An Introduction to Causal Inference

EDLD 650: Advanced Research Methods Seminar

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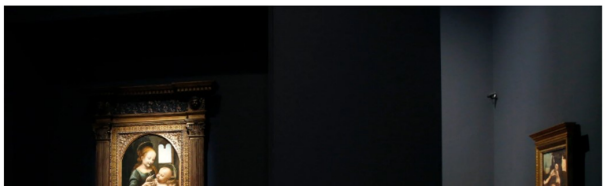
Why causal research? (I)

The New York Times

Another Benefit to Going to Museums? You May Live Longer

Researchers in Britain found that people who go to museums, the theater and the opera were less likely to die in the study period than those who didn't.



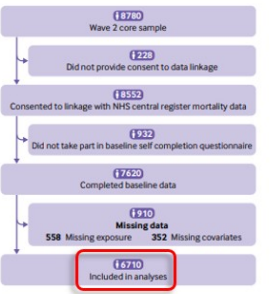


Fig 1 | Flowchart of participants included in the study

average of standardised scores of memory, executive function, processing speed, and orientation in time using validated measures from a neuropsychological battery[29]).

Social covariates included perceived loneliness (measured using the four item University of California, Los Angeles (UCLA) loneliness scale); the number of reported close friends (0, 1-2, 3-5, and 6 or more); whether participants lived alone; the frequency with which participants engaged in civic activities (including political parties, trade unions, environmental groups, tenants or residents associations, neighbourhood watch, church or religious groups, charitable associations, evening classes, social clubs, sports clubs, exercise classes, or other clubs or societies); the frequency with which people saw friends, family, or children (less than once a month, once or twice a month, once or twice a week, or three or more times a week); and whether participants had a hobby or pastime.

Statistical analysis

Table 1 shows the importance of baseline differences between participants based on end mortality status and arts engagement, which was calculated using χ^2 tests. We estimated cumulative mortality by using the Kaplan-Meier method. Unadjusted and adjusted hazard ratios of mortality and 95% confidence intervals were calculated using Cox proportional hazards regression models. We [illegible] death, censoring (the date of the last interview before drop out), or latest available follow-up (165 months from baseline). Sensitivity analyses that used survival time from baseline interview produced comparable results. We adjusted models for demographic variables (age, sex, marital status, ethnicity, educational qualifications, wealth, employment status, and occupational status); health related variables (eyesight, hearing, depressive symptoms, other psychiatric conditions, diagnosis of cancer, lung disease or cardiovascular disease, history of any other long-term condition, smoking, alcohol consumption, sedentary behaviours, mobility, problems in undertaking activities of daily living, osteoporosis, and cognition); and social covariates (loneliness, number of close friends, living alone, frequency of civic engagement, frequency of social engagement, and whether participants had a hobby or pastime).

We stratified analyses by age at which participants' arts engagement was recorded, whether participants had cancer at baseline, and whether participants had problems that affected mobility. With these adjustments made, the proportionate hazards assumption was met (tested using the Schoenfeld residuals test). To explore the minimum strength of association that any unmeasured confounder would need to fully explain away any association, we calculated the E value, which is a measure of whether the inclusion of further confounders is likely to lead to the attenuation of results.[30] All analyses were weighted using inverse probability weights to ensure national representation and to take account of differential non-response. We additionally explored whether differences in baseline factors between those who do and do not engage in arts could explain an association between receptive arts engagement and mortality by rerunning analyses using nested models of covariates and by calculating the percentage of protective association explained (PPAE) by including such variables in the model using the equation: PPAE=(HR (E+C+X)–HR (E+C))/(1–HR (E+C))*100, where HR=hazard ratio, E=exposure, C=covariates, and X=explanatory variable being tested.[31] We confirmed that there were no issues with [illegible] and the models met regression assumptions.

We carried out three sets of sensitivity analyses. Our first set assessed whether results were found consistently across subgroups (by rerunning analyses on subgroups) or if certain factors acted as moderators (by including interaction terms in models). In relation to demographics, we tested age and sex specifically. In relation to socioeconomic factors, we tested employment status, wealth, education, and social status. Finally, in relation to social factors, we tested marital status, living alone, loneliness, number of friends, frequency of social engagement, and civic engagement.

Our second set of sensitivity analyses tested with greater rigour whether some of our identified confounders could account for any associations by including a range of further factors that could have acted as confounders. To test whether results were because of physical function, in addition to controlling for sedentary behaviours, we further adjusted for frequency of vigorous physical activity and presence of any mobility problems that affected walking. To test whether broader aspects of socioeconomic status

Why causal research? (II)

- **Abstract**: We estimate the relationship between X and Y.
- **Intro**: It would be important to know whether X causes Y.
- **Data and Analytic Strategy**: Our data and research design are observational, and so we are unable to identify the causal impact of X on Y.
- **Results**: We find that a one-percentage point difference in X is associated with a 4.5 percentage point difference in Y.
- **Discussion**: A major limitation of our study is that we cannot rule out the possibility of confounders or reverse causality. Thus, while we cannot say whether X causes Y, our findings show this is a strong possibility and future research should explicitly explore it.
- **Conclusion**: But really 😉, X causes Y.

Why *careful* causal research?

Research Report

aps ASSOCIATION FOR PSYCHOLOGICAL SCIENCE

Power Posing: Brief Nonverbal Displays Affect Neuroendocrine Levels and Risk Tolerance

Psychological Science
21(10) 1363–1368

DOI: 10.1177/0956797610383437
http://pss.sagepub.com
SAGE

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Abstract

Humans and other animals express power through open, expansive postures, and they express powerlessness through closed, contractive postures. But can these postures actually cause power? The results of this study confirmed our prediction that posing in high-power nonverbal displays (as opposed to low-power nonverbal displays) would cause neuroendocrine and behavioral changes for both male and female participants: High-power posers experienced elevations in testosterone, decreases in cortisol, and increased feelings of power and tolerance for risk; low-power posers exhibited the opposite pattern. In short, posing in displays of power caused advantaged and adaptive psychological, physiological, and behavioral changes, and these findings suggest that embodiment extends beyond mere thinking and feeling, to physiology and subsequent behavioral choices. That a person can, by assuming two simple 1-min poses, embody power and instantly become more powerful has real-world, actionable implications.

