Causal methods with observational data

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Roadmap

Intro and overview

Methods in brief

Re-cap and essay guidelines

Causal methods in detail
- Controlling and matching
- Fixed effects
- Difference-in-differences
- Regression discontinuity
- Instrumental variables

Re-cap

1. Problem/topic
2. Stories, arguments about mechanisms
3. Research question
4. Proper theory, concepts and operationalization
5. Measurement, unit of analyses, data sources, etc
6. Inference strategy
7. Results & interpretation

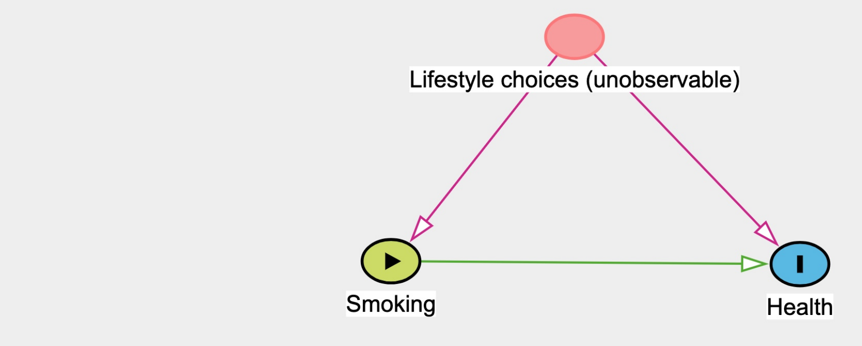
Methods and causal inference

- Most of the time is **impossible** to control for all relevant variables (i.e. not able to close all back-door paths)
- So what do we do? We try to find ways to control for unobserved confounding
- One option is to rely on additional controlling techniques
  - → Matching: also depends on observables, but parametric advantages, etc (anyway, not a solution for U)
  - → Fixed effects: can control for group-level unobservables, and correct issues related to ecological fallacy
- But there are other methods often related to causal inference, not because they uncover causal relationships, but because they allow you to exploit 'typical' exogenous sources of variation
- (In H-K's *The Effect*, they're called 'template causal diagrams')

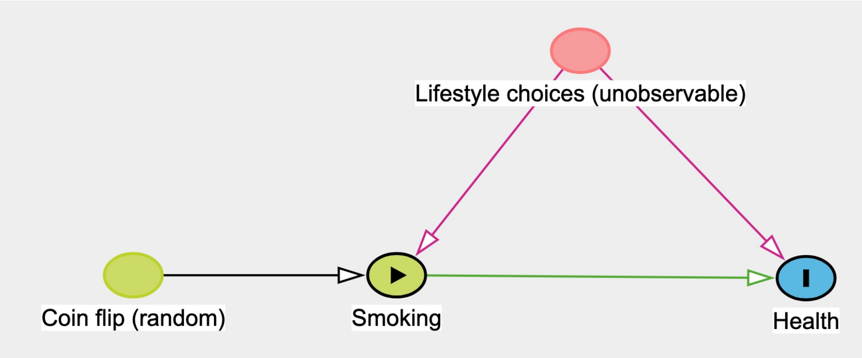
Methods and causal inference

- Another option is to find a source of exogenous variation and exploit
- There are a few methods related to causal inference not because they uncover causal relationships but because they are designed to exploit *'typical' exogenous sources of variation*
- Situations where there is *something* that introduces variation in the treatment that is independent from confounders and you can exploit to analyse
  - → Same as randomization (of treatment assignment) in experiments
- (In H-K's *The Effect*, they're called 'template causal diagrams')

Methods and causal inference



Methods and causal inference



Methods and causal inference

1. Time
   - → we can exploit changes over time in treated vs control units, e.g. imagine checking effect of good nutrition on a child who is growing anyway
2. Cut-offs
   - → sometimes something happens just when you cross a cut-off (getting into university, winning an election, a geographical border, being born Jan 1st...)
3. A third, unrelated variable
   - → you win the lottery, you get a sudden increase in disposable income

Methods and causal inference

- Five techniques commonly used in causal inference
  - → two of them use for controlling (closing back-doors), and the other three to exploit 'pre-made' causal models
  - → btw, what is controlling? when you control for Z, you remove the variation in X and Y that is *explained by Z* (see this)

Methods and causal inference

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1. Fixed effects
2. Difference-in-differences
3. Regression discontinuity design
4. Instrumental variables
5. Matching

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Difference-in-differences

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Regression discontinuity design

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Instrumental variables

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Matching

Matching

Overall inference strategy

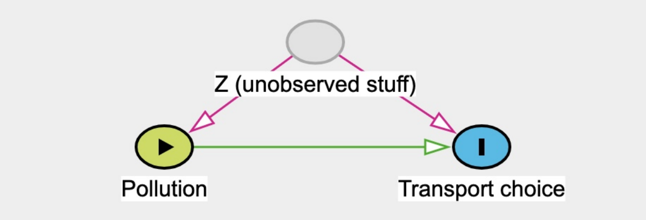
- In ideal experimental setting, we actually don't need any sophisticated statistics
  - → We can just compare the *mean* of the treatment group and the *mean* of the control group, that's it

Overall inference strategy

- In ideal experimental setting, we actually don't need any sophisticated statistics
  - $\rightarrow$ We can just compare the *mean* of the treatment group and the *mean* of the control group, that's it
- In observational data, these two types of tools (controlling + exploiting exogenous variation) are often used in combination
- But **always remember** these methods **depend** on a **causal model**
- Let's look at an example (from Huntington-Klein *The Effect*, kind of)

Controlling **and** exploting exogeneity

- Q: **Does pollution determine transport choice?**
- And say we are going to observe variation across *days*
  - → Outcome: car driving
  - → Treatment: pollution levels
- Clear problem of endogeneity, no? (driving in t_{-1}, economic activity, etc)



Controlling **and** exploting exogeneity

Chasing Clean Air: Pollution-Induced Travels in China

Shuai Chen, Yuyu Chen, Ziteng Lei, Jie-Sheng Tan-Soo

Abstract: This study uses "big" data to empirically investigate a highly effective, but underexamined way of reducing one's exposure toward air pollution—short-term travel. We determine subscribers' locations using mobile phones' signals and thereby establish linkages between air pollution and short-term population movements between cities in China. Using an instrumental variable based on daily variation in wind directions and pollution levels from distant upwind locations, we find that a one-unit increase in the origin city's air quality index (AQI) over the destination city's AQI increases short-term population flow from origin to destination by 0.15%. Further analyses provide richer characterizations of the decision-making process behind travel movements. Our findings add to the evidence base by examining in detail an understudied behavioral response toward air pollution.

JEL Codes: O15, Q53, Q56

Keywords: air pollution, short-term population movement, avoidance cost, averting

Controlling **and** exploting exogeneity

- But it's not enough with wind direction, no?

Controlling **and** exploting exogeneity

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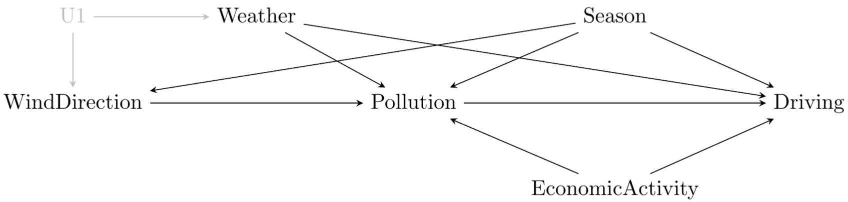

(And city, in this case)

Controlling **and** exploting exogeneity

- So in this case, we need to do two things:

1. Exploit exogenous variation
   - → In this case, using an instrumental variables approach, which isolates the variation in pollution which is explained by variation in wind direction

Controlling **and** exploting exogeneity

- So in this case, we need to do two things:

1. Exploit exogenous variation
   → In this case, using an instrumental variables approach, which isolates the variation in pollution which is explained by variation in wind direction
2. *And* control for weather, season, and city
   → Using a regression with control variables, or matching, or fixed effects

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Re-cap and final essay

- **Groups?** Send me an email

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Controlling in regression

- We probably already know this
- We've seen what it means controlling: just adjusting for the variation already predicted by the other variables
- One limitation though: we need to *observe* those variables

Controlling

- We are going to see two further methods of controlling widely used:
  - → Matching
  - → Fixed effects

Matching

- Adding control variables is not the only way to control / close back doors
- Imagine we have the following model:
  - $\rightarrow$ Z → X → Y ← Z
  - $\rightarrow$ Where Z is whether someone is retired or not
- If we select a sample of *only* retired people, we are closing that back door
  - $\rightarrow$ (X ← Z → Y)
- Matching is something like this, it's basically about creating groups of comparison where Z (which can be several variables) does not vary

