outcomes framework

- The **potential outcomes framework** gives us a precise framework for thinking about when we can correctly claim to estimate the causal effect of some treatment
- The goal is to estimate the **causal effect** of some treatment, e.g.,
  - "Small class at school"
  - "Job training program"
  - ...

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- The goal is to estimate the **causal effect** of some treatment, e.g.,
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  - "Job training program"
  - ...
- Note that you can also have multiple treatments (e.g., small class, medium class, large class) and continuous treatments (University fees), but we'll get to that later

Counterfactuals

- **Outcome measure** is the outcome we care about
- Let Y_i be the observed outcome for individual i
- For example:
  - i's wages in adulthood when examining impact of a job training program
  - i's test scores at school (for class size)
  - How much i discriminates against a minority (for prejudice-reduction intervention)

Counterfactuals

For each person i we assume there are two potential outcomes:

- $Y_i(1)$ is the outcome we would observe **if** she received the treatment
- $Y_i(0)$ is the outcome we would observe **if** she did not receive the treatment

We can compactly write this by defining $D_i \in \{0, 1\}$ as the treatment status of individual i, and the counterfactuals are $Y_i(D_i)$

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If some individuals get the treatment, and some don't... **what can we observe?**

Counterfactuals

- If i is not treated ($D_i = 0$) then we **only** observe $Y_i(0)$ and $Y_i(1)$ is an unobserved counterfactual
- If i is not treated ($D_i = 1$) then we **only** observe $Y_i(1)$ and $Y_i(0)$ is an unobserved counterfactual

Counterfactual quiz

Let's say our "treatment" (D_i) is a **job training program**:

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- What is $Y_i(0)$ for someone in the job training?
- Can we observe $Y_i(1)$ for someone who doesn't get the training?
- What are $Y_i(0)$ and $Y_i(1)$ for someone who isn't even in the data?

Causal effects

Using this framework, how would we write the **causal effect of the treatment on individual** *i*?

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

This is the main thing we are trying to estimate!

In general, $Y_i(1)$ and $Y_i(0)$ can be different across people, and so Δ_i may be different for each person too ("heterogeneous treatment effects")

Fundamental identification problem

Question: What is the fundamental difficulty with estimating Δ_i, the causal effect of the treatment on individual i?

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Answer: For a specific person i, we do not and **cannot even principle** observe both $Y_i(1)$ and $Y_i(0)$.

Example: We do not know what exactly what would have happened to Donald Trump (and the world) if he had not been shot, because in fact he was. So we cannot know for sure the causal effect of him being shot.

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This is called the **fundamental identification problem**.

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1. **Partial equilibrium**: these counterfactuals are implicitly defined in a given environment, e.g., what would have happened to Homer *if* he got a job training program, but holding fixed the macroeconomic situation

Some assumptions

What are some assumptions built into my stipulation that there are some values $Y_i(1)$, $Y_i(0)$?

1. **Partial equilibrium**: these counterfactuals are implicitly defined in a given environment, e.g., what would have happened to Homer *if* he got a job training program, but holding fixed the macroeconomic situation
2. **Treatment doesn't affect untreated** ("Stable Unit Treatment Value Assumption"): if Homer gets a job training program, that doesn't affect the counterfactuals of Marge or Ned Flanders
   - So this rules out general equilibrium effects, externalities, Hawthorne effects, etc.

Heterogeneous treatment effects

Call $D_i = 0$ if untreated and $D_i = 1$ if treated

Because the effect can be heterogenous, many evaluation parameters. In particular:

$$ATE := \mathbb{E}[Y_i(1) - Y_i(0)] \tag{2}$$
$$ATT := \mathbb{E}[Y_i(1) - Y_i(0)|D_i = 1] \tag{3}$$

- **ATE**: Average treatment effect - all population
- **ATT**: Average treatment on the treated - treated only
(for instance, weak students are treated first)

Observed outcomes

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If $D_i = 1$ then we observe $Y_i(1)$
If $D_i = 0$ then we observe $Y_i(0)$
Think of it as a "binary switch"

NB: We can equivalently write this as $Y_i = Y_i(0) + D_i(Y_i(1) - Y_i(0)) = Y_i(0) + D_i\Delta_i$ (i.e., in terms of the effect)

Second quiz

- What is the observed outcome when $D_i = 1$?

Second quiz

- What is the observed outcome when $D_i = 1$?
- What is the unobserved counterfactual when $D_i = 0$?

Regression with constant treatment effects

Consider a simple regression model:

$$Y_i = \alpha + D_i\beta + u_i \tag{5}$$

Question: What counterfactuals $Y_i(0)$ and $Y_i(1)$ generate this model?

What does OLS estimate?

What does the OLS estimator of β measure in this regression?

$$Y_i = \alpha + D_i\beta + u_i \tag{6}$$

OLS estimates the difference in means

Under $\mathbb{E}[u_i|D_i] = 0$ (no selection bias):

$$\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0] = \alpha + \beta - \alpha + \mathbb{E}[u_i|D_i = 1] - \mathbb{E}[u_i|D_i = 0] = \beta \tag{7}$$

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So OLS gives us β. But what exactly *is* β in terms of treatment effects?

(Note: This model assumes constant treatment effects, i.e., $\Delta_i = \beta$ for all i. We'll revisit what happens when effects are heterogeneous later.)

Outline

What do we observe?

Let's think again about what can we actually observe in the data?

$$\mathbb{E}[Y_i(0)|D_i = 0]? \tag{8}$$
$$\mathbb{E}[Y_i(1)|D_i = 1]? \tag{9}$$
$$\mathbb{E}[Y_i(1)|D_i = 0]? \tag{10}$$
$$\mathbb{E}[Y_i(0)|D_i = 1]? \tag{11}$$

What do we observe?