Method

Participants and overview of procedure

Forty-two participants (26 females and 16 males) were randomly assigned to the high-power-pose or low-power-pose condition. Participants believed that the study was about the science of physiological recordings and was focused on how placement of electrocardiography electrodes above and below the heart could influence data collection. Participants' bodies

Error bars represent standard errors of the mean.

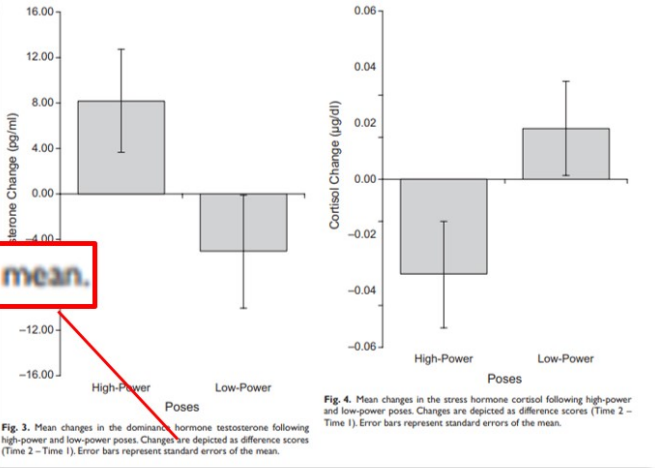


Fig. 3. Mean changes in the dominance hormone testosterone following high-power and low-power poses. Changes are depicted as difference scores (Time 2 – Time 1). Error bars represent standard errors of the mean.

Fig. 4. Mean changes in the stress hormone cortisol following high-power and low-power poses. Changes are depicted as difference scores (Time 2 – Time 1). Error bars represent standard errors of the mean.

Descriptive and causal research

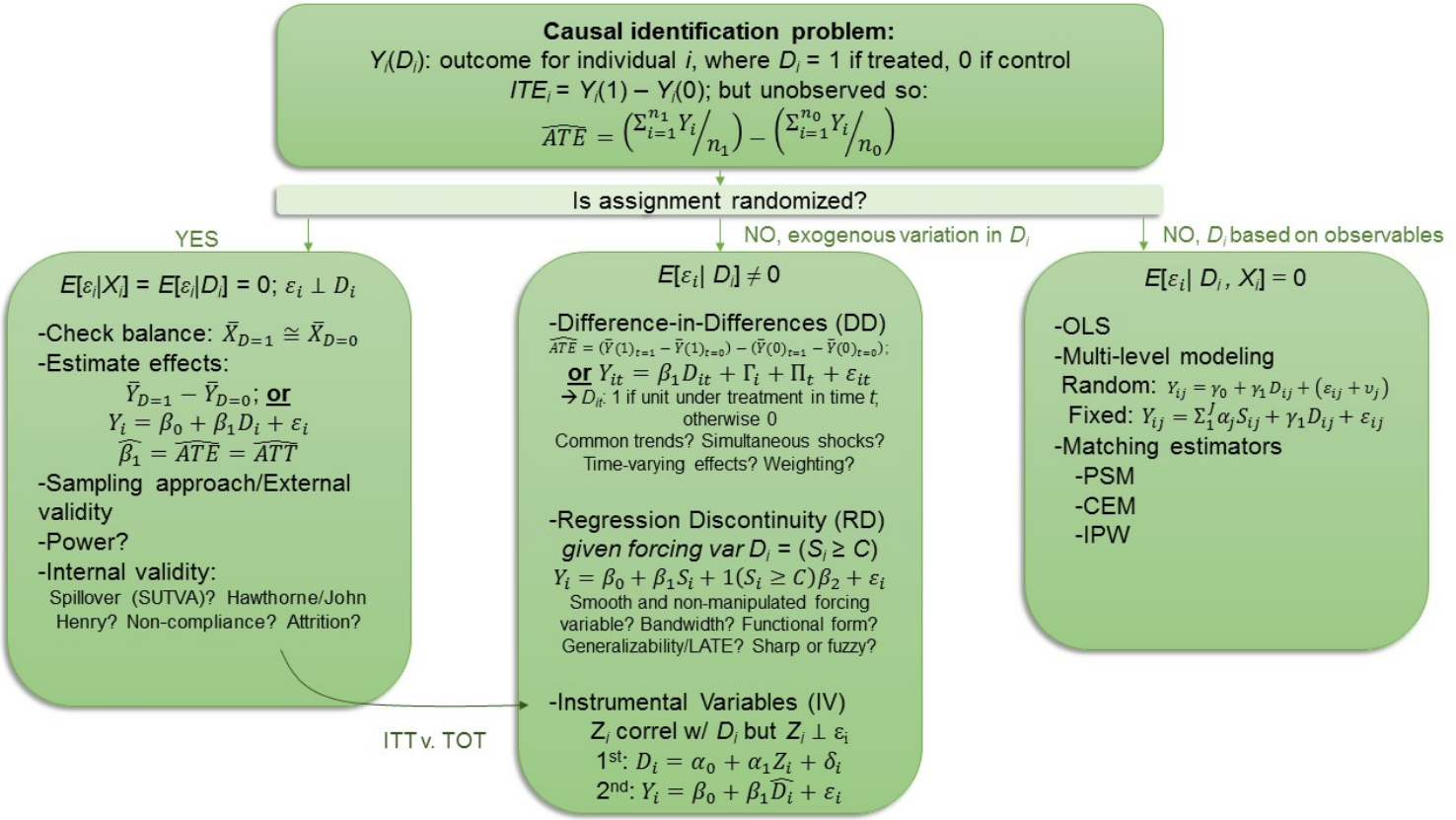
> **Quality causal research question**: Did the Success for All whole-school intervention improve students' reading achievement?
>
> **Quality descriptive research question**[1]: Do the teachers of English Learner students in self-contained classrooms have different pedagogical skill levels than teachers of non-English Learners?