How matching works

- We have a treatment group and a control group (so: *binary* treatment variables)
- The main idea: give different *weights* to treated and control observations, so we eliminate
- We get these weights by using one or more *matching variables* (i.e. confounding variables)

How matching works

- Imagine we have:
  - → Treatment: get some specific skill training
  - → Outcome: jet a job afterwards
  - → Confounding variable: gender
- Control group: 80 men and 20 women
  - → 75% of men get a job, 60% of women do
- Treatment group: 500 men and 500 women
  - → 70% of men get a job, 55% of women do
- Comparing within each group, we know the treatment effect is a 5% increase in the odds of getting a job (70 → 75, 55 → 60)
- But if we do the global comparison, it's almost 10%
  - → 60 men + 12 women out of 100 = 72/100 = 72%, vs (350 + 275)/1000 = 62.5%, so a difference of 9.5 points

How matching works

- The problem is we have 4 times more men in the treated group than women, whereas we have equal proportion on the control group
- So we'll weight the control observations by gender, giving *more* weight to the men observations, so it looks more similar to the treatment group
- $(4 * 350 + 1 * 275)/(4 * 500 + 1 * 500) = 67\%$
- Now, the unweighted difference in the treatment group was 72%, and now the weighted difference in the control group is 67%
- The difference is 5 points, the same as the within-group calculation we did before

Two approaches to matching

1. **Distance matching**
   - → We want to create a dataset where treatment and control observations have similar values (distance) in the confounding variables
   - → If, say, our confounding variable is income, we'll pick control observations that have a similar value on income to each of the treatment observations
   - → https://nickchk.com/causalgraphs.html

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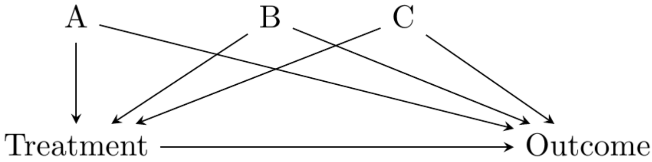

Two approaches to matching

2. **Propensity score matching**
   - → We want to account for the differential likelihood in getting into treatment depending on the value of the confounding variables
   - → We estimate the probability of getting into treatment, usually by doing a regression where the outcome is the treatment and the right-hand variables are the confounders
   - → We control for the propensity score matching, or select based on it (or both)

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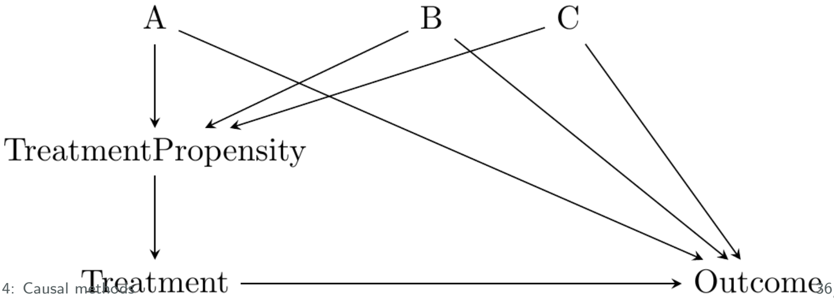


Two approaches to matching: propensity score

```r
df = data.frame(
  treat = c(rep(1, 100), rep(0, 1000)),
  gender = c(rep("M", 80), rep("F", 20), rep(c("M","F"), each = 500)),
  y = NA
)

df$y[df$treat == 1 & df$gender == "M"] = rbinom(80, 1, 0.75)
df$y[df$treat == 1 & df$gender == "F"] = rbinom(20, 1, 0.6)
df$y[df$treat == 0 & df$gender == "M"] = rbinom(500, 1, 0.7)
df$y[df$treat == 0 & df$gender == "F"] = rbinom(500, 1, 0.55)

m1 = glm(y ~ treat, data = df)
m2 = glm(y ~ treat + gender, data = df)
modelsummary(list(m1, m2))

ps = glm(treat ~ gender, data = df)
df$propensity_score = predict(ps, newdata = df)
m3 = glm(y ~ treat + propensity_score, data = df)
modelsummary(list(m1, m2, m3))
```

Two approaches to matching: propensity score

	Model 1	Model 2	Model 3
(Intercept)	0.623	0.550	0.493
	(0.015)	(0.021)	(0.030)
treat	0.007	−0.037	−0.037
	(0.051)	(0.051)	(0.051)
genderM		0.147	
		(0.029)	
propensity_score			1.478
			(0.296)
Num.Obs.	1100	1100	1100

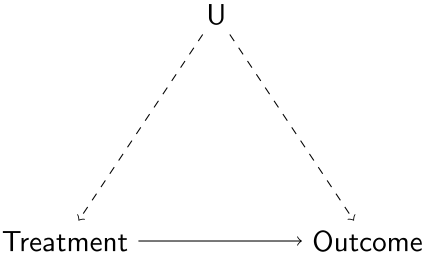
Matching vs regression

- Matching and regression are complementary approaches
- e.g. regression doesn't waste any information, but has a linearity assumption
- It's usual to use both at the same time

Fixed effects

- The problem with covariate adjustment, regardless of whether we use regression or matching, is that we need to *observe* those variables
- But another strategy when we have unobserved confounders is to try within-group comparisons, which will work when the unobserved variance is contact within some group
- For example, imagine cases when our U variable is:
  → *Country history*, in a cross-national analysis
  → *City of origin*, in an individual-level analysis
  → *Individual background*, in a panel survey analysis
  → *Company effects*, if we look at the effects of English courses on internal promotion using individual data from many different companies
  → etc

Fixed effects - when do we use them?



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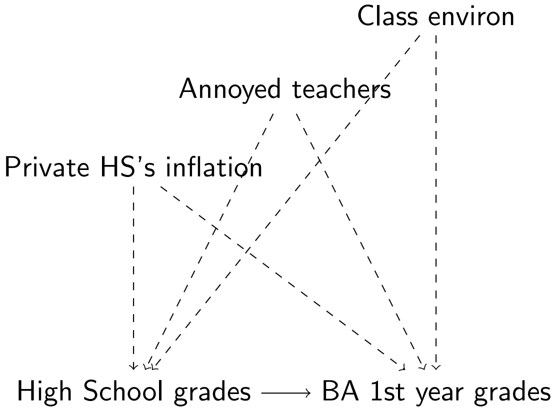

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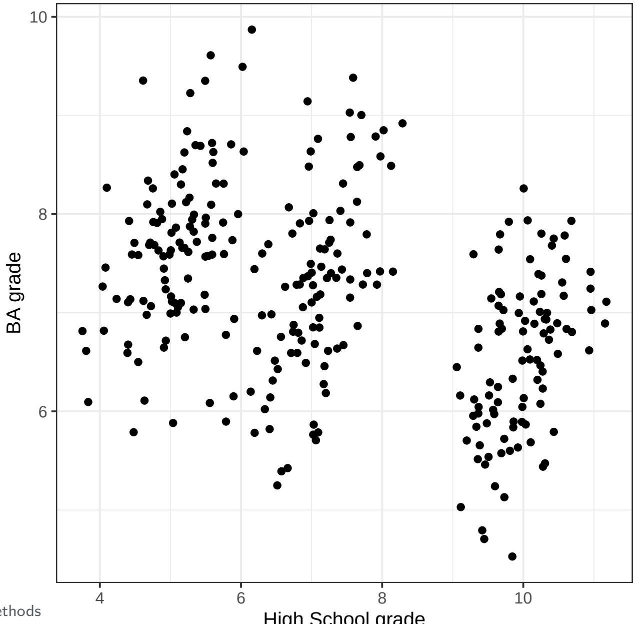

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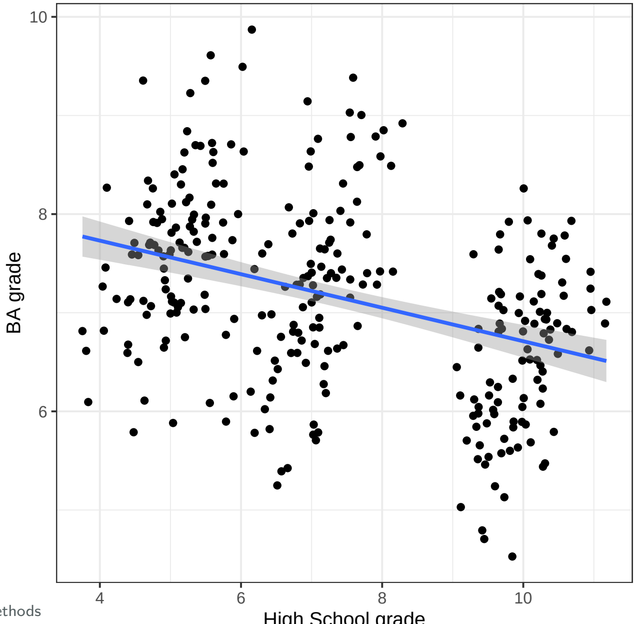