Identification problem: we "**observe**"

$$\mathbb{E}[Y_i(0)|D_i = 0] \tag{12}$$
$$\mathbb{E}[Y_i(1)|D_i = 1] \tag{13}$$

but not the counterfactuals

$$\mathbb{E}[Y_i(1)|D_i = 0] \tag{14}$$
$$\mathbb{E}[Y_i(0)|D_i = 1] \tag{15}$$

But what would we need to estimate the ATT, for instance?

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But what would we need to estimate the ATT, for instance?

$$ATT = \overbrace{\mathbb{E}[Y_i(1)|D_i = 1]}^{\text{"observed"}} - \overbrace{\mathbb{E}[Y_i(0)|D_i = 1]}^{\text{"unobserved"}} \tag{16}$$

Hypothesis to identify ATT:

$$\mathbb{E}[Y_i(0)|D_i = 1] = \mathbb{E}[Y_i(0)|D_i = 0] = \mathbb{E}[Y_i(0)]$$

i.e. no selectivity: treated "are like" untreated

Then

$$ATT = \overbrace{\mathbb{E}[Y_i(1)|D_i = 1]}^{\text{"observed"}} - \overbrace{\mathbb{E}[Y_i(0)|D_i = 1]}^{\text{"unobserved"}} \tag{17}$$

$$= \overbrace{\mathbb{E}[Y_i(1)|D_i = 1]}^{\text{"observed"}} - \overbrace{\mathbb{E}[Y_i(0)|D_i = 0]}^{\text{"observed"}} \tag{18}$$

In this very simple case, compare empirical means in each group

The counterfactual for a group is simply the observed outcome of the other group

NB: to identify *ATE* we need more:

NB: to identify ATE we need more:

$$\mathbb{E}[Y_i(0)|D_i] = \mathbb{E}[Y_i(0)] \text{ and } \mathbb{E}[Y_i(1)|D_i] = \mathbb{E}[Y_i(1)]$$

$$ATE = \overbrace{\mathbb{E}[Y_i(1)]}^{\text{“unobserved”}} - \overbrace{\mathbb{E}[Y_i(0)]}^{\text{“unobserved”}} \tag{19}$$

$$= \overbrace{\mathbb{E}[Y_i(1)|D_i = 1]}^{\text{“observed”}} - \overbrace{\mathbb{E}[Y_i(0)|D_i = 0]}^{\text{“observed”}} \tag{20}$$

Under these assumptions, we also identify the ATT - **why**?

NB: to identify ATE we need more:

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Under these assumptions, we also identify the ATT - **why**?

$\implies$ Because we assumed that the counterfactuals are similarly distributed (on average) in both populations

But, in general

$$\mathbb{E}[Y_i(0)|D_i = 1] \neq \mathbb{E}[Y_i(0)|D_i = 0]$$

The "naive" estimator (difference in observed means) is then a biased estimator for ATT:

$$\mathbb{E}[Y_i(1)|D_i = 1] - \mathbb{E}[Y_i(0)|D_i = 0] \tag{21}$$

$$= \underbrace{[\mathbb{E}[Y_i(1)|D_i = 1] - \mathbb{E}[Y_i(0)|D_i = 1]]}_{} + \underbrace{[\mathbb{E}[Y_i(0)|D_i = 1] - \mathbb{E}[Y_i(0)|D_i = 0]]}_{} \tag{22}$$

$$= ATT + \text{Selection Bias} \tag{23}$$

where bias $[\mathbb{E}[Y_i(0)|D_i = 1] - \mathbb{E}[Y_i(0)|D_i = 0]]$ is the difference between (average) counterfactual $Y_i(0)$ in the two populations (treated and untreated)

Why is selection bias quite likely?

Simple **Roy model**: "I am in if this is worth it"

$$D_i = 1 \text{ if } Y_i(1) - Y_i(0) > c$$

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Then, in general

$$\mathbb{E}[Y_i(0)|D_i = 1] = \mathbb{E}[Y_i(0)|Y_i(0) < Y_i(1) - c] \tag{24}$$
$$\neq \mathbb{E}[Y_i(0)|Y_i(0) \geq Y_i(1) - c] = \mathbb{E}[Y_i(0)|D_i = 0] \tag{25}$$

In this case, selectivity stems from

- **Comparative advantages** ($Y_i(1) - Y_i(0)$ large for some, small for others). Most simple instance: participants have smaller $Y_i(0)$, thus larger potential gain
- **Heterogeneity in cost** c (if it is correlated with $Y_i(0)$)

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Another reason for selection bias: **administrative rules**

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For instance:

- **"Cream-skimming"**: they choose "the best", and $\mathbb{E}[Y_i(0)|D_i = 1] > \mathbb{E}[Y_i(0)|D_i = 0]$
- **Remedial targeting**: e.g. focus on intervening with weak kids in the class, so $\mathbb{E}[Y_i(0)|D_i = 1] < \mathbb{E}[Y_i(0)|D_i = 0]$

Link with endogeneity

Selectivity will lead to to **endogeneity** of D_i in a regression

For simplicity, focus on a simple model with homogenous effects, i.e. $\Delta_i = \beta$ for everyone, and $u_1 = u_0 = u_i$:

$$Y_i = \alpha + D_i\beta + u$$

Selectivity is then:

$$\mathbb{E}[Y_i(0)|D_i = 1] \neq \mathbb{E}[Y_i(0)|D_i = 0] \tag{26}$$

$$\Rightarrow \mathbb{E}[u_i|D_i = 1] \neq \mathbb{E}[u_i|D_i = 0] \tag{27}$$

A least-squares regression would confound treatment effect (β) with the fact that the distribution of u_o is different in treated and untreated populations:

$$\mathbb{E}[Y_i|D_i = 0] = \alpha + \mathbb{E}[u_i|D_i = 0] \tag{28}$$
$$\mathbb{E}[Y_i|D_i = 1] = \alpha + \beta + \mathbb{E}[u_i|D_i = 1] \tag{29}$$

and OLS is an estimator of

$$\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0] = \beta + (\mathbb{E}[u_i|D_i = 1] - \mathbb{E}[u_i|D_i = 0]) \tag{30}$$
$$= \beta + \text{Bias} \tag{31}$$

A second problem: Heterogeneous treatment effects

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> What if treatment effects **vary across individuals**?
> That is, what if $\Delta_i = Y_i(1) - Y_i(0)$ differs from person to person?

