Lecture 6: Causal Inference - Part 2

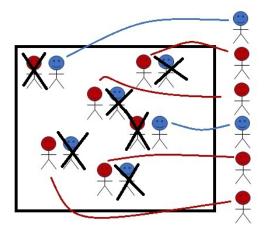
LSE ME314: Introduction to Data Science and Machine Learning (https://github.com/me314-lse)

2025-07-22

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2 Causal 2 Inference

The Fundamental Problem of Causal Inference



Solutions: Assumptions + Data

Yesterday we learned about:

- 1. Experiments when you get to control the assignment of D
- 2. SOO when you assume the DGP of D and Y, and model them

Today:

- 3. Instrumental Variables
- 4. Regression Discontinuity
- 5. Difference-in-Differences

Instrumental Variables

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We want to estimate the effect of watching Squid Game (D) on churn (Y).

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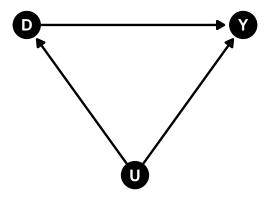
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So any variation in *D* will have **selection** problems.

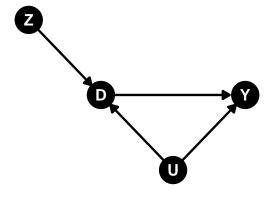
What to do?

(This example is derived from Spotify)

In DAG terms, our Squid Game problem looks like this: U is a canonical confounder driving selection into the show and churn.

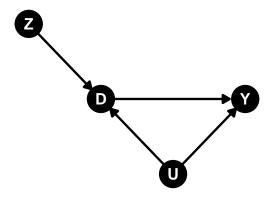


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For example, maybe we can randomize a banner advertising Squid Game on your home page.

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Intuition: We will use only the variation in D induced by Z to study the effect of D on Y.

IV: Setup

Let's start with our building blocks:

- → D is a binary treatment
- → Y is a continuous outcome

We want to know the effect of D on Y.

But the relationship between D and Y is confounded in some way.

We will refer to D as the 'endogenous regressor.'

IV: Setup

Consider now an **encouragement** or **instrument**: $Z_i \in \{0,1\}$

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Treatment potential outcomes under Z = z: $D_{zi} \in \{D_{1i}, D_{0i}\}$

- → $D_{zi} = 1$: would receive the treatment if $Z_i = z$
- → $D_{zi} = 0$: would not receive the treatment if $Z_i = z$
- ightharpoonup e.g., $D_{1i}=1$ encouraged to take treatment and takes treatment

Note: encouragement \neq treatment

Instead: treatment = f(encouragement)

IV: Compliance Types

Compliance: Whether a unit follows the encouragement Z_i .

Given our setup, we can define four compliance types:

- \rightarrow Unit *i* is a complier if: $D_{1i} = 1$ and $D_{0i} = 0$
- → And a non-complier of type:
 - \rightarrow Always-takers: $D_{1i} = D_{0i} = 1$
 - \rightarrow Never-takers: $D_{1i} = D_{0i} = 0$
 - \rightarrow **Defiers**: $D_{1i} = 0$ and $D_{0i} = 1$

IV: Compliance Types

Or, written as principal strata:

	$Z_i = 1$	$Z_i = 0$
$D_i = 1$ $D_i = 0$	Complier / Always-taker Defier / Never-taker	Defier / Always-taker Complier / Never-taker

IV: Potential and Realized Outcomes

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Outcome potential outcomes: $Y_{(Z_i,D_{Z_i})i}$

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What is observed in a given trial?

- → Observed treatment indicator: $D_i = D_{Z_i}$ for $Z_i = z$
- → Observed outcome of Y_i : $Y_i = Y_{(Z_i, D_{Z_i})i}$ for $Z_i = z$
- \rightarrow Thus observed outcome of Y_i can also be written as $Y_i = Y_{Z_i i}$

IV: Estimands

Intention-to-Treat (*ITT*)

$$ITT = \mathbb{E}[Y_{(1,D_{1i})i} - Y_{(0,D_{0i})i}]$$

Read: Effect of encouragement on outcome (regardless of treatment status). We sometimes call this the **reduced form**.

Note: If there is non-compliance, self-selection into the treatment/control groups may imply $ITT \neq ATE$

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If you randomize Z, this is called an **encouragement design**, where $\{Y_{zd}\} \perp Z$.