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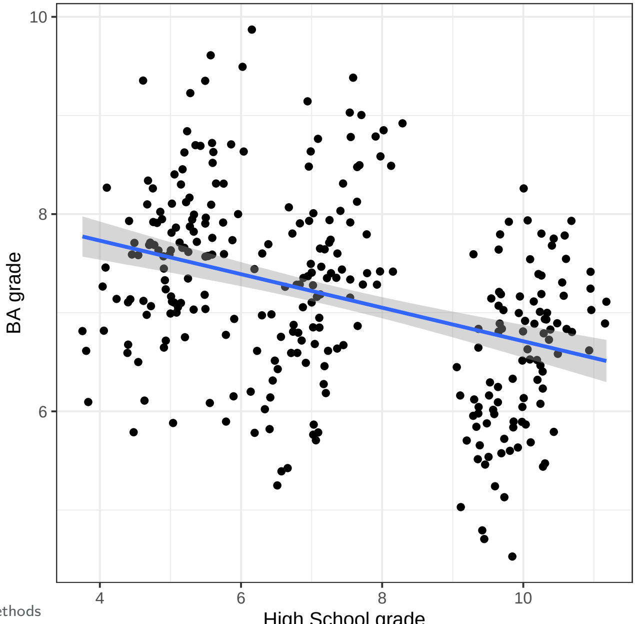

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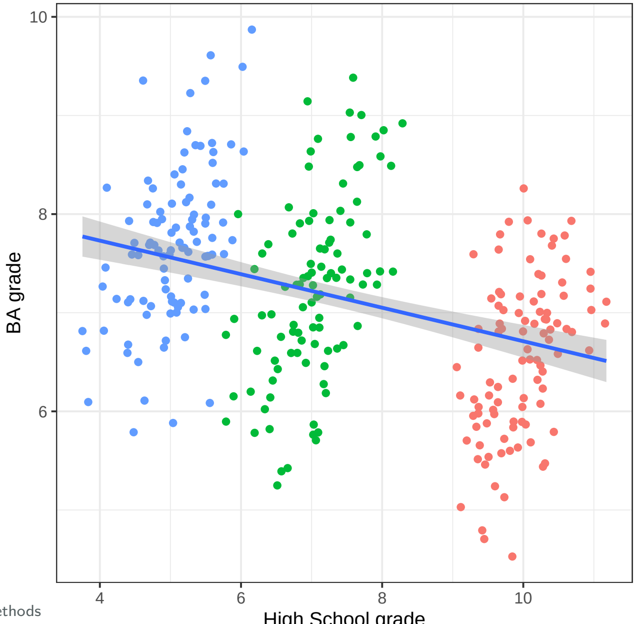

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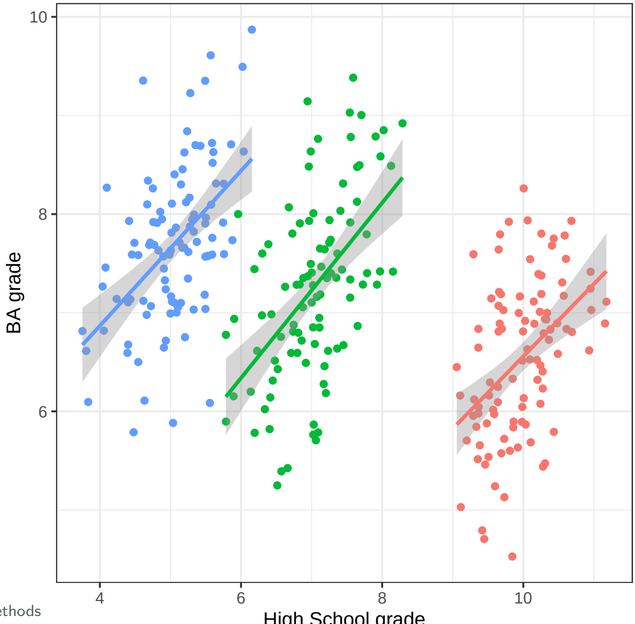

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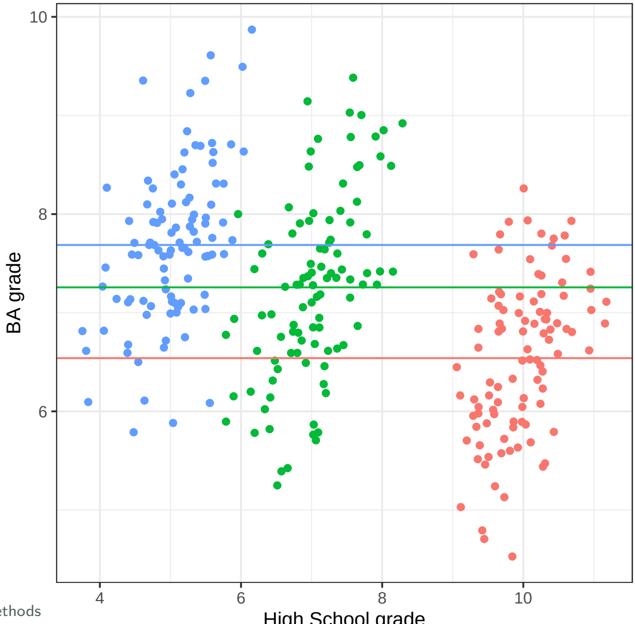

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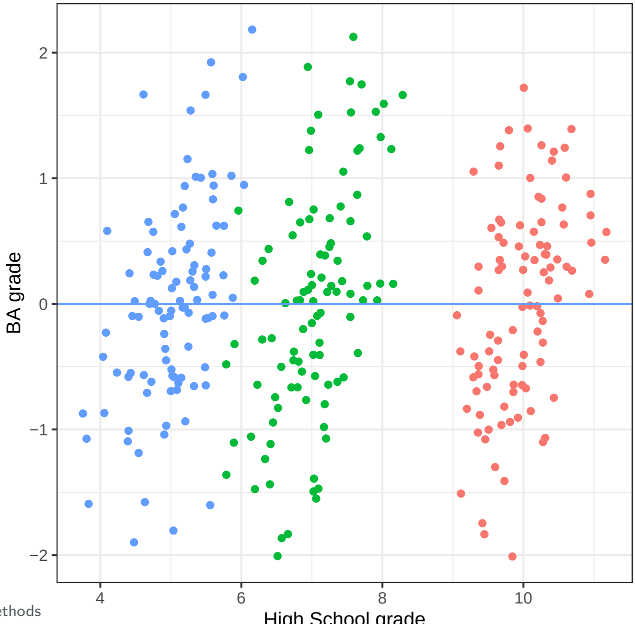

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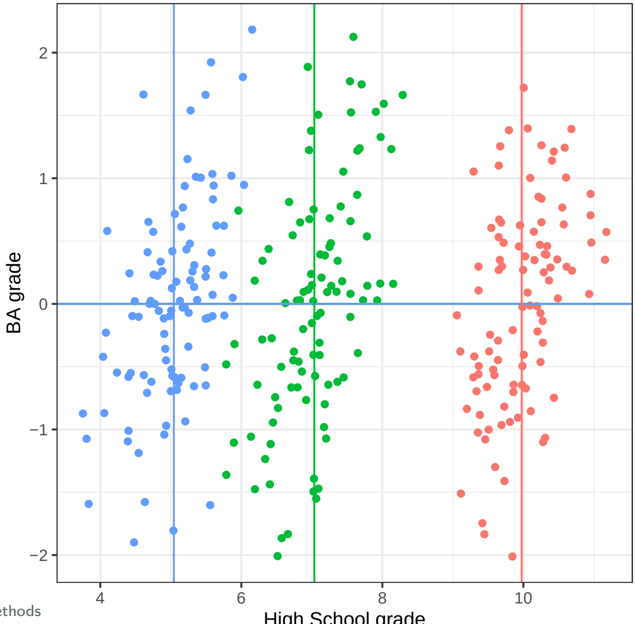

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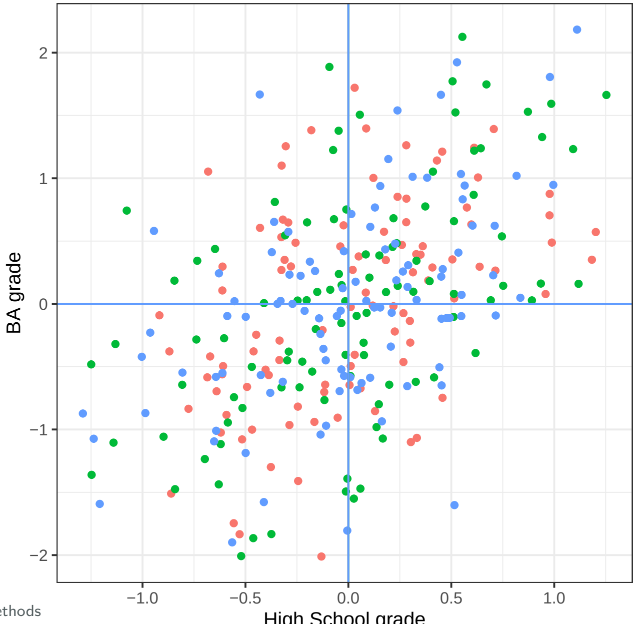