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This matters because even **without selection bias**, OLS may not estimate ATE or ATT if effects are heterogeneous.

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Examples where effects vary:

- Job training: More effective for workers with less experience
- Class size reduction: May help struggling students more
- Medicine: Effects vary with age, weight, genetics
- Education subsidies: Returns higher for high-ability students

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$\Rightarrow$ In most applications, treatment effects are **heterogeneous** across individuals

A more general model

Allow potential outcomes to differ flexibly across individuals:

$$Y_i(0) = g_0(X_i) + u_{0i} \tag{32}$$
$$Y_i(1) = g_1(X_i) + u_{1i} \tag{33}$$

where:

- g_0, g_1 = functions of observable characteristics X_i
- u_{0i}, u_{1i} = unobservable components (can differ by treatment status)

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Key difference from simple model:

- The effect can vary with X_i (observable heterogeneity)
- The effect can vary with $u_{1i} - u_{0i}$ (unobservable heterogeneity)

Heterogeneous treatment effects

Recall: $Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0)$

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Substituting our general model:

$$Y_i = g_0(X_i) + D_i \underbrace{[g_1(X_i) - g_0(X_i) + u_{1i} - u_{0i}]}_{\Delta_i} + u_{0i} \tag{34}$$

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The individual treatment effect is now:

$$\Delta_i = g_1(X_i) - g_0(X_i) + u_{1i} - u_{0i} \tag{35}$$

This varies across individuals—it's a **random coefficient**!

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$$\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0] = \mathbb{E}[g_1(X_i) + u_{1i}|D_i = 1] - \mathbb{E}[g_0(X_i) + u_{0i}|D_i = 0] \tag{36}$$

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Problem: OLS compares g_1 for *treated* against g_0 for *untreated*

But treated and untreated may have different X_i distributions.

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Problem: OLS compares g_1 for *treated* against g_0 for *untreated*

But treated and untreated may have different X_i distributions.

Key result: Even if $\mathbb{E}[u_{ki}|D_i] = 0$ (no selection bias), OLS estimates **neither** ATE nor ATT in general.

Summing up: Two problems for causal inference

Problem 1: Selection bias

- Treated and untreated differ in ways that affect outcomes
- $\mathbb{E}[Y_i(0)|D_i = 1] \neq \mathbb{E}[Y_i(0)|D_i = 0]$
- OLS confounds treatment effect with pre-existing differences

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Problem 2: Heterogeneous effects

- Treatment effects vary across individuals (Δ_i not constant)
- Even without selection bias, OLS may not estimate ATE or ATT
- Depends on how X_i is distributed across treatment groups

Summing up: Two problems for causal inference

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- $\mathbb{E}[Y_i(0)|D_i = 1] \neq \mathbb{E}[Y_i(0)|D_i = 0]$
- OLS confounds treatment effect with pre-existing differences

Problem 2: Heterogeneous effects

- Treatment effects vary across individuals (Δ_i not constant)
- Even without selection bias, OLS may not estimate ATE or ATT
- Depends on how X_i is distributed across treatment groups

Question: How can we solve **both** problems?

Outline

Idea behind experiments

Simplest way to identify treatment causal effect: make likely the hypotheses

$$\mathbb{E}[Y_i(0)|D_i = 1] = \mathbb{E}[Y_i(0)|D_i = 0] = \mathbb{E}[Y_i(0)] \tag{37}$$
$$\mathbb{E}[Y_i(1)|D_i = 1] = \mathbb{E}[Y_i(1)|D_i = 0] = \mathbb{E}[Y_i(1)] \tag{38}$$

If we draw treated and untreated **randomly** from a population then:

$$ATT = \overbrace{\mathbb{E}[Y_i(1)|D_i = 1]}^{\text{“observed”}} - \overbrace{\mathbb{E}[Y_i(0)|D_i = 1]}^{\text{“unobserved”}} \tag{39}$$
$$= \overbrace{\mathbb{E}[Y_i(1)|D_i = 1]}^{\text{“observed”}} - \overbrace{\mathbb{E}[Y_i(0)|D_i = 0]}^{\text{“observed”}} \tag{40}$$

Can be estimated with empirical means (or via regressions)

Idea behind experiments

- **Intuition**: if we randomly select who receives the treatment and who doesn't, then on average it will be similar types of people in each group, and so the average counterfactuals will be the same
- Therefore, any difference we **do** observe after the treatment must be **caused by the treatment**
- This is why randomized controlled trials are called the **gold standard** of evidence (somewhat controversially)
- **Other methods for causal inference** are built on this paradigm – other identification methods "mimic" random assignment into treatment

Randomization solves both problems

Problem 1: Selection bias

- Randomization ensures $\mathbb{E}[Y_i(0)|D_i = 1] = \mathbb{E}[Y_i(0)|D_i = 0]$
- No systematic differences between treated and untreated groups

Randomization solves both problems

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Problem 2: Heterogeneous effects

- Randomization ensures X_i is distributed identically across groups
- So $\mathbb{E}[g_k(X_i)|D_i = 1] = \mathbb{E}[g_k(X_i)|D_i = 0]$ for $k = 0, 1$

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- Randomization ensures X_i is distributed identically across groups
- So $\mathbb{E}[g_k(X_i)|D_i = 1] = \mathbb{E}[g_k(X_i)|D_i = 0]$ for $k = 0, 1$

Key insight: Randomization ensures OLS = ATE, **even with heterogeneous treatment effects**!