In that case, our identification result is:

$$ITT = \mathbb{E}[Y_i \mid Z_i = 1] - \mathbb{E}[Y_i \mid Z_i = 0]$$

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$$0 < P(Z = 1) < 1$$
 and $P(D_1 = 1) \neq P(D_0 = 1)$

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 $Z \perp \{Y_{zd}, D_z\}$ (sufficient for ITT)

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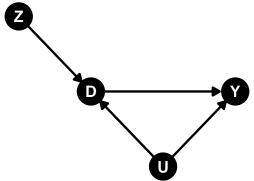
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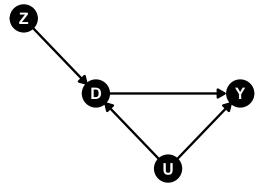
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4. Monotonicity:

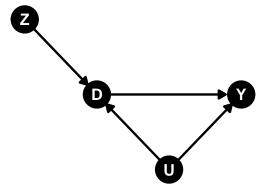
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 ("no defiers")

Intuition: Under these assumptions, we can express the **effect of** D **on** Y in terms of the **ITT** (which is hopefully identified).

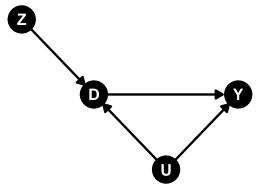




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- 3. Exclusion restriction: Absence of any front-door path between Z and Y.

ITT can be decomposed into a combination of subgroup-specific estimands:

```
ITT = ITT^{c} \times Pr(compliers) + ITT^{a} \times Pr(always-takers) + ITT^{n} \times Pr(never-takers) + ITT^{d} \times Pr(defiers)
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Where:

$$\begin{split} & \textit{ITT}^c = \mathbb{E}[Y_{1i,D_{1i}} - Y_{0i,D_{0i}} \mid D_{1i} = 1, D_{0i} = 0] \\ & \textit{ITT}^a = \mathbb{E}[Y_{1i,D_{1i}} - Y_{0i,D_{0i}} \mid D_{1i} = D_{0i} = 1] \quad \text{etc.} \end{split}$$

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Under monotonicity and exclusion, this simplifies to:

$$ITT = ITT^c \times Pr(text compliers)$$

Therefore, ITT^c can be nonparametrically identified:

$$ITT^c = \frac{ITT}{\mathsf{Pr}(\mathsf{compliers})} = \frac{\mathbb{E}[Y_i \mid Z_i = 1] - \mathbb{E}[Y_i \mid Z_i = 0]}{\mathbb{E}[D_i \mid Z_i = 1] - \mathbb{E}[D_i \mid Z_i = 0]}$$

Therefore, *ITT^c* can be **nonparametrically identified**:

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ITT^c is called the **Local Average Treatment Effect (LATE)** for compliers:

$$ITT^c = LATE = \mathbb{E}[Y_{1i} - Y_{0i} \mid D_{1i} = 1, D_{0i} = 0]$$

IV: Estimation

LATE has a clear **causal interpretation**, but it raises important questions:

- → How do we generalize from compliers to the entire population?
- → Are compliers even interesting?
- → We can never identify individual compliers we only know the group average.
- → Different encouragements (instruments) may identify different complier groups (uh oh).

IV: Estimation

How should we estimate LATE?

Option 1: A plug-in estimator called the Wald estimator:

$$\widehat{\tau_{LATE}} = \frac{\frac{1}{n_1} \sum_{i=1}^{n} Z_i Y_i - \frac{1}{n_0} \sum_{i=1}^{n} (1 - Z_i) Y_i}{\frac{1}{n_1} \sum_{i=1}^{n} Z_i D_i - \frac{1}{n_0} \sum_{i=1}^{n} (1 - Z_i) D_i} = \frac{\widehat{Cov}(Y_i, Z_i)}{\widehat{Cov}(D_i, Z_i)}$$

Where: $n_1 = \sum_{i=1}^{n} D_i$, $n_0 = n - n_1$

IV: Estimation

Option 2: A two-stage least squares (2SLS) estimator:

We assume two DGPs for the potential outcomes:

- 1. First stage: $D_z = \mu + \rho Z + \eta$
- 2. **Second stage**: $Y_{zd} = \gamma + \alpha D + \epsilon$

2SLS estimates these via two OLS steps:

- → Stage 1: Regress D on Z and obtain fitted values \hat{D}_i
- → Stage 2: Regress Y on \hat{D}_i

In R, 2SLS can be implemented with:

- → lm() (manually, but SEs need correction)
- → AER::ivreg (handles SEs properly)

Let's consider some simulated Squid Game viewership data:

	ID	Boredom	Income	${\tt Banner_Ad_SG}$	${\tt Watched_SG}$	Churned_6mo
1	1	-0.31423130	47187.00	0	1	1
2	2	1.87160961	40549.78	0	0	1
3	3	1.26168761	51694.72	0	1	0
4	4	0.85594984	51766.90	0	0	1
5	5	0.72861292	77088.54	0	0	1
6	6	-0.04080788	28675.99	0	0	0

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Finally, we can estimate the **LATE** using the Wald estimator:

```
reduced_form/first_stage
```

```
[1] -0.4526535
```

We can also estimate the LATE using **2SLS**:

```
AER::ivreg(Churned_6mo ~ Watched_SG | Banner_Ad_SG, data = sg_data) %%
 summary()
Call:
AER::ivreg(formula = Churned_6mo ~ Watched_SG | Banner_Ad_SG,
   data = sg_data)
Residuals:
   Min 1Q Median 3Q Max
-0.4629 -0.4629 -0.0102 0.5371 0.9898
Coefficients:
           Estimate Std. Error t value Pr(>|t|)
(Intercept) 0.46285 0.06238 7.420 2.5e-13 ***
Watched SG -0.45265 0.14642 -3.091 0.00205 **
Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
Residual standard error: 0.4458 on 998 degrees of freedom
Multiple R-Squared: 0.005351, Adjusted R-squared: 0.004355
Wald test: 9.557 on 1 and 998 DF, p-value: 0.002047
```

IV: Good Practice

Things to pay attention to:

- 1. Strength of first stage (test this)
- 2. Assignment of and properties of Z (e.g. balance)
- 3. Plausibility of exclusion restriction (theory)
- 4. Focus on interpration (e.g. compliers)
- 5. Characterize the compliers if you can!



RDD: Motivation

I've just hired you at my e-commerce store. We're trying to understand the value of our promised '1-day delivery' product.

Question: Does 1-day delivery (D) affect whether a customer completes a transaction (Y).

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I've just hired you at my e-commerce store. We're trying to understand the value of our promised '1-day delivery' product.

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But promised 1-day delivery is a function of the time of day you checkout.

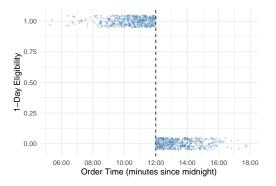
So we have a **selection** problem: when you order is not random, so neither is D!

What to do?

(This example is derived from Instacart)

RDD: Motivation

Our company has a **sharp** rule: If you checkout at 11:59am or earlier, you get 1-day. If it's 12:00pm or later, you get 2+ day delivery.



Intuition: If people who checkout at 11:59am are exactly the same as those who checkout at 12:00pm, then the variation in D among just those customers is independent of potential outcomes.

Formalising the RDD:

- → $D_i \in \{0,1\}$: Treatment
- → X_i: Forcing variable (aka running variable or score) that perfectly determines D_i at cutpoint c:

$$D_i = \mathbf{1}\{X_i \ge c\}$$
 or equivalently $D_i = \begin{cases} 1 & \text{if } X_i \ge c \\ 0 & \text{if } X_i < c \end{cases}$

→ Potential outcomes: $\mathbb{E}[Y_{0i} \mid X_i]$ and $\mathbb{E}[Y_{1i} \mid X_i]$, defined for every value of X_i

Note: X_i may be correlated with Y_{0i} and Y_{1i} ! (It likely is – why?)

Hold up – this looks kind of like selection on observables. . .

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If potential outcomes are a deterministic function of X_i , why not just adjust or control for X_i ?

Lack of common support \rightarrow across all i, only **one** of Y_{0i} and Y_{1i} can be observed for each level of X_i .

RDD: Identification

Intuition: suppose there is no discontinuity in **potential outcomes** $\mathbb{E}[Y_{0i} \mid X_i = x]$ and $\mathbb{E}[Y_{1i} \mid X_i = x]$ at the threshold c.

If $\mathbb{E}[Y_{0i} \mid X_i = x]$ and $\mathbb{E}[Y_{1i} \mid X_i = x]$ can be approximated by some $f(X_i)$, estimate missing potential outcomes by **extrapolating** to $X_i = c$.

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Any difference in Y_i at $X_i = c$ is a causal effect!

Estimand: Local Average Treatment Effect (LATE) at the threshold

$$LATE_{SRD} = \mathbb{E}[Y_{1i} - Y_{0i} \mid X_i = c]$$

Continuity of average potential outcomes:

$$\lim_{\epsilon \uparrow 0} \mathbb{E}[Y_{0i} \mid X_i = c + \epsilon] = \mathbb{E}[Y_{0i} \mid X_i = c]$$
$$\lim_{\epsilon \downarrow 0} \mathbb{E}[Y_{1i} \mid X_i = c + \epsilon] = \mathbb{E}[Y_{1i} \mid X_i = c]$$

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QED

In the continuity framework, estimation is an **extrapolation problem**.

One very simple approach would be to assume a parametric DGP, where LATE is constant, and potential outcomes are linear in X_i :

$$Y_{di} = \alpha + LATE_{SRD} \cdot d + \beta X_i$$

Note: This is an assumption about the DGP – it may well be wrong.

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- 2. Regress: $Y_i = \hat{\alpha} + L\hat{ATED}_i + \hat{\beta}\tilde{X}_i$

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- 1. Recenter forcing variable: $\tilde{X}_i = X_i c$
- 2. Regress: $Y_i = \hat{\alpha} + L\hat{ATED}_i + \hat{\beta}\tilde{X}_i$
- 3. LÂTE is an unbiased estimator of the LATE

We could assume a more **flexible** (realistic?) functional form, e.g., varying slopes in X_i , or polynomial functions of X_i , and fit that regression too.

RDD: Estimation (Local Polynomial Approximation)

Whatever function we choose, we make strong parametric assumptions.

Current state of the art is **local polynomial approximation**, which offers a **non-parametric** estimator of $LATE_{SRD}$.

Proceeds as follows:

- Choose bandwidth or window h
- 2. Choose polynomial order p and kernel function $K(\cdot)$
- 3. Fit two weighted regressions (for $X_i \ge c$ and $X_i < c$) on either side of c to estimate two intercepts: μ_{\downarrow} and μ_{\uparrow}
- 4. Calculate $L\hat{ATE}_{SRD} = \hat{\mu}_{\downarrow} \hat{\mu}_{\uparrow}$

Implemented with rdrobust() in R.

RDD in Action

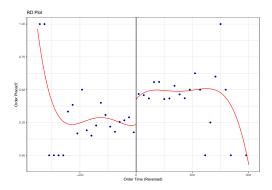
First, let's clean our data and estimate the effect of 1-day delivery on completion using linear regression:

```
# Some data cleaning we need to do:
delivery_data <- delivery_data %>%
 mutate(Order Minutes Rev = 1439 - Order Minutes, # Reverse the minutes
        OMR c = Order Minutes Rev - 720) # Recenter forcing variable
# Now we can estimate the LATE using linear regression:
lm robust(Order Placed ~ OneDay + OMR c, data = delivery data) %>%
 summary()
Call:
lm robust(formula = Order Placed ~ OneDay + OMR_c, data = delivery_data)
Standard error type: HC2
Coefficients:
             Estimate Std. Error t value Pr(>|t|) CI Lower CI Upper DF
(Intercept) 0.2403031 0.027032 8.890 2.821e-18 0.1872569 0.2933493 997
          0.2636326 0.049136 5.365 1.005e-07 0.1672103 0.3600550 997
OneDay
DMR c -0.0002181 0.000211 -1.034 3.015e-01 -0.0006322 0.0001959 997
Multiple R-squared: 0.05407, Adjusted R-squared: 0.05217
F-statistic: 28.64 on 2 and 997 DF, p-value: 8.073e-13
```

RDD in Action

Now let's visualize the RDD effect using rdplot from {rdrobust}:

[1] "Mass points detected in the running variable."



RDD in Action

Number of Obs.

Use rdrobust() to implement local polynomial approx.:

Sharp RD estimates using local polynomial regression.

1000

Number of obs.	1000	
BW type	mserd	
Kernel	Triangular	
VCE method	NN	
Number of Obs.	503	497
Eff. Number of Obs.	281	275
Order est. (p)	1	1
Order bias (q)	2	2
BW est. (h)	89.274	89.274
BW bias (b)	139.518	139.518
rho (h/b)	0.640	0.640
Unique Obs.	204	206

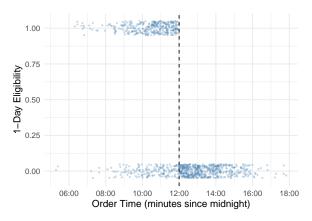
Method	Coef. St	d. Err.	Z	P> z	[95% C.I.]				
Conventional	0.239	0.083	2.878	0.004	[0.076 , 0.402]				
Robust	-	-	2.402	0.016	[0.044 , 0.431]				

RDD: Good Practice

- 1. Check that your setting is actually an RDD (!)
- 2. Visualise the jump in Y as a function of X be very skeptical of these plots!
- Check for discontinuities in background covariates (e.g. lagged Y is great)
- Check 'placebo' discontinuities (other parts of X) but do this on either side of c separately
- Generally stick with rdrobust() defaults. But also see rdhonest.

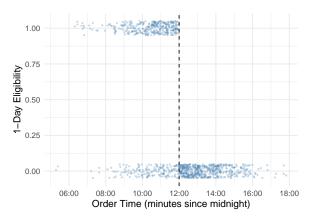
RDD Meets IV: Getting Fuzzy

What if, instead of our company having a sharp cut-off at noon, we had a fuzzy cut-off?



RDD Meets IV: Getting Fuzzy

What if, instead of our company having a sharp cut-off at noon, we had a fuzzy cut-off?



What's going on? If you order late, you will *never* get 1-day delivery, but if you order early, you might (or might not) get it.

This looks like IV!

Formalising this research setting:

→ $Z_i \in \{0,1\}$: Encouragement

This looks like IV!

Formalising this research setting:

- → $Z_i \in \{0,1\}$: Encouragement
- → $D_i \in \{0,1\}$: Treatment, a probabilistic function of Z_i

This looks like IV!

Formalising this research setting:

- → $Z_i \in \{0,1\}$: Encouragement
- → $D_i \in \{0,1\}$: Treatment, a probabilistic function of Z_i
- \rightarrow X_i : Forcing variable perfectly determines Z_i with cutpoint c:

$$Z_i = \mathbf{1}\{X_i \ge c\}$$
 or equivalently $Z_i = \begin{cases} 1 & \text{if } X_i \ge c \\ 0 & \text{if } X_i < c \end{cases}$

Note: The reduced form (effect of Z_i on Y_i) is just a sharp RDD!

Local ITT (LITT) of encouragement at the threshold:

$$LITT = \mathbb{E}[Y_{1i} - Y_{0i} \mid X_i = c]$$

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LATE for compliers at the threshold

$$LATE^c = \mathbb{E}[Y_{1i} - Y_{0i} \mid \text{unit } i \text{ is a complier and } X_i = c]$$