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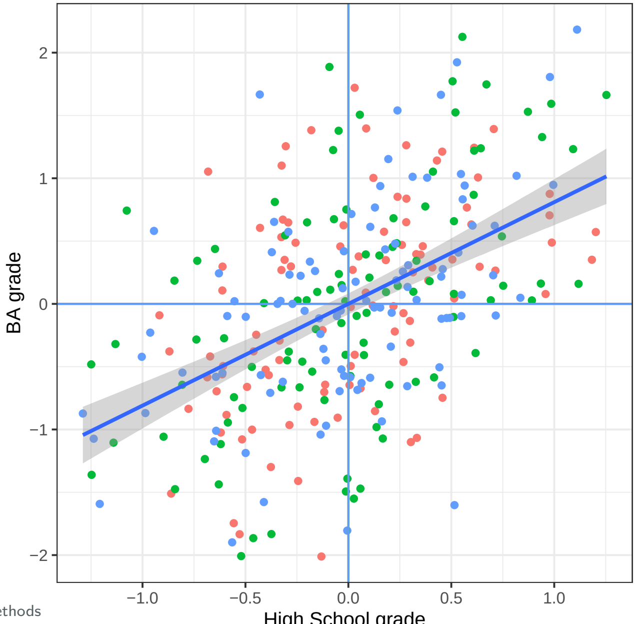


Fixed effects and regression

```r
# Simulate data: 300 students from three high schools,
# normally distributed grades
df = data.frame(high_school = rep(c("A", "B", "C"), each = 100)) %>%
  group_by(high_school) %>%
  mutate(hs_grade = rnorm(100, 7, 0.5)) %>%
  ungroup()

# But school A inflated grades a lot, and C was notoriously difficult
df$hs_grade[df$high_school == "A"] = df$hs_grade[df$high_school == "A"] + 3
df$hs_grade[df$high_school == "C"] = df$hs_grade[df$high_school == "C"] - 2

# First-year BA grades are a function of the HS grade +/- the school inflation
df$ba_grade = 2 + 0.75 * df$hs_grade + rnorm(300, 0, 0.75)
df$ba_grade[df$high_school == "A"] = df$ba_grade[df$high_school == "A"] - 3
df$ba_grade[df$high_school == "C"] = df$ba_grade[df$high_school == "C"] + 2
```

Our true causal model

Fixed effects and regression

```
Call:
lm(formula = ba_grade ~ hs_grade, data = df)

Residuals:
     Min       1Q   Median       3Q      Max
-2.42897 -0.57050 -0.05923  0.66227  2.76385

Coefficients:
            Estimate Std. Error t value             Pr(>|t|)
(Intercept)  8.70808    0.19412   44.86 < 0.0000000000000002 ***
hs_grade    -0.22059    0.02544   -8.67 0.000000000000000283 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error: 0.9061 on 298 degrees of freedom
Multiple R-squared:  0.2014,  Adjusted R-squared:  0.1988
F-statistic: 75.17 on 1 and 298 DF,  p-value: 0.0000000000000002826
```

Fixed effects and regression

```
Call:
lm(formula = ba_grade ~ hs_grade + factor(high_school), data = df)

Residuals:
    Min      1Q  Median      3Q     Max 
-2.0124 -0.5464  0.0042  0.4984  2.2539 

Coefficients:
                     Estimate Std. Error t value             Pr(>|t|)    
(Intercept)          -1.68390    0.98549  -1.709               0.0886 .  
hs_grade              0.80803    0.09871   8.186  0.00000000000000811 ***
factor(high_school)B  3.23797    0.30984  10.451 < 0.0000000000000002 ***
factor(high_school)C  5.26454    0.49388  10.659 < 0.0000000000000002 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error: 0.7719 on 296 degrees of freedom
Multiple R-squared:  0.4243,  Adjusted R-squared:  0.4185 
F-statistic: 72.73 on 3 and 296 DF,  p-value: < 0.00000000000000022
```

Difference-in-differences

- Treatments usually occur at a particular moment in time, e.g.:
  - → Minimum wage increase
  - → Terrorist attack
  - → Influx of refugees
  - → ...
- In those cases, if we have before & after observations, we have something like this:

$$\begin{tikzcd} & \text{Time} \arrow[dl] \arrow[dr] & \\ \text{Treatment} \arrow[rr] & & \text{Outcome} \end{tikzcd}$$

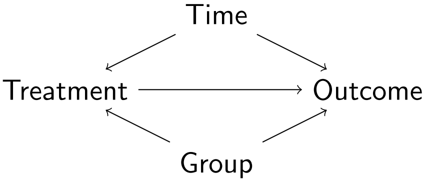
- The **problem** is that **all** the variation goes through time, so if we close that back door, we're left with nothing

Difference-in-differences

- So one strategy we can use is to bring additional group that is *not treated* and for which we also have before/after observations
  - → Minimum wage increase: maybe those earning above MW?
  - → Influx of refugees: other countries? regions far from the border?
  - → Terrorist attack: do we have a control (untreated) group?
  - → ...

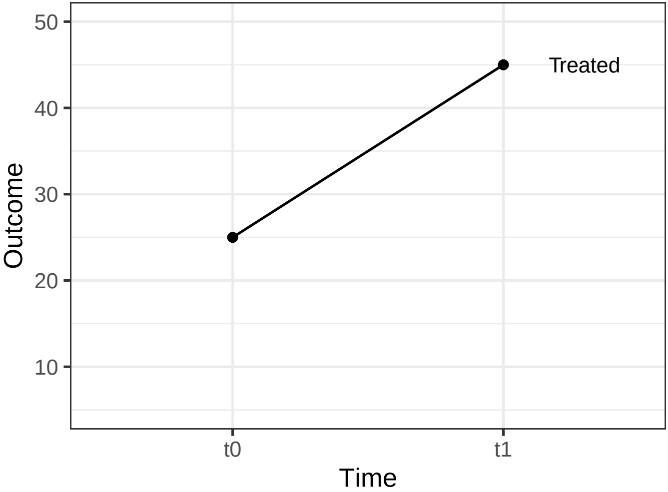
$$
\begin{tikzcd}
 & \text{Time} \arrow[dl] \arrow[dr] & \\
\text{Treatment} \arrow[rr] & & \text{Outcome} \\
 & \text{Group} \arrow[ul] \arrow[ur] &
\end{tikzcd}
$$

Difference-in-differences



- We can compare changes across time *within* the treated and control groups (closing the back door through `group`)
- Compare within-group variation between treated and control (since time affects both 'within-variations' the same way, we are closign the other back door through `time`)

Difference-in-differences



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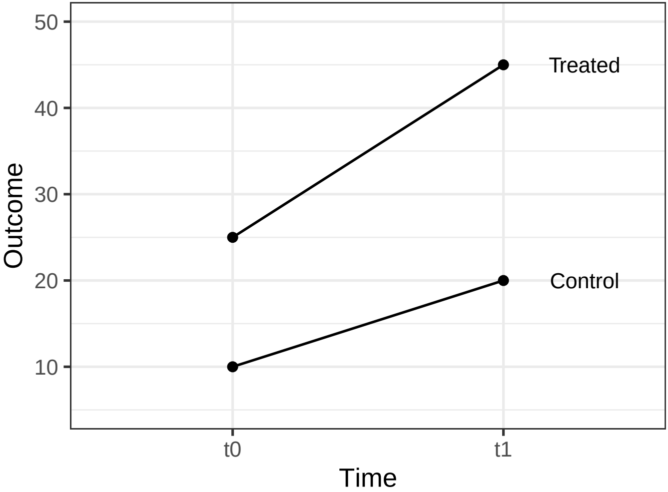

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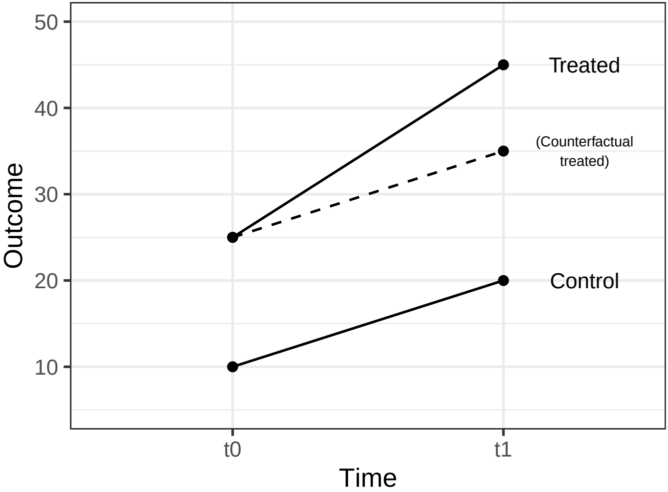