Don't attempt to answer a question that is inherently (or implicitly) causal using a correlational approach! *We only care about the relationship between museum-going and mortality if it is a directionally causal one!*

The overarching goal of this course: To provide you with (some of) the tools to be effective consumers and producers of causal research

[1] Helpful resource: Loeb et al. (2017). Descriptive analysis in education: A guide for researchers. (NCEE 2017-4023). Washington, DC: US DoE, IES

Roadmap


Causal identification problem:
$Y_i(D_i)$: outcome for individual i, where D_i = 1 if treated, 0 if control
$ITE_i = Y_i(1) - Y_i(0)$; but unobserved so:
$\widehat{ATE} = \left(\Sigma_{i=1}^{n_1} Y_i / n_1\right) - \left(\Sigma_{i=1}^{n_0} Y_i / n_0\right)$
Is assignment randomized?
YES
NO, exogenous variation in D_i
NO, D_i based on observables
$E[\varepsilon_i|X_i] = E[\varepsilon_i|D_i] = 0$; $\varepsilon_i \perp D_i$
-Check balance: $\bar{X}_{D=1} \cong \bar{X}_{D=0}$
-Estimate effects:
$\bar{Y}_{D=1} - \bar{Y}_{D=0}$; or
$Y_i = \beta_0 + \beta_1 D_i + \varepsilon_i$
$\widehat{\beta_1} = \widehat{ATE} = \widehat{ATT}$
-Sampling approach/External validity
-Power?
-Internal validity:
Spillover (SUTVA)? Hawthorne/John Henry? Non-compliance? Attrition?
ITT v. TOT
$E[\varepsilon_i| D_i] \neq 0$
-Difference-in-Differences (DD)
$\widehat{ATE} = (\bar{Y}(1)_{t=1} - \bar{Y}(1)_{t=0}) - (\bar{Y}(0)_{t=1} - \bar{Y}(0)_{t=0})$;
or $Y_{it} = \beta_1 D_{it} + \Gamma_i + \Pi_t + \varepsilon_{it}$
→ D_{it}: 1 if unit under treatment in time t, otherwise 0
Common trends? Simultaneous shocks? Time-varying effects? Weighting?
-Regression Discontinuity (RD)
given forcing var D_i = ($S_i \geq C$)
$Y_i = \beta_0 + \beta_1 S_i + 1(S_i \geq C)\beta_2 + \varepsilon_i$
Smooth and non-manipulated forcing variable? Bandwidth? Functional form? Generalizability/LATE? Sharp or fuzzy?
-Instrumental Variables (IV)
Z_i correl w/ D_i but $Z_i \perp \varepsilon_i$
1st: $D_i = \alpha_0 + \alpha_1 Z_i + \delta_i$
2nd: $Y_i = \beta_0 + \beta_1 \widehat{D_i} + \varepsilon_i$
$E[\varepsilon_i| D_i, X_i] = 0$
-OLS
-Multi-level modeling
Random: $Y_{ij} = \gamma_0 + \gamma_1 D_{ij} + (\varepsilon_{ij} + \upsilon_j)$
Fixed: $Y_{ij} = \Sigma_1^J \alpha_j S_{ij} + \gamma_1 D_{ij} + \varepsilon_{ij}$
-Matching estimators
-PSM
-CEM
-IPW

Agenda

1. **Introduction**
    - Correlation $\neq$ causality
    - Roadmap
    - Agenda/goals
2. **A Causal Framework**
    - Experiments and potential outcomes
        - Class 1 Questions (Sections I and II)
    - Complexificating it
        - A word about DAGs
3. **Break**
4. **Nested data**
    - Class 1 Questions (Section III)
5. **Difference-in-differences**
6. **Conclusions**
    - Key course expectations & logistics
    - To-dos
    - Plus/deltas

Goals for today

1. Articulate in words and simple graphical representations challenges in identifying causal relationships in quantitative data

1. Articulate in words and using simple mathematical terms a framework for identifying causal relationships in quantitative data

2. Describe (conceptually) unit fixed effects and their strengths (and limitations) in research designs seeking to identify causal relationships

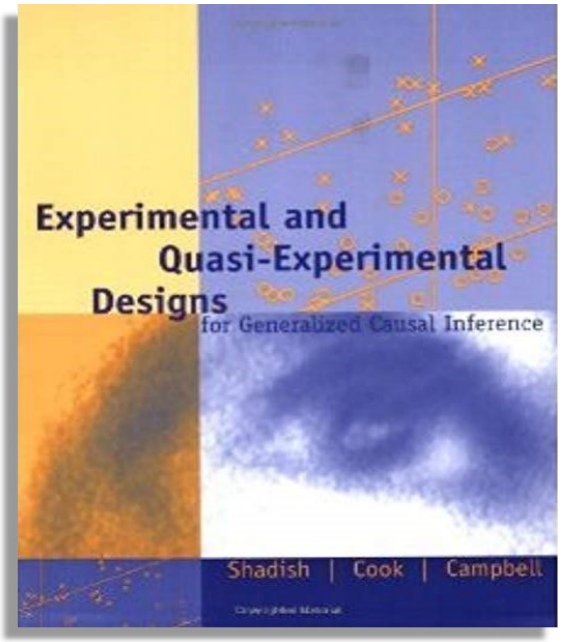
3. Describe the conceptual approach to identifying causal effects using the difference-in-differences framework

Causal frameworks

5 conditions of causal claims

William Shadish, Donald Cook and Thomas Cambpell (2002) adapt John Stuart Mill's critical conditions that must exist in order to defend the claim that one thing causes another:

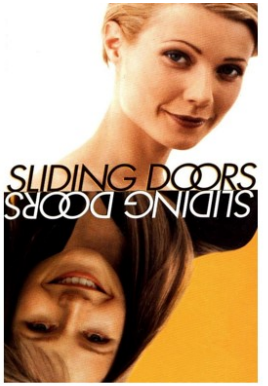
1. Cause must precede effect in time
2. Identified mechanism
3. Consistency
4. Responsiveness
5. No plausible alternative explanation



Experiments and potential outcomes

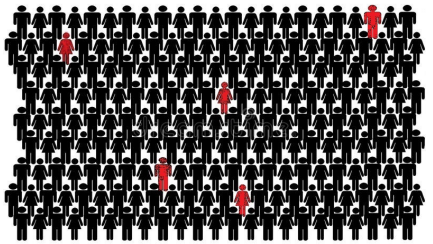
Sliding doors

- What if you missed your train (or didn't)?
- What if you had never been born?
- What if the Beatles never existed?
- What if the Nazis won WWII?



An "ideal" experiment

Hypothetically, we could draw a random sample from a defined population:



- We could implement the treatment for each participant
- And also concurrently NOT implement the treatment
  - We would need to be able to turn back time, and erase the impact and memory of the treatment in each case

While this is obviously impossible, we can imagine that each participant has a value of the outcome that could potentially be revealed under the following experimental conditions:

Y_i^1 = potential value of outcome for i^{th} person, when treated $(D_i = 1)$

Y_i^0 = potential value of outcome for i^{th} person, when NOT treated $(D_i = 0)$

An "ideal" experiment

Y_i^1 = potential value of outcome for i^{th} person, when treated $(D_i = 1)$

Y_i^0 = potential value of outcome for i^{th} person, when NOT treated $(D_i = 0)$

The Individual Treatment Effect (ITE) is the difference in potential outcome values between treatment and control conditions, for each individual:

$$ITE_i = Y_i^1 - Y_i^0$$

We never actually observe this!!!

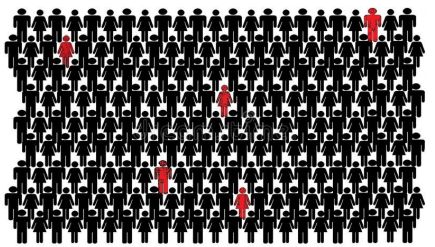
The Average Treatment Effect (ATE) is the average of the individual treatment effects across all participants:

$$\hat{ATE}_i = \frac{1}{n}\sum_i^n ITE_i$$

If the ATE differed from zero, we could claim that the treatment *caused* the effect because there would be no other explanation for the differences detected between the treatment and control conditions!

RCTs: the next best thing?

An "ideal" experiment such as this one is impossible because the same group of people cannot concurrently receive and not receive treatment. We have a missing data problem. We cannot actually estimate individual treatment effects in practice, but if we are willing to make a few reasonable assumptions, we can still estimate the the average treatment effect. This is particularly true when we conduct a randomized control trial (RCT).



We can draw our random sample, and randomly assign each participant to the Treatment (where we measure their value of Y_i^1) or Control (where we measure their value of Y_i^0) condition.

$$\hat{ATE}_i = \frac{1}{n_1}\sum_{i}^{n_1} ITE_i - \frac{1}{n_0}\sum_{i}^{n_0} ITE_i$$

Importance of exogeneity

The big idea in a randomized experiment is that treatment variation is **exogenously and randomly assigned**. An external (or "exogenous") agent, usually the researcher, determines who is treated $(D_i = 1)$ and who is not $(D_i = 0)$.

- Values of all observed and unobserved characteristics of the participants are randomized across treatment and control groups.
- Members of the treatment and control groups are then equivalent, on average, in the population ("equal in expectation") before the experiment begins, on every possible dimension.
- The values of treatment variable, D, will also be completely uncorrelated with all characteristics of participants, observed and unobserved, in the population.

- Exogenous and random treatment variation validates the causal attribution of an experiment. This is referred to as the research design's **internal validity**.

A simple *t*-test

The great thing about experiments is the cleaner the design, the simpler the analysis:

Population average treatment effect: $\mu_1 - \mu_0$

Estimated by the sample mean difference: $\bar{Y_1} - \bar{Y_0}$

To test for a treatment effect, conduct a two-sample *t*-test:

$$t_{obs} = \frac{(\bar{Y_1} - \bar{Y_0})}{\sqrt{\frac{s^2}{n_1} + \frac{s^2}{n_0}}}$$

$$s^2 = \frac{(n_1 - 1)s_1^2 + (n_0 - 1)s_0^2}{n_1 + n_2 - 2}$$

$t_{crit} = t_{df=n_1+n_2-2}^{(\alpha=0.05)}$; if $t_{obs} > t_{crit}$, then reject H_0!!!

No need for a pre-test, no need for controls, no need for complex statistical models!

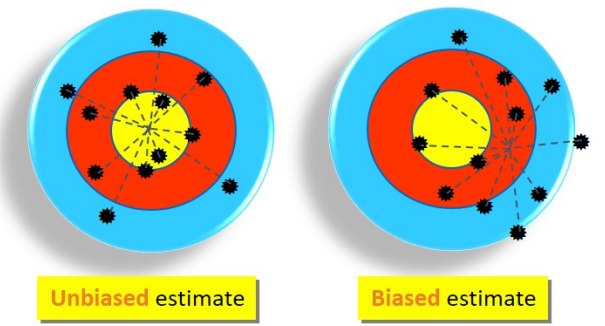
But OLS works too

In an experiment, a critical assumption of the generalized linear model (the foundation for OLS) is automatically satisfied:

$$Y_i = \beta_0 + \beta_1 D_i + \varepsilon_i$$

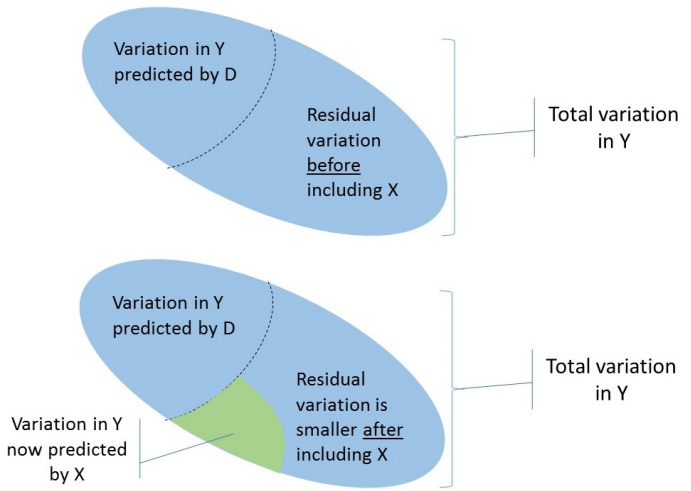
In a randomized experiment, the residuals are uncorrelated with the values of the treatment variable (D_i) because the values of the treatment variable are assigned at random, rendering them uncorrelated with everything, including the residuals.

Reminder of key OLS assumption: residuals must be "independent and identically distributed" (i.i.d.). By independent we mean residuals must be uncorrelated with everything else, including the predictor(s) in the model, otherwise our estimates of the regression parameters will be biased.



But OLS works BETTER!

Even in the most basic of well-executed RCTs, researchers will add covariates.



$$Y_i = \beta_0 + \beta_1 D_i + \varepsilon_i$$

Once you add X, part of Y that is now predicted by X (but wasn't predicted by D by design), is no no longer part of residual

$$Y_i = \beta_0 + \beta_1 D_i + \beta_2 X_i + \varepsilon_i'$$

Reduced residual variance means smaller standard errors, larger t-statistics and **MAWWR POWER!!!**

Cold-calling

Purpose

- Formative assessment
- Fair distribution of participation
- Shared accountability for deep understanding of complex and technical readings

Norms

- Questions posted by Wednesday
- Preparation is expected
- These are hard concepts; mistakes are expected
- Judgments on accuracy of responses are about the responses, not the individual
- Questions and response are about learning, not performance

Structure

- All cold calls will be telegraphed
- Questions will come directly from question list
- Random draw (w/ replacement) from class list
- Ample wait time; multiple "at-bats"
- Teaching staff will identify incomplete or incorrect response and seek clarification
- Extension questions on a volunteer basis

Class 1 Discussion Questions

Sections I and II

More complexity

Threats to experimental validity

1. Contamination of treatment-control contrast

- violations of Stable Unit Treatment-Value Assumption (SUTVA)
- an important assumption: selection of others into an intervention should not affect your outcome

2. Cross overs (aka non-compliance)

3. Attrition

4. Participation in experiment affects behavior

- Hawthorne and John Henry effects

> There is much to explore in these threats to validity. We will address some in the Instrumental Variables unit, but could form entire courses.

Keep it real

Of course, in the real world, there are many reasons researchers are unable to conduct experiments:

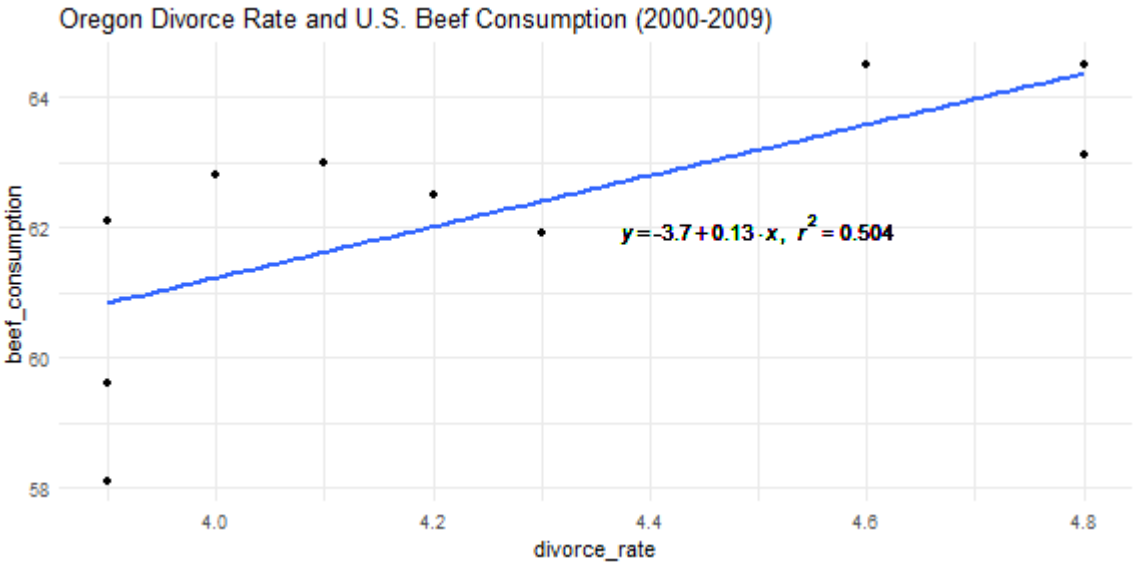
- Cost
- Time
- Willing partners
- Ethics
- Representativeness
- Power
- ...

Thus, in this course, we will primarily concern ourselves with the goal of recovering credibly causal estimates of treatment effects in observational data.

but this is **hard**.