Use of randomized controlled trials

- **Popularity**: AER+JPE+QJE, 0.8% of published articles in 1983 $\rightarrow$ 8.2% in 2011 (while theory: 58% $\rightarrow$ 19%)
- **Nobel Prize** in economics to Esther Duflo, Abhijit Banerjee, Michael Kremer for pioneering this methodology in development economics.
- **Infrastructure** - organisations like J-PAL and IPA provide infrastructure for this kind of research

Critiques of randomized controlled trials

Critiques of randomized controlled trials

- **Equilibrium effects** - standard methodology ignores spillover effects or equilibrium effects, although frontier methods and large trials can understand these (see e.g., Egger et al, *Econometrica* 2022)
- **External validity** - how to generalize from the context of the RCT to other contexts
- **Ethics** - is it ethical to deny treatment to the control group? This depends on the context, and what the alternative is, e.g., it's no longer ethical to deny proven medical treatment
- **Mechanisms** - early RCTs tried to measure treatment effects, but high-quality studies now focus a lot on understanding mechanisms using additional treatments or by explicitly testing theory-driven models

Simplest design

Randomize individuals and compare treated and untreated:

$$\bar{Y}_1 - \bar{Y}_0 = \frac{1}{N_1} \sum_{i \in D_i = 1} Y_i \quad - \quad \frac{1}{N_0} \sum_{i \in D_i = 0} Y_i$$

Similar to OLS on

$$y = \alpha + \beta D_i + u$$

(Leave as an exercise to prove it algebraically using OLS matrix formula)

Real life design issues in RCTs

1. **Balance** between treatment groups in a finite sample
2. **Adding controls**
3. **Imperfect compliance**

Real example: reducing class size in "STAR" program

EXPERIMENTAL ESTIMATES OF EDUCATION PRODUCTION FUNCTIONS*

ALAN B. KRUEGER

This paper analyzes data on 11,600 students and their teachers who were randomly assigned to different size classes from kindergarten through third grade. Statistical methods are used to adjust for nonrandom attrition and transitions between classes. The main conclusions are (1) on average, performance on standardized tests increases by four percentile points the first year students attend small classes; (2) the test score advantage of students in small classes expands by about one percentile point per year in subsequent years; (3) teacher aides and measured teacher characteristics have little effect; (4) class size has a larger effect for minority students and those on free lunch; (5) *Hawthorne* effects were unlikely.

1. **Balance** between treatment groups

TABLE I

COMPARISON OF MEAN CHARACTERISTICS OF TREATMENTS AND CONTROLS: UNADJUSTED DATA

A. Students who entered STAR in kindergarten[b]

Variable	Small	Regular	Regular/Aide	Joint *P*-Value[a]
1. Free lunch[c]	.47	.48	.50	.09
2. White/Asian	.68	.67	.66	.26
3. Age in 1985	5.44	5.43	5.42	.32
4. Attrition rate[d]	.49	.52	.53	.02
5. Class size in kindergarten	15.1	22.4	22.8	.00
6. Percentile score in kindergarten	54.7	49.9	50.0	.00

2. **Adding controls** with an OLS regression of an RCT

This shows the effect of D_i ("small class") on Y_i (percentile on standardized test score).

TABLE V

OLS AND REDUCED-FORM ESTIMATES OF EFFECT OF CLASS-SIZE ASSIGNMENT ON AVERAGE PERCENTILE OF STANFORD ACHIEVEMENT TEST

Explanatory variable	Reduced form: initial class size (5)	(6)	(7)	(8)
Small class	4.82	5.37	5.36	5.37
	(2.19)	(1.25)	(1.21)	(1.19)
Regular/aide class	.12	.29	.53	.31
	(2.23)	(1.13)	(1.09)	(1.07)
White/Asian (1 = yes	—	—	8.35	8.44
			(1.35)	(1.36)
Girl (1 = yes)	—	—	4.48	4.39
			(.63)	(.63)
Free lunch (1 = yes)	—	—	−13.15	−13.07
			(.77)	(.77)
White teacher	—	—	—	−.57
				(2.10)
Teacher experience	—	—	—	.26
				(.10)
Master's degree	—	—	—	−.51
				(1.06)
School fixed effects	No	Yes	Yes	Yes
R^2	.01	.25	.31	.31

Adding controls

OLS often used because it allows you to add controls: **Why?**

Adding controls

OLS often used because it allows you to add controls: **Why?**

If assignment is truly random, conditioning on X_i should not affect point estimates

We have $\mathbb{E}[Y_i(0)|D_i] = \mathbb{E}[Y_i(0)]$ and $\mathbb{E}[X_i|D_i] = \mathbb{E}[X_i]$

Therefore OLS on $Y_i = X_i\gamma + \beta D_i + u$ gives (asymptotically) the same β as $Y_i = \beta D_i + u_i'$ (Frisch-Waugh theorem)

Adding controls

But it's still useful because it increases precision: **Why?**

Adding controls

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$$V(\hat{\beta}) = \sigma^2(X'X)^{-1}$$

where σ^2 is residual variance

$$y = \beta D_i + u' \tag{41}$$
$$y = \beta D_i + x\gamma + u \tag{42}$$

$V(u') = V(x\gamma) + V(u) > V(u)$

Thus, the second equation estimates the same β but with more precision

Depends on how much X_i explain Y_i (and may **not hold in finite samples**)

Controls and precision

Effect of D_i ("small class") on Y_i (percentile on standardized test score):

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Example: Stratify by education (e.g., high school vs college) and gender $\Rightarrow$ 4 strata. Within each, randomly assign 50% to treatment.