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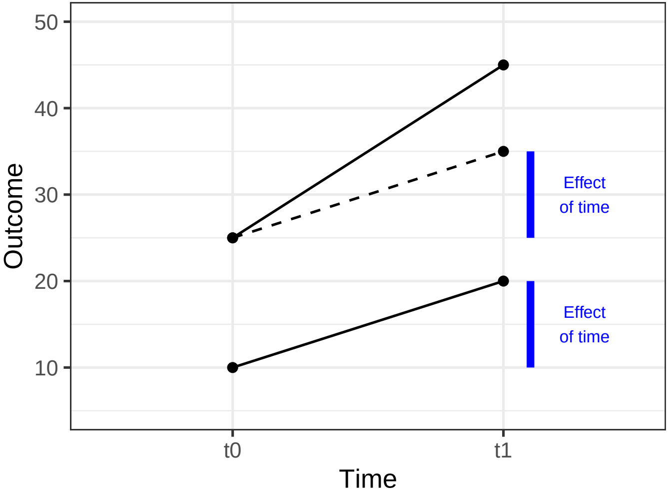

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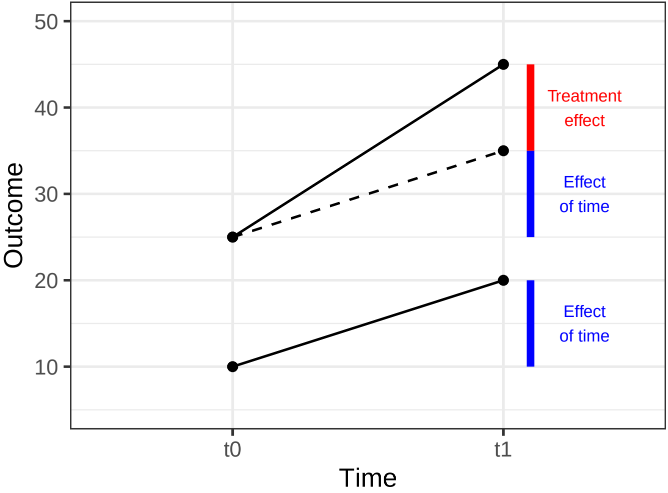


Difference-in-differences: Cholera in London

- Probably first use of DiD and natural experiments?
- Lambeth Company moved water intake upriver in 1952, Southwark & Vauxhall Company still got it from downstream

Difference-in-differences: Cholera in London

2261	2458	Southwk. & Vauxhall.
3905	2547	Both Companies.
162	37	Lambeth Company.

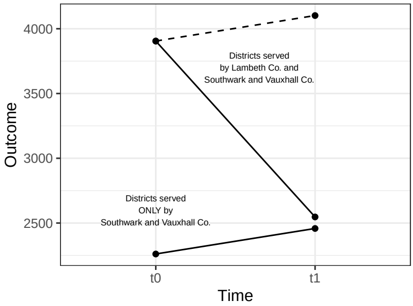


TABLE XII.

Sub-Districts.	Deaths from Cholera in 1849.	Deaths from Cholera in 1854.	Water Supply.
St. Saviour, Southwark	283	371	Southwark & Vauxhall Company only.
St. Olave	157	161	
St. John, Horsleydown	192	148	
St. James, Bermondsey	249	362	
St. Mary Magdalen	259	244	
Leather Market	226	237	
Rotherhithe*	352	282	
Wandsworth	97	59	
Battersea	111	171	
Putney	8	9	
Camberwell	235	240	
Peckham	92	174	
Christchurch, Southwark	256	113	Lambeth Company, and Southwark and Vauxhall Compy.
Kent Road	267	174	
Borough Road	312	270	
London Road	257	93	
Trinity, Newington	318	210	
St. Peter, Walworth	446	388	
St. Mary, Newington	143	92	
Waterloo Road (1st)	193	58	
Waterloo Road (2nd)	243	117	
Lambeth Church (1st)	215	49	
Lambeth Church (2nd)	544	193	
Kennington (1st)	187	303	
Kennington (2nd)	153	142	
Brixton	81	48	
Clapham	114	165	
St. George, Camberwell	176	132	
Norwood	2	10	Lambeth Company only.
Streatham	154	15	
Dulwich	1	—	
Sydenham	5	12	
First 12 sub-districts	2261	2458	Southwk. & Vauxhall.
Next 16 sub-districts	3905	2547	Both Companies.

Difference-in-differences

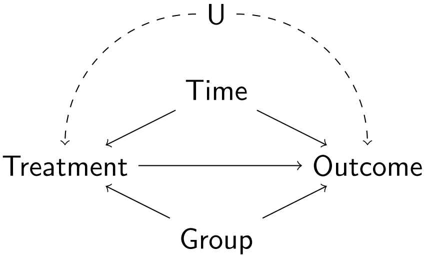
- You can estimate this effect just with group means
- But it is often easier to use regression, also because you can include controls:

$$\begin{aligned} Y_{it} =& \beta_0 + \beta_1 Treated_i + \beta_2 After_t + \\ & \beta_3 (Treated_i \times After_t) + \beta^\top x_i + \epsilon_{it} \end{aligned} \tag{1}$$

- But why do we need all this?

Difference-in-differences

- Because DiD identification **depends** on the assumption that the **control group is a good counterfactual to the treated group**

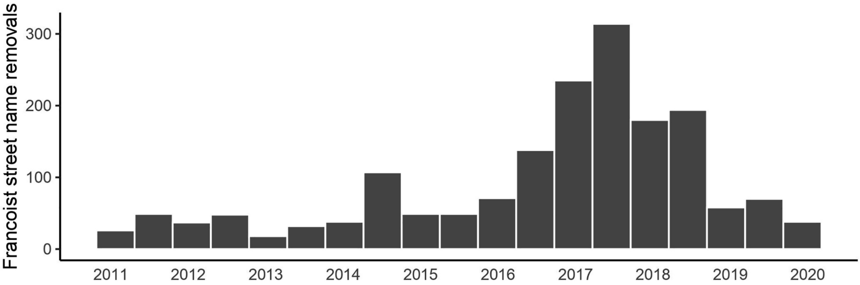


- One way to test this is checking if the **parallel trends assumption** holds (we need data further back in time)

DiD example

- What is the effect of symbolic TJ policies?
- journals.sagepub.com/doi/full/10.1177/20531680211058550

DiD example



Francoist street name removals
300
200
100
0
2011
2012
2013
2014
2015
2016
2017
2018
2019
2020

DiD example

Francoist names	**Removed Francoist names, 2016–2018?**	
In June 2016?	**No**	**Yes**
No	6455	0
	(100%)	(0%)
Yes	1184	454
	(72%)	(28%)

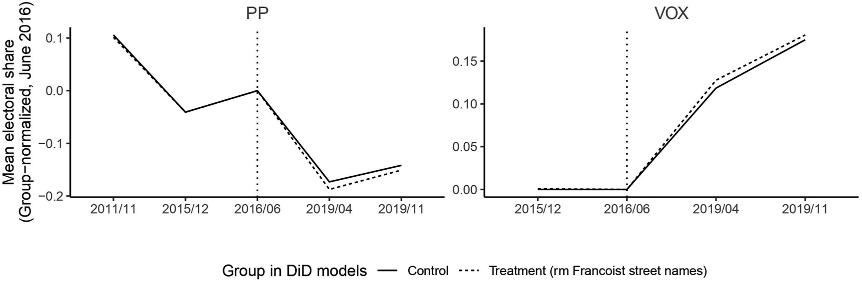
Note: Row percentages. Changes in 2016–2018 refer to the period between 30/06/2 016 and 31/12/2 018.

DiD example

Table 2. Mean electoral share in sample.