Correlation ≠ causation pt. 562

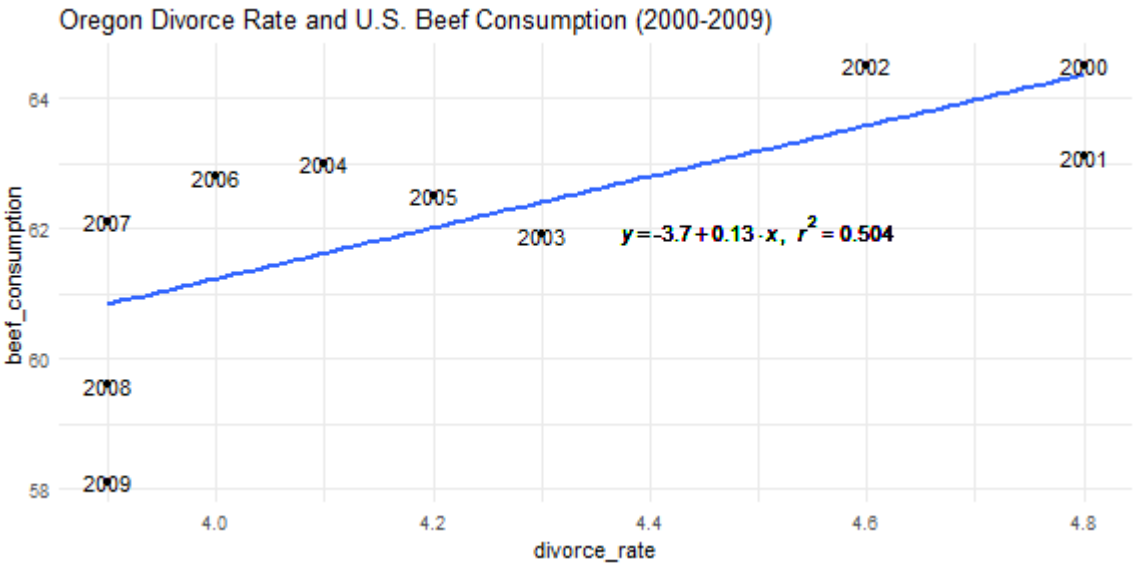
RQ: What is the relationship between Oregon's annual per capita divorce rate and the U.S. per capita annual beef consumption?



On the 10 o'clock news tonight: does U.S. beef consumption cause more "beefs" between Oregonians and their spouses?

Divorce and Beef

Do increases in beef consumption in Oregon **cause** increases in the U.S. divorce rate?



This is a classic problem of a confounder![1]

[1] More fun with spurious correlations

Why correlation $\neq$ causation?

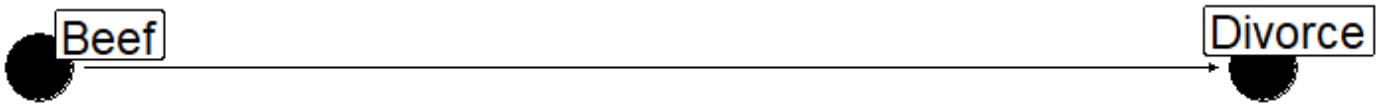
Common barriers in attributing causality to observed co-relationships include:

- Confounders: a third variable causes changes in X and also in Y
- Colliders: a third variable that is caused by both the predictor and outcome; controlling for this can make a true causal relationship disappear!
- Reverse causation: X may cause Y **or** Y may cause X
- Simpson's Paradox: a third variable may reverse the correlation
- Also, **lack** of correlation $\neq$ **lack** of causality

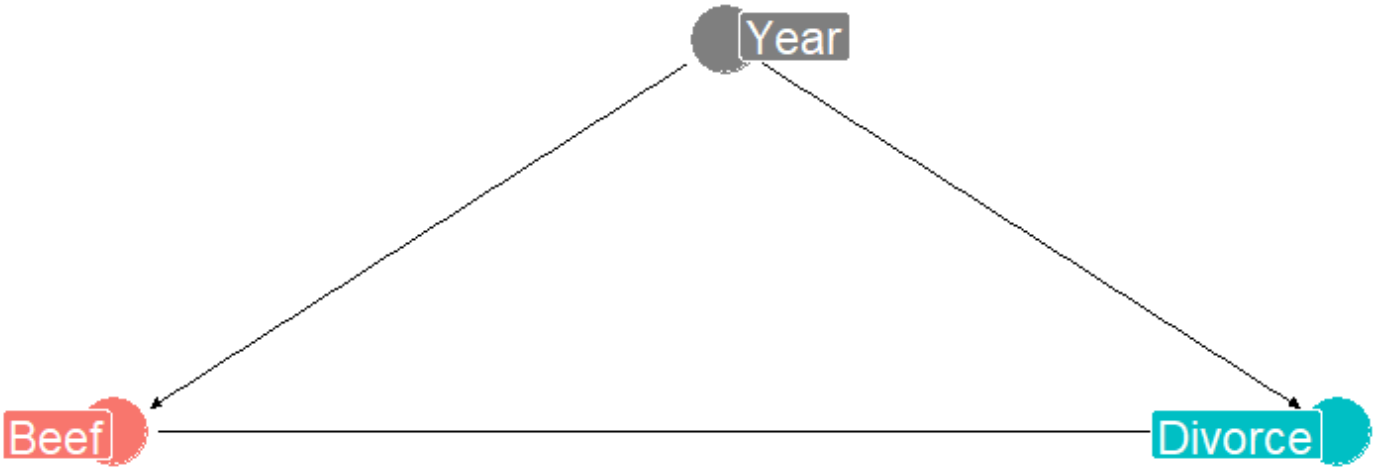
h/t @causalinf

Directed acyclic graphs (DAGs)

Directed Acyclical Graphs (DAGs) can help us visualize the assumptions necessary to estimate causal relationships in observational data through graphical representation.