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Key difference: Stratified randomization ensures representation from each subgroup

Why use stratified randomization?

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1. Ensures balance on stratification variables

- With simple randomization, treatment and control groups may differ on key characteristics (especially with small samples)
- Stratification **guarantees** balance on the stratification variables
- Example: Exactly 50% of treated are female if you stratify by gender

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- Stratification **guarantees** balance on the stratification variables
- Example: Exactly 50% of treated are female if you stratify by gender

2. Increases precision (lowers standard errors)

- If stratification variables predict outcomes, controlling for strata reduces residual variance
- Recall: $V(\hat{\beta}) = \sigma^2(X'X)^{-1}$ where σ^2 is residual variance
- Lower residual variance $\Rightarrow$ smaller standard errors $\Rightarrow$ more statistical power

Implementing stratified randomization

How to choose stratification variables?

- Pick variables that are:
  - Strong predictors of the outcome (increases precision)
  - Measured before randomization (ensures exogeneity)
  - Create a manageable number of strata (rule of thumb: at least 4-6 observations per stratum-treatment combination)

- Common choices: baseline outcome, gender, age groups, geographic location

Implementing stratified randomization

Specification with stratified randomization:

Include **stratum fixed effects** in your regression:

$$Y_i = \alpha + \beta D_i + \sum_{s=1}^{S} \gamma_s \mathbb{1}[\text{Stratum}_i = s] + u_i \tag{43}$$

- This accounts for how randomization was done
- Improves precision (even though β estimate is similar without FE)
- Standard practice: always control for strata used in randomization

3. Imperfect Compliance

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But what if they don't?

- What happens when some people assigned to treatment don't take it?
- What happens when some people assigned to control get treated anyway?
- Can we still estimate causal effects? If so, *what* causal effects?

Imperfect Compliance: The Problem

In practice, we often **cannot force people to comply** with their assignment

Two types of non-compliance:

- **Non-take-up**: Assigned to treatment but don't take it
- **Crossover**: Assigned to control but take treatment anyway

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Two types of non-compliance:

- **Non-take-up**: Assigned to treatment but don't take it
- **Crossover**: Assigned to control but take treatment anyway

Key question: How does this affect our ability to estimate causal effects?

Example: Krueger Class Size Experiment

What went wrong?

1. Approx. 10% changed class type during the experiment
   - Teacher requests (behavioral problems)
   - Parent pressure
2. Some children changed school or moved (“attrition”)

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- We *encourage* but don’t *force* treatment
- Simpler to implement, more acceptable, often no choice
- Comes at a cost to precision

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Question: What can we still learn?

The Fundamental Insight

Problem: We can no longer directly compare treated vs untreated

- Actual treatment receipt (T_i) is now *endogenous*—a choice!
- Selection bias is back

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- Assignment (D_i) is still *random*
- This comparison is "clean"

This gives us **two distinct questions**:

1. What is the effect of being *assigned* to treatment? (ITT)
2. What is the effect of actually *receiving* treatment? (Wald/IV)

Intention-to-Treat (ITT)

Intention-to-Treat (ITT):

$$ITT = \mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0]$$

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The ITT compares outcomes by **assignment**, ignoring actual treatment

Is this causal? Yes! Assignment is random

Interpretation: The effect of being assigned/offered/encouraged to treatment

When is ITT Useful?

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Policy perspective: If you're implementing a program, ITT tells you the effect of *your policy* (including non-compliance)

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Example: Vaccine rollout

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- Accounts for people who won't show up, refuse, etc.
- This is what a policymaker cares about!

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Policy perspective: If you're implementing a program, ITT tells you the effect of *your policy* (including non-compliance)

Example: Vaccine rollout

- ITT captures real-world effectiveness
- Accounts for people who won't show up, refuse, etc.
- This is what a policymaker cares about!

Limitation: Doesn't tell us the effect of the treatment *itself*

Question: What if we want to know the effect of actually receiving treatment?

Notation: Assignment vs Treatment

We need to distinguish two things:

- $D_i \in \{0, 1\}$: Random **assignment** (what we control)
- $T_i \in \{0, 1\}$: Actual **treatment received** (what we observe)

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- $D_i \in \{0, 1\}$: Random **assignment** (what we control)
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Define compliance probabilities:

$$p_1 = P(T_i = 1 | D_i = 1) \quad \text{– compliance rate among assigned-to-treatment} \tag{44}$$

$$p_0 = P(T_i = 1 | D_i = 0) \quad \text{– crossover rate among assigned-to-control} \tag{45}$$

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$$p_0 = P(T_i = 1 | D_i = 0) \quad \text{– crossover rate among assigned-to-control} \tag{45}$$

Perfect compliance: $p_1 = 1$ and $p_0 = 0$

Imperfect compliance: $p_1 < 1$ or $p_0 > 0$ (or both)

Visualizing Compliance

$D = 0$ *Assigned to control*	$D = 1$ *Assigned to treatment*
Stay in control: $(1 - p_0)$ fraction $T_i = 0$	
———————	**TREATED** $(T_i = 1)$ p_1 fraction
Cross to treatment: p_0 fraction **TREATED** $(T_i = 1)$	

Visualizing Compliance

$D = 0$ *Assigned to control*	$D = 1$ *Assigned to treatment*
Stay in control: $(1 - p_0)$ fraction $T_i = 0$ ——————— **Cross to treatment**: p_0 fraction **TREATED** $(T_i = 1)$	**TREATED** $(T_i = 1)$ p_1 fraction

Key insight: Only D groups are comparable (random), not T groups!