	June 2016			**April 2019**			
Party	***Control***	***Treated***	**Δ**	***Control***	***Treated***	**Δ**	$\Delta_{2019}-\Delta_{2016}$
Vox	0.21	0.21	0	12.54	13.28	0.74	0.74
PP	41.22	46.77	5.55	23.83	27.68	3.85	−1.7
PSOE	29.13	28.01	−1.12	33.38	32.03	−1.35	−0.23

DiD example


PP
VOX
Mean electoral share
(Group−normalized, June 2016)
0.1
0.0
−0.1
−0.2
2011/11
2015/12
2016/06
2019/04
2019/11
0.15
0.10
0.05
0.00
Group in DiD models
Control
Treatment (rm Francoist street names)

Regression discontinuity

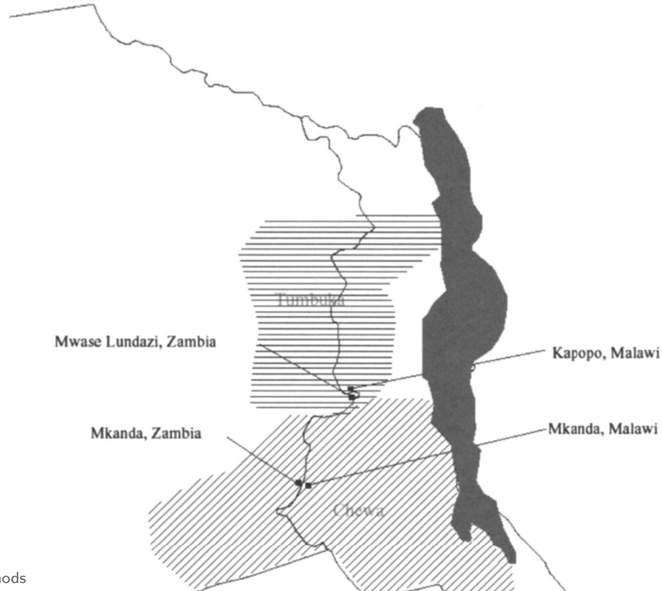
The Political Salience of Cultural Difference: Why Chewas and Tumbukas Are Allies in Zambia and Adversaries in Malawi

DANIEL N. POSNER *University of California, Los Angeles*

This paper explores the conditions under which cultural cleavages become politically salient. It does so by taking advantage of the natural experiment afforded by the division of the Chewa and Tumbuka peoples by the border between Zambia and Malawi. I document that, while the objective cultural differences between Chewas and Tumbukas on both sides of the border are identical, the political salience of the division between these communities is altogether different. I argue that this difference stems from the different sizes of the Chewa and Tumbuka communities in each country relative to each country's national political arena. In Malawi, Chewas and Tumbukas are each large groups vis-à-vis the country as a whole and, thus, serve as viable bases for political coalition-building. In Zambia, Chewas and Tumbukas are small relative to the country as a whole and, thus, not useful to mobilize as bases of political support. The analysis suggests that the political salience of a cultural cleavage depends not on the nature of the cleavage itself (since it is identical in both countries) but on the sizes of the groups it defines and whether or not they will be useful vehicles for political competition.

Regression discontinuity

FIGURE 1. Research Sites

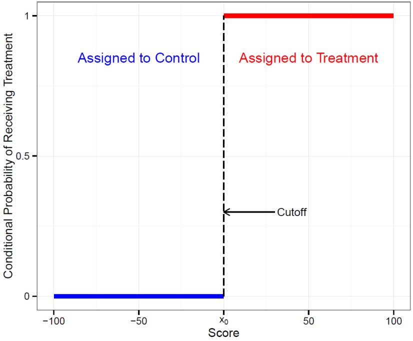


Regression discontinuity

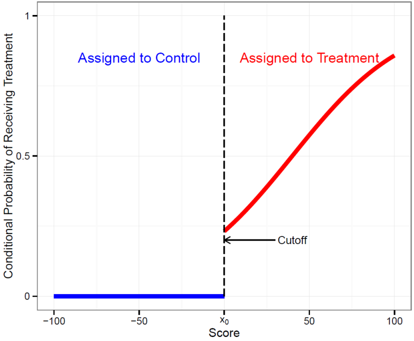
- RDD works well when assignment into treatment depends on a cutoff along a **running variable**
  - → Do incumbent politicians have an electoral advantage? (vote sahre)
  - → What is the effect of being drafted into the military? (birth year)
  - → Effect of national policies in ethnic identification in Africa? (distance to colonial borders)
- This is the source of the exogenous variation (or if you will, the natural experiment):
  - → Although many variables confound the relationships between X and Y, nothing should be too different *around the cutoff* between treatment and control groups (local randomization assumption)
  - → Sometimes you look at different *bandwidths* to check this

RDD

Figure 1: Conditional Probability of Receiving Treatment in Sharp vs. Fuzzy RD Designs



(a) Sharp RD

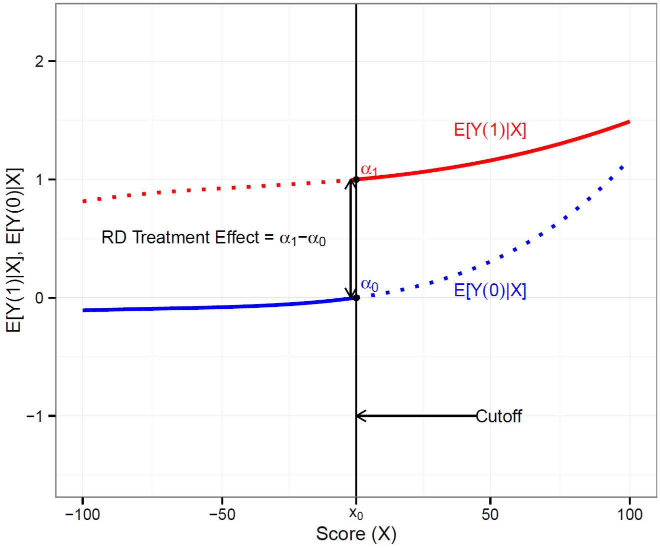


(b) Fuzzy RD (One-Sided)

https://bookdown.org/paul/applied-causal-analysis/rddbasics2.html

RDD

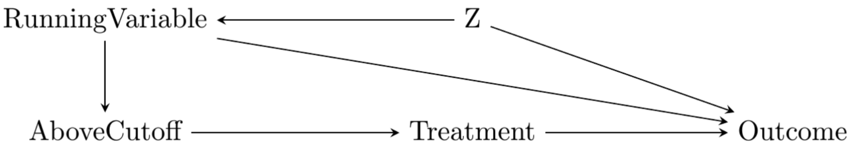
Figure 2: RD Treatment Effect in Sharp RD Design



https://bookdown.org/paul/applied-causal-analysis/rddbasics3.html

RDD

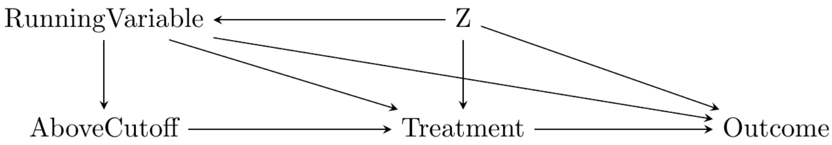
- Underlying assumption: other confounders also vary along the running variable, but are independent to the *jump*



Huntington-Klein, *The Effect*, p.508

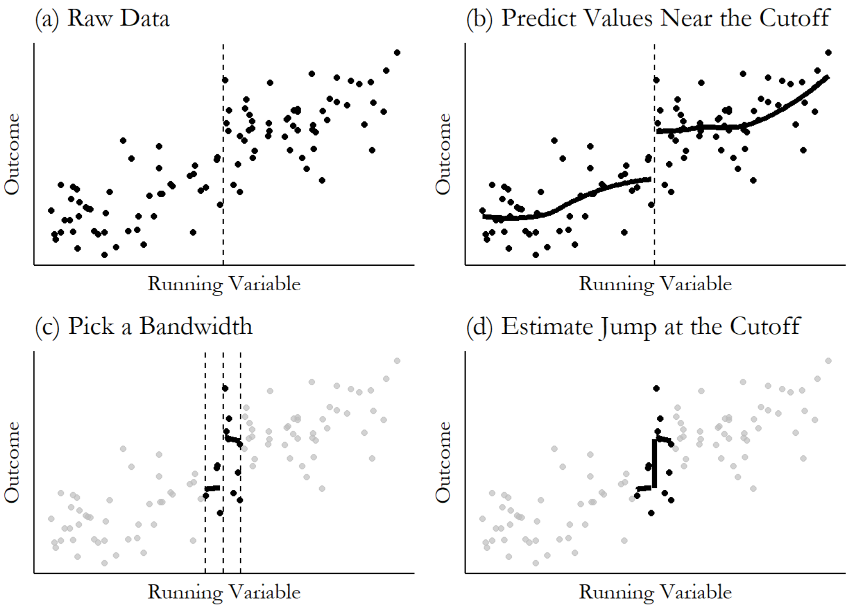
RDD

- Underlying assumption: other confounders also vary along the running variable, but are independent to the *jump*
- Even if in a *fuzzy* design