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$$LATE^c = \mathbb{E}[Y_{1i} - Y_{0i} \mid \text{unit } i \text{ is a complier and } X_i = c]$$

Assumptions:

1. 'Augmented' continuity: Both $\mathbb{E}[D_{zi} \mid X_i = x]$ (p.o. for treatment) and $\mathbb{E}[Y_{zi} \mid X_i = x]$ (p.o. for outcome) are continuous in x around $X_i = c$, for z = 0, 1

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- 2. From IV: Monotonicity, exclusion restriction, relevance of Z_i

Parametric estimation for LATE^c:

1. Code instrument: $Z = \mathbf{1}\{X > c\}$

Parametric estimation for LATE^c:

- 1. Code instrument: $Z = \mathbf{1}\{X > c\}$
- 2. Fit 2SLS:

First Stage:
$$D_i = f(X_i) + \beta Z_i + \epsilon_i$$

Second Stage:
$$Y_i = f(X_i) + \alpha \hat{D}_i + \nu_i$$

Note: Specification of $f(\cdot)$ is flexible but must be the same in both stages.

Non-parametric estimation:

- 1. LITT can be estimated using local polynomial approximation, as the LATE was for a sharp RDD. (Why?)
- 2. Proportion of compliers can likewise be estimated with D_i as the outcome
- 3. $LATE_c$ (for compliers at the threshold) is just:

$$LATE_c = \frac{LITT}{Pr(Compliers \mid X_i = c)}$$

Non-parametric estimation:

- 1. LITT can be estimated using local polynomial approximation, as the LATE was for a sharp RDD. (Why?)
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- 3. $LATE_c$ (for compliers at the threshold) is just:

$$LATE_c = \frac{LITT}{Pr(Compliers \mid X_i = c)}$$

Whatever you do, it is **critical** that you test and visualise the first stage. A weak (or non-existent) first stage generates severe bias, and misleads.

Fuzzy RDD in Action

Fuzzy RD estimates using local polynomial regression.

Number of Obs.	1000	
BW type	mserd	
Kernel	Triangular	
VCE method	NN	
v 1 4 60	500	
Number of Obs.	503	497
Eff. Number of Obs.	281	275
Order est. (p)	1	1
Order bias (q)	2	2
BW est. (h)	89.274	89.274
BW bias (b)	139.518	139.518
rho (h/b)	0.640	0.640
Unique Obs.	204	206

First-stage estimates.

Method	Coef. St	d. Err.	z	P> z	[95% C.I.]
Conventional	0.658	0.065	10.195	0.000	[0.532 , 0.785]
Robust	-	-	8.205	0.000	[0.489 , 0.795]

Treatment effect estimates.

Method	Coef. St	d. Err.	z	P> z	[95% C.I.]
Conventional	0.363	0.123	2.956	0.003	[0.122 , 0.604]
Robust	-	-	2.525	0.012	[0.083 , 0.657]







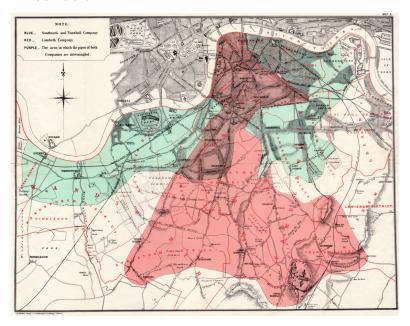


TABLE XII.

Sub-Districts.	Deaths from Cholera in 1849.	Deaths from Cholera in 1854.	Water Supply.
St. Saviour, Southwark . St. Olave	283 157	371 161	
St. John, Horsleydown .	192	148	
St. James, Bermondsey .	249	362	
St. Mary Magdalen .	259	244	
Leather Market	226	237	
Rotherhithe*	352	282	Southwark & Vaux-
Wandsworth	97	59	hall Company only.
Battersea	111	171	,
Putney	8	9	
Camberwell	235	240	
Peckham	92	174	
Christchurch, Southwark Kent Road Borough Road Trinity, Newington St. Peter, Walworth St. Mary, Newington Waterloo Road (1st) Waterloo Road (1st) Waterloo Road (1st) Lambeth Church (1st) Lambeth Church (2nd) Kennington (1st) Kennington (2nd) Brixton Cupton St. George, Camberwell	256 267 312 257 318 446 143 193 243 215 544 187 153 81 114	113 174 270 93 210 388 92 58 117 49 193 303 142 48 165 132	Lambeth Company, and Southwark and Vauxhall Compy.
Norwood Streatham	2 154	10 15	Lambeth Company
Dulwich	1		only.
Sydenham	5	12	The second