The choice to cross over is *endogenous*

From ITT to Treatment Effect: Intuition

Core logic:

$$\text{ITT} = (\text{effect of treatment}) \times (\text{change in treatment probability})$$

$$\Downarrow$$

$$\text{Treatment effect} = \frac{\text{ITT}}{\text{change in treatment probability}}$$

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$$\Downarrow$$

$$\text{Treatment effect} = \frac{\text{ITT}}{\text{change in treatment probability}}$$

Intuition: If assignment shifts treatment probability by 50%, and outcomes improve by 3, then treatment must improve outcomes by $3/0.5 = 6$

Numerical Example Setup

Setting: 8 students, assigned to small ($D = 1$) or large ($D = 0$) class

$D = 0$ *Assigned to large class*	$D = 1$ *Assigned to small class*
1: untreated – score 5	5: treated – score 17
2: untreated – score 5	6: treated – score 5
3: treated – score 15	7: treated – score 15
4: treated – score 15	8: treated – score 15

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1: untreated – score 5	5: treated – score 17
2: untreated – score 5	6: treated – score 5
3: treated – score 15	7: treated – score 15
4: treated – score 15	8: treated – score 15

Compliance:

- All $D = 1$ students get treatment: $p_1 = 1$
- 2 of 4 $D = 0$ students *also* get treatment: $p_0 = 0.5$

Calculating ITT and Treatment Effect

Step 1: Calculate mean outcomes by assignment

- $\mathbb{E}[Y|D=1] = (17+5+15+15)/4 = 13$
- $\mathbb{E}[Y|D=0] = (5+5+15+15)/4 = 10$

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Step 2: ITT

$$ITT = 13 - 10 = 3$$

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$$\tau = \frac{ITT}{p_1 - p_0} = \frac{3}{1-0.5} = \frac{3}{0.5} = 6$$

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Interpretation: Moving a student from large to small class increases score by 6 points

The Wald Estimator: Formal Derivation

Under constant treatment effects, we can derive:

$$\mathbb{E}[Y|D=1] = \mathbb{E}[Y_i(0)] + \tau \cdot p_1 \tag{46}$$

$$\mathbb{E}[Y|D=0] = \mathbb{E}[Y_i(0)] + \tau \cdot p_0 \tag{47}$$

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Subtracting:

$$\mathbb{E}[Y|D=1] - \mathbb{E}[Y|D=0] = \tau(p_1 - p_0)$$

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Subtracting:

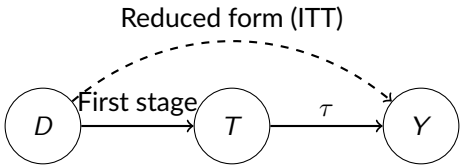
$$\mathbb{E}[Y|D=1] - \mathbb{E}[Y|D=0] = \tau(p_1 - p_0)$$

Solving for τ:

$$\tau = \frac{\mathbb{E}[Y|D=1] - \mathbb{E}[Y|D=0]}{P(T=1|D=1) - P(T=1|D=0)} = \frac{\text{ITT}}{p_1 - p_0}$$

Both numerator and denominator are **observable**

Reduced Form and First Stage



- **Reduced form** ($D \to Y$): Effect of assignment on outcome = ITT
- **First stage** ($D \to T$): Effect of assignment on treatment = $p_1 - p_0$
- **Wald estimator** = Reduced form / First stage

Two-Stage Least Squares (2SLS)

Implementation as a simultaneous equation model:

First stage: Predict treatment from assignment

$$\hat{T}_i = \hat{\pi}_0 + \hat{\pi}_1 D_i$$

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Second stage: Regress outcome on *predicted* treatment

$$Y_i = \beta_0 + \tau \hat{T}_i + u_i$$

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D is an "**instrument**" for T:

- D is exogenous (random assignment)
- D affects Y only through T (exclusion restriction)
- D predicts T (relevance: $\pi_1 \neq 0$)

This is **instrumental variables**—more detail next lecture!

Important Caveat: Who Does This Apply To?

Warning: The Wald/IV estimate is a **Local Average Treatment Effect (LATE)**

It applies to "**compliers**"—those whose treatment status is *changed* by assignment

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This is **NOT the same** as the ATE in general:

- People who always take treatment regardless of assignment? Not included
- People who never take treatment regardless of assignment? Not included
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Preview: We'll study this carefully in the IV lecture—understanding who the compliers are is crucial for interpreting IV estimates

The Cost of Non-Compliance

What is the cost of low compliance?

The Cost of Non-Compliance

What is the cost of low compliance? Lower precision in our estimates

The Cost of Non-Compliance

What is the cost of low compliance? Lower precision in our estimates

With **full compliance**, estimating $Y_i = \alpha + \tau D_i + u_i$ gives:

$$V(\hat{\tau}) = \frac{1}{\bar{D}(1-\bar{D})} \cdot \frac{V(u)}{N}$$

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With **imperfect compliance**, the IV estimator has variance:

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Key insight: Standard errors are inflated by factor $\frac{1}{p_1 - p_0}$

Design Implications

Setting: Population N_0, anticipated compliance rate $\pi_1 = p_1 - p_0$

Two options:

1. Randomize 50% of N_0 and have compliance π_1
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Lesson: More precise estimates if you randomize among a (smaller) population of likely compliers

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With volunteers: You know exactly who is complying

Trade-off: External validity (volunteers may differ from population) vs precision

Summary: Two Approaches to Imperfect Compliance

	ITT	**Wald/IV**
Estimand	Effect of assignment	Effect of treatment
Formula	$\mathbb{E}[Y\|D=1] - \mathbb{E}[Y\|D=0]$	$\frac{\text{ITT}}{p_1 - p_0}$
Identified?	Always	Requires $p_1 \neq p_0$
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Key insight: The choice between ITT and IV depends on your research question