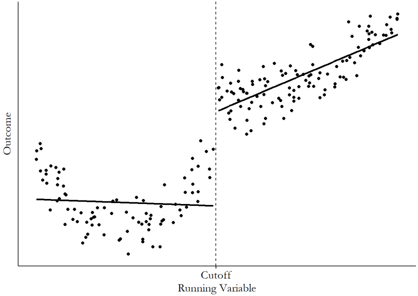
Huntington-Klein, *The Effect*, p.508

RDD implementation



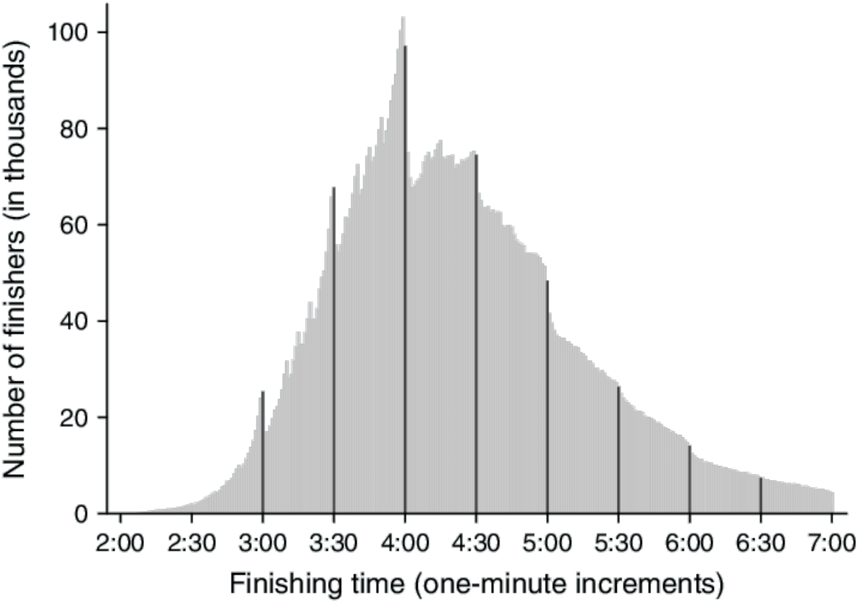
RDD and regression

$$
\begin{aligned}
Y = &\beta_0 + \beta_1 \mathit{Distance} + \\
&\beta_2 \mathit{Treated} + \\
&\beta_3(\mathit{Treated} \times \mathit{Distance}) + \beta^\top x_i
\end{aligned} \tag{2}
$$

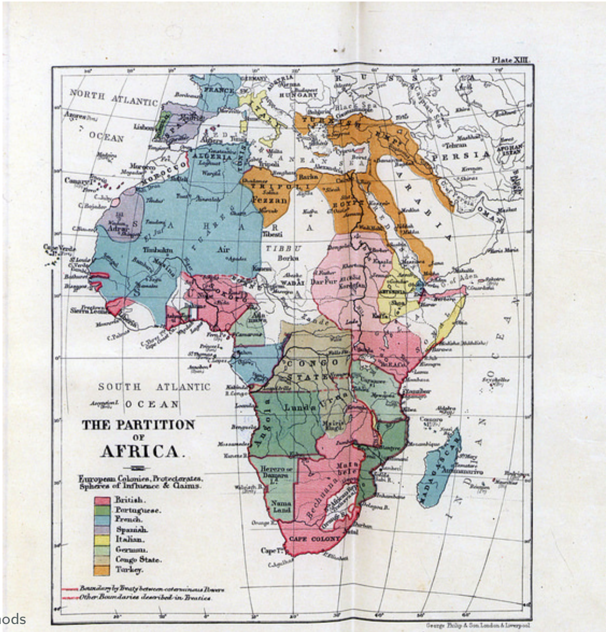


- But there's no need to use linear regression, other methods available as well

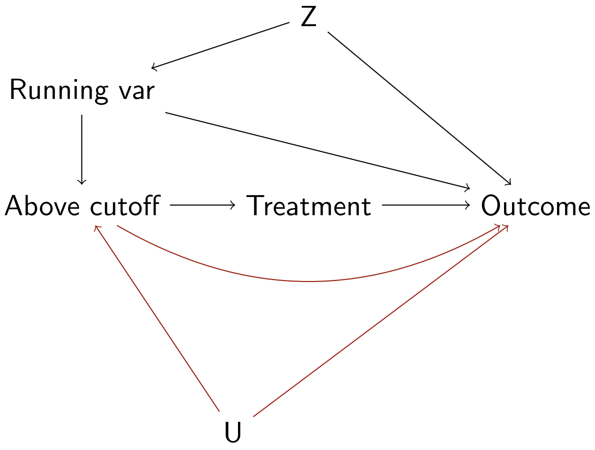
Threats to RDD: precise sorting



Threats to RDD: cutoff ← outcome



Threats to RDD



Some extensions & combinations

- DiD with multiple treatment periods (units being treated at different times)
- Matched DiD
- Difference-in-discontinuities

Instrumental variables

$$
\begin{tikzcd}
& & \mathsf{U} \arrow[dl, dashed] \arrow[dr, dashed] & \\
\texttt{random} \arrow[r] & \mathsf{X} \arrow[rr] & & \mathsf{Y}
\end{tikzcd}
$$

Instrumental variables

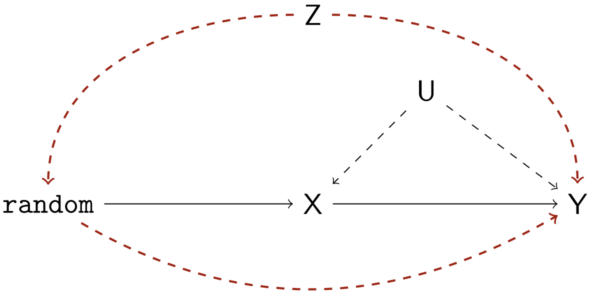
- Find an exogenous source of variation in the treatment variable
- Isolate that variation and use it to identify the causal effect

Instrumental variables

- Find an exogenous source of variation in the treatment variable
- Isolate that variation and use it to identify the causal effect

- Assumptions:
- **Relevance**: the instrument explains at least some part of the treatment variable
- **Validity** or **exclusion restriction**: no back door paths between the instrument and the outcome

Instrumental variables



IV threats

Economic Shocks and Civil Conflict: An Instrumental Variables Approach

Edward Miguel

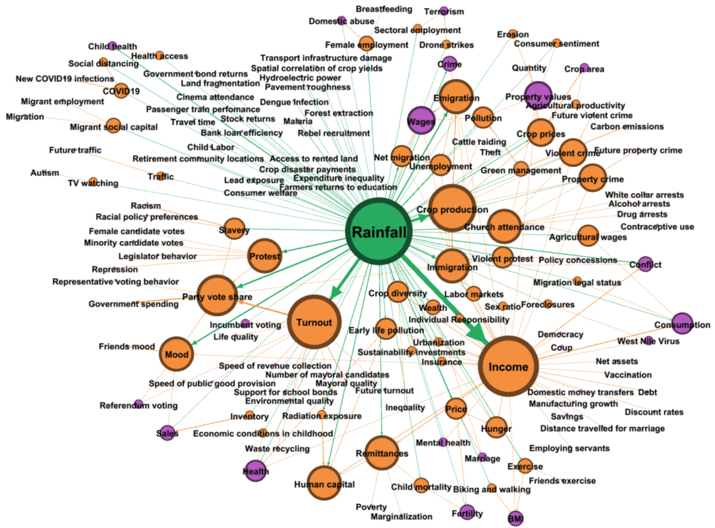
University of California, Berkeley and National Bureau of Economic Research

Shanker Satyanath and Ernest Sergenti

New York University

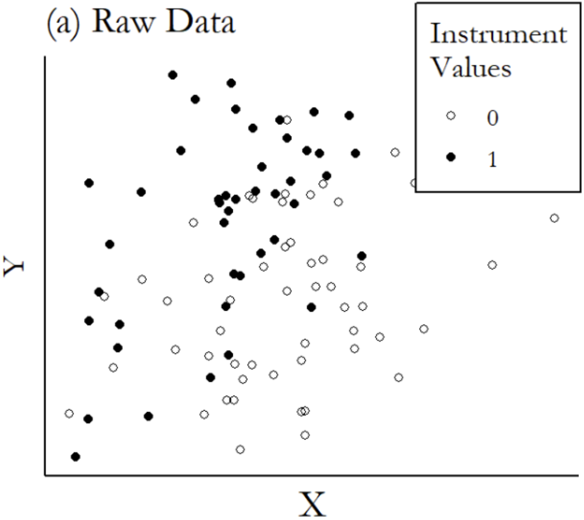
Estimating the impact of economic conditions on the likelihood of civil conflict is difficult because of endogeneity and omitted variable bias. We use rainfall variation as an instrumental variable for economic growth in 41 African countries during 1981–99. Growth is strongly negatively related to civil conflict: a negative growth shock of five percentage points increases the likelihood of conflict by one-half the following year. We attempt to rule out other channels through which rainfall may affect conflict. Surprisingly, the impact of growth shocks

IV threats



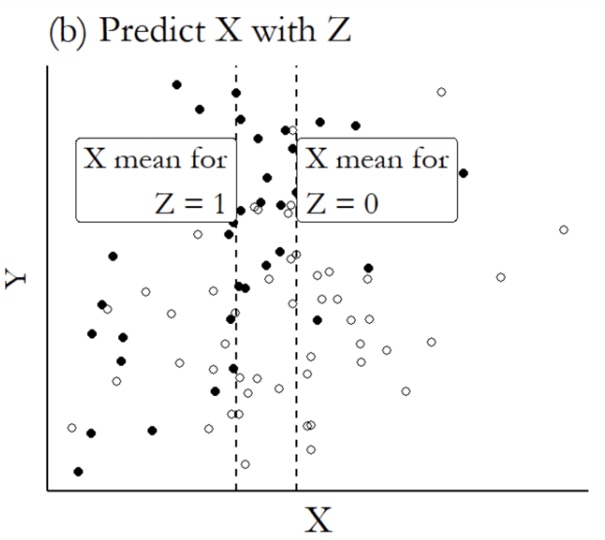
Jonathan Mellon (2022) Rain, Rain, Go Away: 192 Potential Exclusion-Restriction Violations for Studies Using Weather as an Instrumental Variable

How does IV work?