First 12 sub-districts .	2261	2458	Southwk. & Vauxhall.
Next 16 sub-districts .	3905	2547	Both Companies.
Last 4 sub-districts .	162	37	Lambeth Company.

90

206

Table 1 John Snow's data on mortality from cholera in areas served by only one of the Southwark & Vauxhall or Lambeth Water Companies before and after the change in water source for the Lambeth Water Company. Rates for all time points were calculated based on 1851 population census

Water supply	Cholera deaths, 1849, rate per 100,000	Cholera deaths, 1854, rate per 100,000	
Lambeth Company Only	847	193	

206

Table 1 John Snow's data on mortality from cholera in areas served by only one of the Southwark & Vauxhall or Lambeth Water Companies before and after the change in water source for the Lambeth Water Company. Rates for all time points were calculated based on 1851 population census

Water supply	Cholera deaths, 1849, rate per 100,000	Cholera deaths, 1854, rate per 100,000	Difference in rates comparing 1854 to 1849, rate per 100,000
Lambeth Company Only	847	193	-653

206

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Southwark & Vauxhall Company only	1349	1466	118
Lambeth Company Only	847	193	-653

206

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Southwark & Vauxhall Company only	1349	1466	118
Lambeth Company Only	847	193	-653
Difference-in-difference, Lambeth versus Southwark & Vauxhall			- 771

206

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Southwark & Vauxhall Company only	1349	1466	118
Lambeth Company Only	847	193	-653
$Difference-in-difference, \ Lambeth \ versus \ Southwark \ \& \ Vauxhall$	502	1273	-771

Units: $i \in \{1, ..., N\}$

Group indicator:

$$G_i = \begin{cases} 1 & \text{(treatment group)} \\ 0 & \text{(control group)} \end{cases}$$

Units in the treatment group receive treatment in t = 1, so:

Treatment indicator: $Z_{it} \in \{0,1\}$

Group	t = 0	t = 1
$G_i = 1$ (treatment)	$Z_{i0} = 0$ (untreated)	$Z_{i1}=1 \ ext{(treated)}$
$G_i = 0$ (control)	$Z_{i0} = 0$ (untreated)	$Z_{i1} = 0$ (untreated)

Define **potential outcomes** $Y_{it}(z)$ as:

- \rightarrow $Y_{it}(0)$: potential outcome for i in period t when untreated
- \rightarrow $Y_{it}(1)$: potential outcome for *i* in period *t* when treated

Note: Pay attention to the notation above!

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Individual causal effect for unit i at time t is:

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Note: Pay attention to the notation above!

Individual causal effect for unit i at time t is:

$$\tau_{it} = Y_{it}(1) - Y_{it}(0)$$

Observed outcomes Y_{it} are realized as:

$$Y_{it} = Y_{it}(0)(1 - Z_{it}) + Y_{it}(1)Z_{it}$$

DiD: Identification Challenge

Estimand: ATT in the post-treatment period

$$\textit{ATT} = \mathbb{E}[Y_{i1}(1) - Y_{i1}(0) \mid \textit{G}_i = 1] = \mathbb{E}[Y_{i1}(1) \mid \textit{G}_i = 1] - \mathbb{E}[Y_{i1}(0) \mid \textit{G}_i = 1]$$

Observed quantities:

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Observed quantities:

	Pre-Period $(t=0)$	Post-Period $(t=1)$
Treatment Group $(G_i = 1)$	$\mathbb{E}[Y_{i0}(0)\mid G_i=1]$	$\mathbb{E}[Y_{i1}(1) \mid G_i = 1]$
Control Group $(G_i = 0)$	$\mathbb{E}[Y_{i0}(0) \mid G_i = 0]$	$\mathbb{E}[Y_{i1}(0) \mid G_i = 0]$

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Control Group $(G_i = 0)$	$\mathbb{E}[Y_{i0}(0) \mid G_i = 0]$	$\mathbb{E}[Y_{i1}(0) \mid G_i = 0]$