- Policy evaluation? $\rightarrow$ ITT tells you what your program achieves
- Treatment efficacy? $\rightarrow$ IV tells you what the treatment does (for compliers)

Outline

Beyond individual randomization

- So far: randomization at the **individual level**
- Sometimes randomizing individuals is:
  - **Impractical**: Hard to treat some students in a school but not others
  - **Unethical**: Perceived as unfair within communities
  - **Contaminated**: Treatment spills over between individuals

Beyond individual randomization

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- Sometimes randomizing individuals is:
  - **Impractical**: Hard to treat some students in a school but not others
  - **Unethical**: Perceived as unfair within communities
  - **Contaminated**: Treatment spills over between individuals
- **Solution**: Randomize at a higher level - **cluster randomization**
- Treat entire groups (clusters) as units: schools, villages, clinics, firms

Examples of clustered randomization

- **Education**: Randomize schools (not students)
  - Teacher training programs
  - School infrastructure improvements
- **Health**: Randomize clinics or villages
  - Deworming programs (Miguel & Kremer 2004)
  - Community health worker programs
- **Development**: Randomize villages or districts
  - Microfinance expansion
  - Infrastructure projects (roads, electricity)

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Key insight: The unit of randomization $\neq$ unit of analysis

Spillovers and SUTVA violations

Recall our SUTVA assumption: Y_i depends only on own treatment D_i

With spillovers, potential outcomes become:

$$Y_i(D_i, \mathbf{D}_{-i}) \tag{48}$$

where $\mathbf{D}_{-i}$ is the treatment status of others

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Cluster randomization partially solves this:

- Captures within-cluster spillovers
- But still misses cross-cluster spillovers

Notation for clustered designs

Let $c = 1, ..., C$ index clusters, $i = 1, ..., N_c$ index individuals within cluster c

- $D_c \in \{0, 1\}$: treatment status of cluster c
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Observed outcome:

$$Y_{ic} = D_c Y_{ic}(1) + (1 - D_c) Y_{ic}(0) \tag{51}$$

Note: Everyone in the cluster has the same D_c!

Estimation with clustering

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Intuition: 1000 students in 10 schools provides **less information** than 1000 randomly selected students

$\Rightarrow$ Use **cluster-robust standard errors**

Design trade-offs: Number vs size of clusters

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Implications:

- More clusters $>$ bigger clusters (for statistical power)
- If $\rho = 0.05$ and $n = 20$: need $\approx 2\times$ the sample size!
- Rules of thumb: Need at least 20-30 clusters for reliable inference

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Bottom line: Use clustered randomization when spillovers matter or individual randomization is infeasible

Outline

Designing an experiment

Two main questions when designing an experiment:

1. Who to randomize, how, etc.
2. Sample size (and share treated)

Experiments are an unusual case where you have great control over sample size

The last thing you want: go through the whole burden and have insignificant effects because you have high standard errors

Finite sample and inference

So far, we have always considered the asymptotic values of the estimator

For instance:

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

is the asymptotic value of:

$$\frac{1}{N_1} \sum_{i \in D_i = 1} y_i$$

which, inversely, is the empirical counterpart to $\mathbb{E}[Y_i|D_i = 1]$

This is because we have been interested in **identification** (what we would learn in infinite samples)

Finite sample and inference

Random experiment: T and C are similar for $N = \infty$

In finite samples, T and C always *somewhat* different, e.g. by chance my treatment group has slightly older students than the control group

This **imbalance** could be confounded with treatment effect

Inference is accounting for that:
With finite sample, can I consider that the difference T vs. C is high enough to indicate more than unavoidable imbalance?
Yes, if statistically "significant"

Imbalance is not a source of bias; the standard error is there to account for that

Reminder: significance tests

Estimator $\hat{\beta}$ asymptotically normal with mean β and variance $V(\hat{\beta}) = \sigma_\beta^2$

If $\beta = 0$, then, for a risk α (e.g. 5%) we can define $t_{\alpha/2}$ such that:

$$P\left(-t_{\alpha/2} < \frac{\hat{\beta}}{\sigma_\beta} < t_{\alpha/2}\right) = 1 - \alpha$$

Thus

$$2\Phi(t_{\alpha/2}) - 1 = 1 - \alpha$$

and we can read $t_{\alpha/2}$ for the normal distribution table

For $\alpha = 0.05$, $\Phi(1.96) = 0.975$

If $|\hat{\beta}/\sigma_\beta| > 1.96$, we can reject the null $\beta = 0$

Balance table

Table A3: *Baseline balance: covariates*

Variable	(1) Total Mean/(SD)	(2) Control Mean/(SD)	(3) Base + YGL Mean/(SD)	(4) Base Only Mean/(SD)	(3)-(2) Pairwise t-test P-value	(4)-(2) Pairwise t-test P-value
Girl's age (years)	14.000	13.741	14.104	14.033	0.292	0.483
	(6.798)	(6.904)	(6.535)	(7.356)		
Girl has a brother (=1)	0.548	0.574	0.556	0.508	0.512	0.033**
	(0.548)	(0.531)	(0.567)	(0.511)		
Mother passed away (=1)	0.049	0.040	0.050	0.053	0.331	0.282
	(0.210)	(0.183)	(0.223)	(0.209)		
Mother in household (=1)	0.816	0.835	0.805	0.820	0.208	0.595
	(0.450)	(0.469)	(0.439)	(0.459)		
Guardian knows how to read and write (=1)	0.828	0.829	0.836	0.810	0.799	0.474
	(0.485)	(0.479)	(0.514)	(0.428)		
Guardian has no education (=1)	0.095	0.085	0.096	0.102	0.465	0.310
	(0.365)	(0.255)	(0.418)	(0.342)		
Guardian attended secondary or higher education (=1)	0.303	0.308	0.293	0.318	0.648	0.794
	(0.648)	(0.681)	(0.623)	(0.685)		
Guardian occupation: Agriculture (=1)	0.773	0.768	0.781	0.762	0.696	0.899
	(0.666)	(0.716)	(0.632)	(0.697)		
Observations	2390	568	1216	606		
Schools	140	35	70	35		