(Huntington-Klein, *The Effect*, p 472)

How does IV work?



(Huntington-Klein, *The Effect*, p 472)

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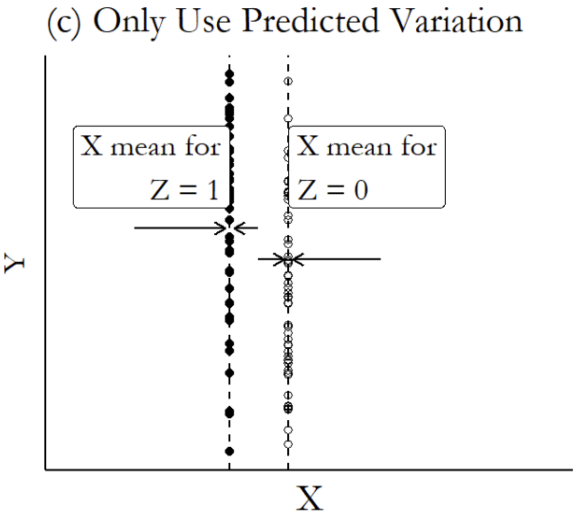
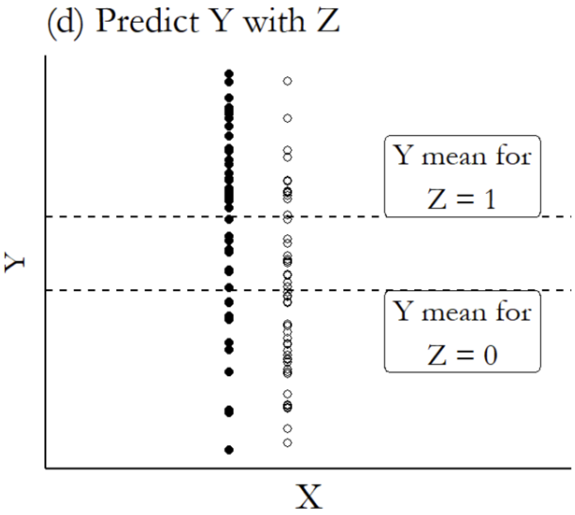


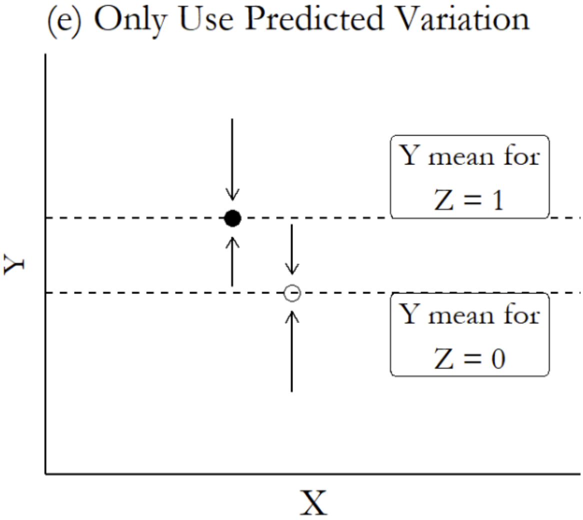
(Huntington-Klein, *The Effect*, p 472)

How does IV work?



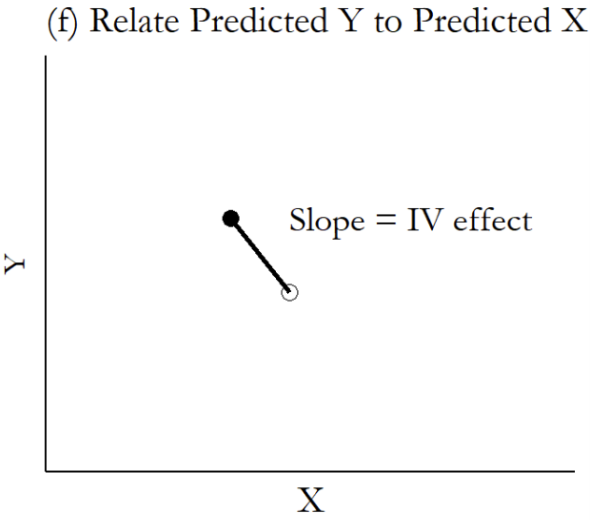
(Huntington-Klein, *The Effect*, p 472)

How does IV work?



(Huntington-Klein, *The Effect*, p 472)

How does IV work?



(Huntington-Klein, *The Effect*, p 472)

How does IV work?

- Usually: two-stage least squares, or **2SLS**

1. Run a 'first-stage' regression to predict the treamtnet with the instrument
2. Use the predicted values to predict the outcome in the 'second-stage'

Alternative approaches to IV: build your own

Journal of Conflict Resolution
2021, Vol. 65(2-3) 563-590

DOI: 10.1177/0022002720963674
journals.sagepub.com/home/jcr
SAGE

Roads to Rule, Roads to Rebel: Relational State Capacity and Conflict in Africa

Carl Müller-Crepon[1], Philipp Hunziker[2], and Lars-Erik Cederman[3]

Abstract

Weak state capacity is one of the most important explanations of civil conflict. Yet, current conceptualizations of state capacity typically focus only on the state while ignoring the relational nature of armed conflict. We argue that opportunities for conflict arise where relational state capacity is low, that is, where the state has less control over its subjects than its potential challengers. This occurs in ethnic groups that are poorly accessible from the state capital, but are internally highly interconnected. To test this argument, we digitize detailed African road maps and convert them into a road atlas akin to Google Maps. We measure the accessibility and internal connectedness of groups via travel times obtained from this atlas and simulate road networks for an instrumental variable design. Our findings suggest that

Alternative approaches to IV: build your own

Instrumental Variable Approach

We complement our robustness checks with an instrumental variable (IV) strategy that addresses potential omitted variable biases not captured by the previous tests. In particular, there might be hitherto unmeasured group-level characteristics that have affected colonial road building and recent conflict. To address such endogeneity as well as potential systematic measurement bias in the Michelin maps, our IV approach exploits variation from road networks simulated on the basis of countries' population distribution. Our IV approach improves identification by isolating the component of RSC that is due to the spatial population distribution within a country. While population distributions are less malleable than road networks, populations are not randomly distributed. We must therefore rely on the assumption that the population distributions that produce our simulated road networks are conditionally exogenous to conflict. We address potential violations of this assumption below.

Alternative approaches to IV: build your own

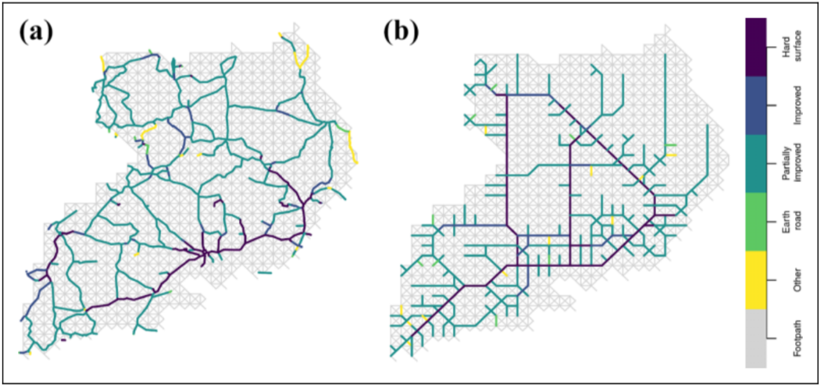


Figure 3. Observed and simulated road network in Uganda, 1966. (a) Observed network. (b) Simulated network.