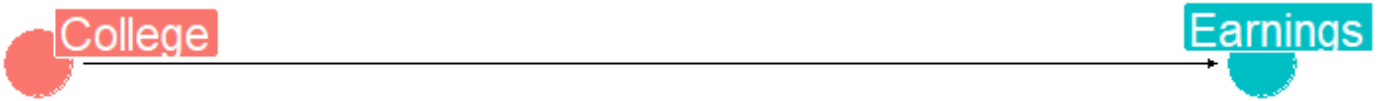


Spurious correlation



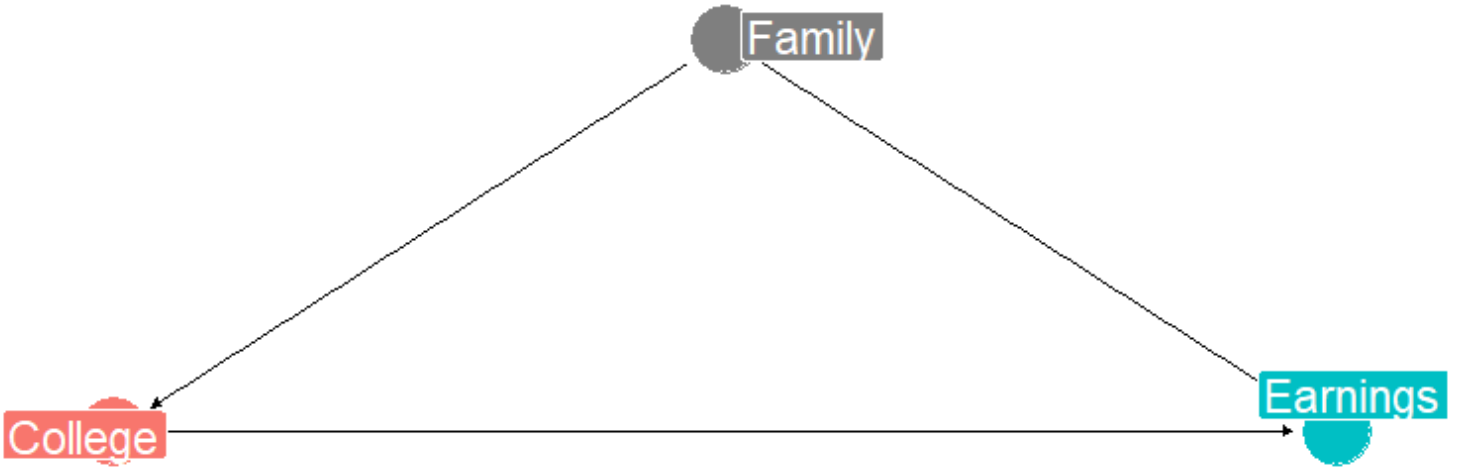
> *It is easy to prove that the wearing of tall hats and the carrying of umbrellas enlarges the chest, prolongs life, and confers comparative immunity from disease…A university degree, a daily bath, the owning of thirty pairs of trousers, a knowledge of Wagner's music, a pew in church, anything, in short, that implies more means and better nurture…can be statistically palmed off as a magic spell conferring all sorts of privileges…The mathematician whose correlations would fill a Newton with admiration, may, in collecting and accepting data and drawing conclusions from them, fall into quite crude errors by just such popular oversights.* -George Bernard Shaw (1906)

A DAG-gone example



- Directed Acyclical Graphs (DAGs)] help us visualize the assumptions necessary to estimate causal relationships in observational data
- Nodes represent variables; arrows represent directional causal effects; missing arrow implies lack of a causal path
- Effects are either:
  - direct $(D \rightarrow Y)$; i.e., the causal effect of D (college) on Y (earnings); **or**

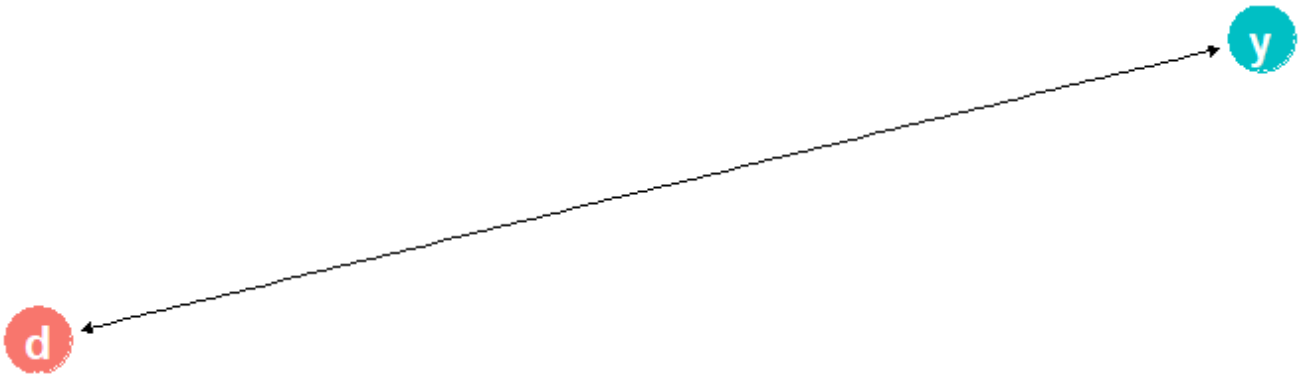
A DAG-gone example



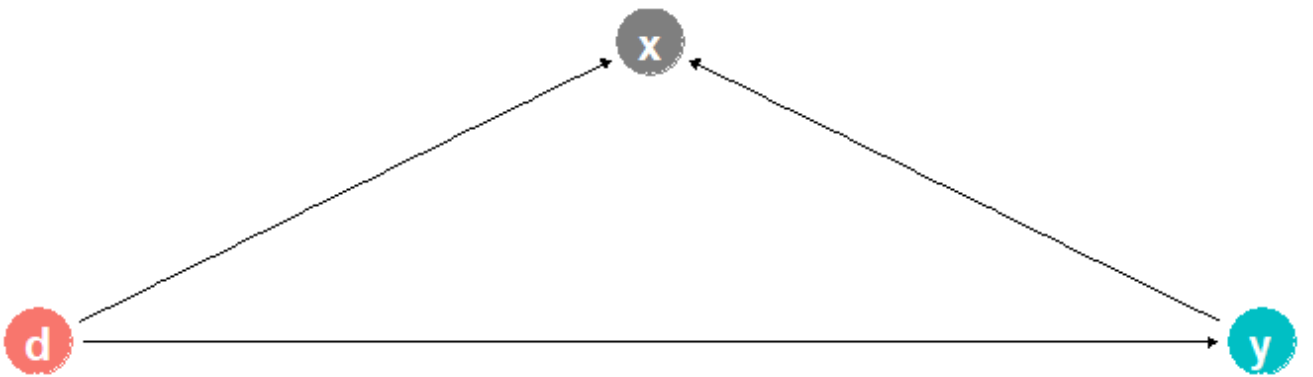
- Directed Acyclical Graphs (DAGs)] help us visualize the assumptions necessary to estimate causal relationships in observational data
- Nodes represent variables; arrows represent directional causal effects; missing arrow implies lack of a causal path
- Effects are either:
  - direct $(D \rightarrow Y)$; i.e., the causal effect of D (college) on Y (earnings); or
  - indirect $(D \leftarrow X \rightarrow Y)$; i.e., a backdoor path created by a confounder
- Here, conditioning on X (observed family characteristics) closes the backdoor and allows a causal estimate