Notes: Sample includes all girls in baseline. Columns (1)-(4) show means and standard deviations of covariates from the girls' baseline survey. Columns (5)-(6) show the p-value of a pairwise test comparing *Base Only* and *Base + YGL* with *control* , respectively. Standard errors cluster at the school level. $* p < 0.1$, $** p < 0.05$, $*** p < 0.01$.

Overall balance

Often, because we are doing **multiple hypothesis tests** we will get a few significant imbalances when looking across multiple outcomes

How can we test for **overall** imbalance?

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How can we test for **overall** imbalance?

1. Run regression:

$$\text{Treated}_i = \beta_0 + \beta_1 \text{Outcome1}_i + \beta_2 \text{Outcome2}_i + ... + \beta_K \text{OutcomeK}_i \quad (53)$$

2. Use an F-test (joint test) of $\beta_1 = \beta_2 = ... = \beta_K = 0$

The power of the experiment

If the policy has an impact, we want to be able to see it

Unless the effect is very small, we want to reject the null

But if there is a lot of imprecision (large estimator variance), we may fail to do so

Type II error is the probability of a **false negative**, i.e., $\beta > 0$, but we fail to reject the null ($\hat{\beta}/\sigma_\beta < 1.96$). In other words, we fail to detect an effect that is really there.

This will happen sometimes, for some samples

Power = $1 - P$(Type II error), i.e. the probability that we detect an effect if there really is one.

The power of the experiment

Usual approach: set an acceptable power (typically 80%), and then:

1. Set a reasonable β that you feel you should be able to "see" (the **minimum detectable effect** you want
2. And figure out the sample size that ensures that power for a true effect β

Computing the power

Let's calculate the power, where ($\hat{\beta}/\sigma_\beta < 1.96$) and β is random:

$$P\left(\frac{\hat{\beta}}{\sigma_\beta} > t_{\alpha/2}|\beta\right) = \kappa$$

where κ is the power.

$$P\left(\frac{\hat{\beta}-\beta}{\sigma_\beta} > t_{\alpha/2} - \frac{\beta}{\sigma_\beta}|\beta\right) = \kappa$$

$$\Phi\left(\frac{\beta}{\sigma_\beta} - t_{\alpha/2}\right) = \kappa$$

Thus:

$$\frac{\beta}{\sigma_\beta} - t_{\alpha/2} = t_{1-\kappa}$$

Minimum detectable effect

The β that will be "significant" 80% of the time (at 5% level) is such that:

$$\frac{\beta}{\sigma_\beta} - t_{\alpha/2} = t_{1-\kappa}$$

or

$$\beta = (t_{\alpha/2} + t_{1-\kappa})\sigma_\beta$$

with $t_{\alpha/2} = 1.96$ if $\alpha = 0.05$ and $t_{1-\kappa} = 0.84$ if $\kappa = 0.80$

$(t_{\alpha/2} + t_{1-\kappa})\sigma_\beta$ is the **minimum detectable effect** (MDE)

MDE and sample size

Consider the model:

$$y = c + \beta D_i + u$$

Remember that:

$$\sigma_\beta^2 = \frac{1}{\bar{D}(1-\bar{D})}\frac{V(u)}{N}$$

Thus:

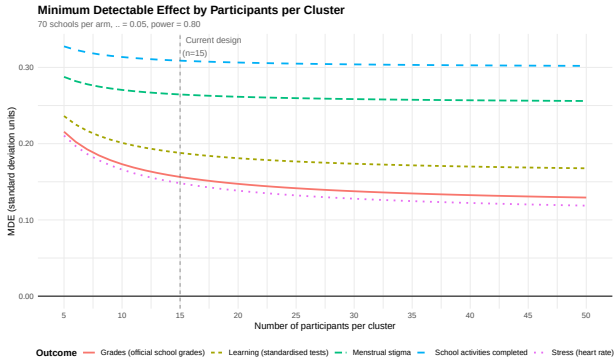
$$\text{MDE} = (t_{\alpha/2} + t_{1-\kappa})\sqrt{\frac{1}{\bar{D}(1-\bar{D})}\frac{V(u)}{N}}$$

Interpret each of those terms... (think in terms of finite sample imbalance)

How does MDE increase with sample size?

MDE and cluster size

Key insight: With clustered randomization, increasing cluster size doesn't decrease MDE much



What matters most is the **number of clusters**, not individuals per cluster

With instrumental variables

$$y = c + \beta T + u$$

where treatment T is instrumented by some random assignment D_i

Reminder:

$$V(\hat{\beta}_{IV}) = \frac{1}{\bar{D}(1-\bar{D})} \frac{V(u)}{N} \frac{1}{\pi_1^2}$$

The precision decreases linearly with the (net) take-up

So does the MDE

If take-up is 50%, implies more than doubling sample size.

Outline

Summing up

1. **Potential outcomes framework** - this gives us a way of conceptualizing counterfactuals and articulating clearly when we can and cannot make causal claims
2. **Randomized controlled trials** are a way to make causal claims with relatively weak assumptions on the data generating process
3. **Design issues** - we learnt about various design issues that come up in RCTs, e.g., dealing with imbalances, calculating statistical power, dealing with imperfect compliance