Problem: Missing potential outcome $\mathbb{E}[Y_{i1}(0) \mid G_i = 1]$

What would the average post-period outcome for the treated group have been in the absence of treatment?

DiD: Possible Comparisons

Estimand: ATT in the post-treatment period

$$ATT = \mathbb{E}[Y_{i1}(1) - Y_{i1}(0) \mid G_i = 1] = \mathbb{E}[Y_{i1}(1) \mid G_i = 1] - \mathbb{E}[Y_{i1}(0) \mid G_i = 1]$$

Observed quantities:

	Pre-Period $(t=0)$	${\sf Post\text{-}Period}\;(t=1)$
Treatment Group $(G_i = 1)$	$\mathbb{E}[Y_{i0}(0)\mid G_i=1]$	$\mathbb{E}[Y_{i1}(1)\mid G_i=1]$
Control Group $(G_i = 0)$	$\mathbb{E}[Y_{i0}(0) \mid G_i = 0]$	$\mathbb{E}[Y_{i1}(0) \mid G_i = 0]$

Comparison: Treated vs. Control, in Post-Period

- ightharpoonup Use $\mathbb{E}[Y_{i1} \mid G_i = 1] \mathbb{E}[Y_{i0} \mid G_i = 1]$ to estimate ATT
- → Assumes: $\mathbb{E}[Y_{i1}(0) \mid G_i = 1] = \mathbb{E}[Y_{i0}(0) \mid G_i = 1]$

Read: No change in average potential outcome over time

DiD: Possible Comparisons

Estimand: ATT in the post-treatment period

$$ATT = \mathbb{E}[Y_{i1}(1) - Y_{i1}(0) \mid G_i = 1] = \mathbb{E}[Y_{i1}(1) \mid G_i = 1] - \mathbb{E}[Y_{i1}(0) \mid G_i = 1]$$

Observed quantities:

	Pre-Period ($t=0$)	Post-Period $(t=1)$
Treatment Group $(G_i = 1)$	$\mathbb{E}[Y_{i0}(0)\mid G_i=1]$	$\mathbb{E}[Y_{i1}(1) \mid G_i = 1]$
Control Group $(G_i = 0)$	$\mathbb{E}[Y_{i0}(0) \mid G_i = 0]$	$\mathbb{E}[Y_{i1}(0) \mid G_i = 0]$

Comparison: Treated vs. Control, in Post-Period

- → Use $\mathbb{E}[Y_{i1} \mid G_i = 1] \mathbb{E}[Y_{i1} \mid G_i = 0]$ to estimate ATT
- → Assumes: $\mathbb{E}[Y_{i1}(0) \mid G_i = 1] = \mathbb{E}[Y_{i1}(0) \mid G_i = 0]$

Read: Mean ignorability of treatment assignment

DiD: Possible Comparisons

Estimand: ATT in the post-treatment period

$$ATT = \mathbb{E}[Y_{i1}(1) - Y_{i1}(0) \mid G_i = 1] = \mathbb{E}[Y_{i1}(1) \mid G_i = 1] - \mathbb{E}[Y_{i1}(0) \mid G_i = 1]$$

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Control Group $(G_i = 0)$	$\mathbb{E}[Y_{i0}(0) \mid G_i = 0]$	$\mathbb{E}[Y_{i1}(0) \mid G_i = 0]$

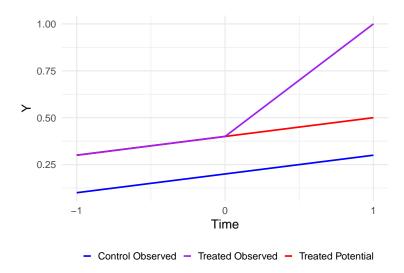
Comparison: Treated vs. Control, in Post-Period

→ Use:
$$[\mathbb{E}[Y_{i1} \mid G_i = 1] - \mathbb{E}[Y_{i1} \mid G_i = 0]] - [\mathbb{E}[Y_{i0} \mid G_i = 1] - \mathbb{E}[Y_{i0} \mid G_i = 0]]$$

→ Assumes:
$$\mathbb{E}[Y_{i1}(0) - Y_{i0}(0) \mid G_i = 1] = \mathbb{E}[Y_{i1}(0) - Y_{i0}(0) \mid G_i = 0]$$

Read: Parallel trends

DiD: Visual Representation of Parallel Trends



Consider two data structures: panel or repeated cross-sectional.

First, let's focus on panel data.

A theoretical data structure:

Unit	Time	Y_{it}	G_i	Z_{it}	X_{it}
1	0	<i>y</i> 1,0	g ₁	<i>z</i> _{1,0}	<i>x</i> _{1,0}
1	1	<i>y</i> 1,1	g_1	$z_{1,1}$	$x_{1,1}$
2	0	<i>y</i> 2,0	g_2	<i>z</i> _{2,0}	<i>X</i> 2,0
2	1	<i>y</i> _{2,1}	g_2	$z_{2,1}$	$x_{2,1}$
:	:	÷	:	÷	:
n	0	<i>y</i> _{n,0}	gn	$z_{n,0}$	$x_{n,0}$
n	1	$y_{n,1}$	g_n	$z_{n,1}$	$x_{n,1}$

Consider two data structures: panel or repeated cross-sectional.

First, let's focus on panel data.

A particular realisation might be:

Unit	Time	Y_{it}	G_i	Z_{it}	X_{it}
1	0	<i>y</i> 1,0	1	0	<i>X</i> _{1,0}
1	1	<i>y</i> _{1,1}	1	1	$x_{1,1}$
2	0	<i>y</i> 2,0	0	0	X2,0
2	1	<i>y</i> _{2,1}	0	0	$x_{2,1}$
:	:	÷	:	:	÷
n	0	<i>y</i> _{n,0}	1	0	$x_{n,0}$
n	1	$y_{n,1}$	1	1	$x_{n,1}$

Now consider the data structure for repeated cross-sections.

A particular realisation might be:

Unit	Time	Y_i	G_i	Z_i	X_i
1	0	<i>y</i> ₁	g ₁	<i>z</i> _{1,0}	<i>x</i> ₁
2	1	<i>y</i> 2	g_2	$z_{2,1}$	<i>x</i> ₂
3	0	<i>y</i> ₃	g 3	<i>z</i> _{3,0}	<i>X</i> 3
4	1	<i>y</i> ₄	g ₄	$z_{4,1}$	<i>x</i> ₄
:	÷	:	:	÷	:
n-1	0	y_{n-1}	g_{n-1}	z_{n-1}	x_{n-1}
n	1	y_n	gn	z_n	Xn

Now consider the data structure for **repeated cross-sections**.

A particular realisation might be:

Unit	Time	Y_i	G_i	Z_i	X_i
1	0	<i>y</i> 1	1	0	<i>x</i> ₁
2	1	<i>y</i> 2	1	1	<i>x</i> ₂
3	0	<i>y</i> 3	0	0	<i>X</i> 3
4	1	<i>y</i> ₄	0	0	<i>x</i> ₄
:	:	:	:	:	:
n-1	0	y_{n-1}	1	0	x_{n-1}
n	1	Уn	1	1	x_n

DiD: Estimation (Repeated Cross-Sections)

Because G_i and T_i are both binary, the same estimator can be calculated via **regression**:

$$\hat{Y}_i = \hat{\mu} + \hat{\gamma}G_i + \hat{\delta}T_i + \hat{\tau}G_iT_i$$

where $\hat{\mu}$, $\hat{\gamma}$, $\hat{\delta}$ and $\hat{\tau}$ are estimated with OLS regression.

It's easy to show that $\hat{\tau} = A\hat{T}T$:

	After $(T_i = 1)$	Before $(T_i = 0)$	After-Before
Treated $(G_i = 1)$	$\hat{\mu} + \hat{\gamma} + \hat{\delta} + \hat{\tau}$	$\hat{\mu} + \hat{\gamma}$	$\hat{\delta}+\hat{ au}$
Control $(G_i = 0)$	$\hat{\mu}+\hat{\delta}$	$\hat{\mu}$	$\hat{\delta}$
Treated-Control	$\hat{\gamma} + \hat{ au}$	$\boldsymbol{\hat{\gamma}}$	$\hat{ au}$

DiD: Estimation (Panel Data)

For panel data, consider an additive linear model for potential outcomes:

$$Y_{it}(z) = \alpha_i + \gamma t + \tau z + \epsilon_{it}$$

where α_i is a time-invariant unobserved parameter for unit *i*.

The **first-differenced regression** of $\Delta Y_i = Y_{i1} - Y_{i0}$ on G_i can unbiasedly estimate ATT = ATE.

Notice that panel data allow for *unit-level* unobserved confounding beyond *group-level* unobserved confounding, but it must be additive and time-invariant.

DiD: Estimation (Panel Data)

What if we have many periods?

Standard for many years was the 'two-way fixed effects' regression:

$$\hat{Y}_{it} = \hat{\alpha}_i + \hat{\gamma}_t + \hat{\tau} Z_{it}$$

Where α_i is a unit fixed effect, γ_t is a time fixed effect, and Z_{it} gives time-varying treatment status.

As before, $\hat{\tau}$ is the DiD estimator of ATT (and ATE if our DGP is right).

DiD: Good Practice

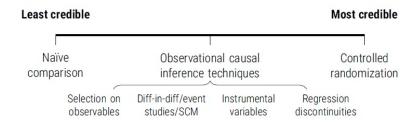
- 1. The key assumption is parallel trends we cannot test this directly. (Why?). Instead, test parallel pre-trends.
- For canonical DiD, use lm() or lm_robust() for cross-sectional, or fixest::feols() for panel.
- 3. We have covered 'canonical 2-period difference-in-differences.' What if you have more that two periods, and treatment is staggered?
- 4. For these cases, look at {fect} and {did} packages in R.



The Lamp Post Problem



Continuum of 'Credibility'TM



The art (and science) of applied causal inference is making defensible assumptions. There is no 'magic' to the FPCI, just assumptions all the way down.

Assumptions All the Way Down



Wrapping Up

Today we covered:

A trio of ways to make causal claims in the absence of randomized *D*:

- 1. Instrumental variables
- 2. Regression discontinuity
- 3. Difference-in-differences

Tomorrow:

- → Intro to machine learning (ML): predicting 'missing values'
- → Performance metrics for ML
- → Doing prediction with regression and naive Bayes