DAGs follow two rules

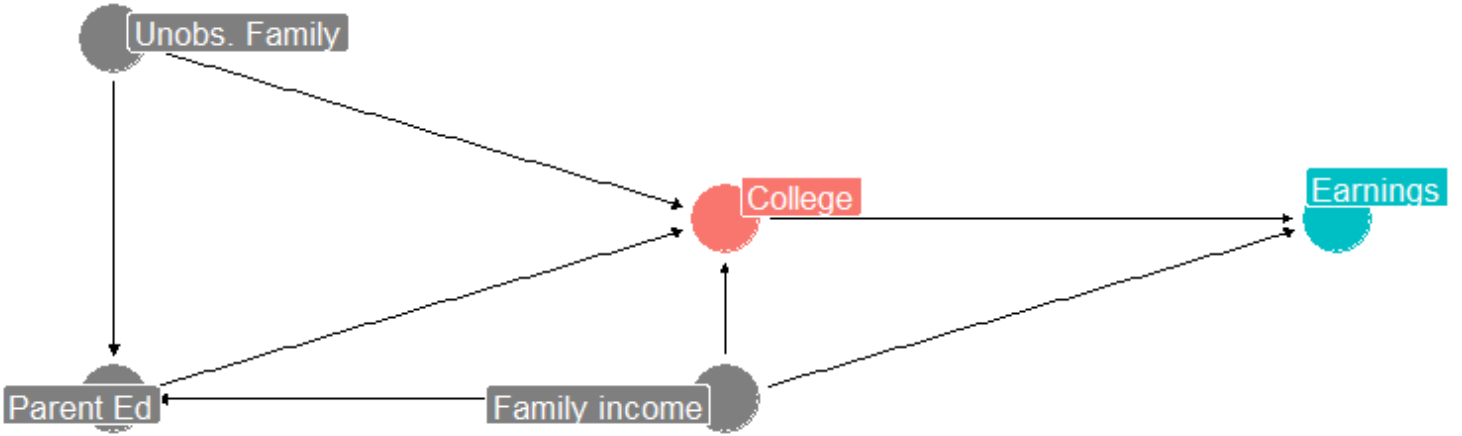
Rule 1: No bidirectional arrows ILLEGAL!! Not "directed"



Rule 2: No feedback loops ILLEGAL!! Not "acyclic"



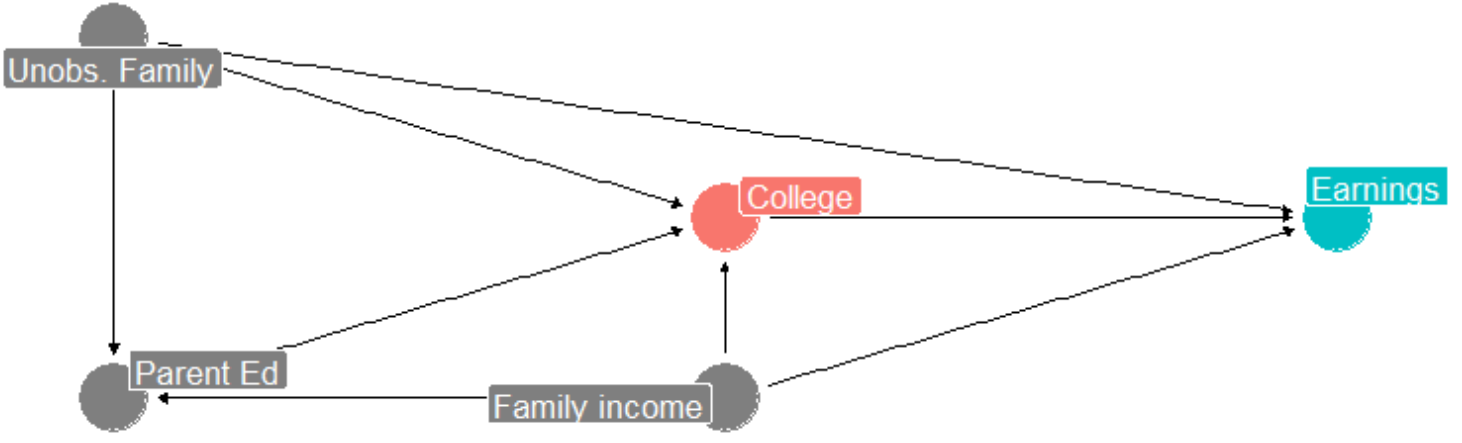
Confounders



We often hope that conditioning on the confounder closes **all** backdoor paths and thus allows us to estimate the direct effect of D on Y:

- $D \rightarrow Y$: causal effect of D on Y
- $D \leftarrow I \rightarrow Y$: income influences both college and earnings
- $D \leftarrow PE \rightarrow I \rightarrow Y$: parental education influences family income which influences own earnings
- $D \leftarrow X \rightarrow PE \rightarrow I \rightarrow Y$: unobserved background characteristics influence parental education, family income, college attendance and own earnings

Confounders



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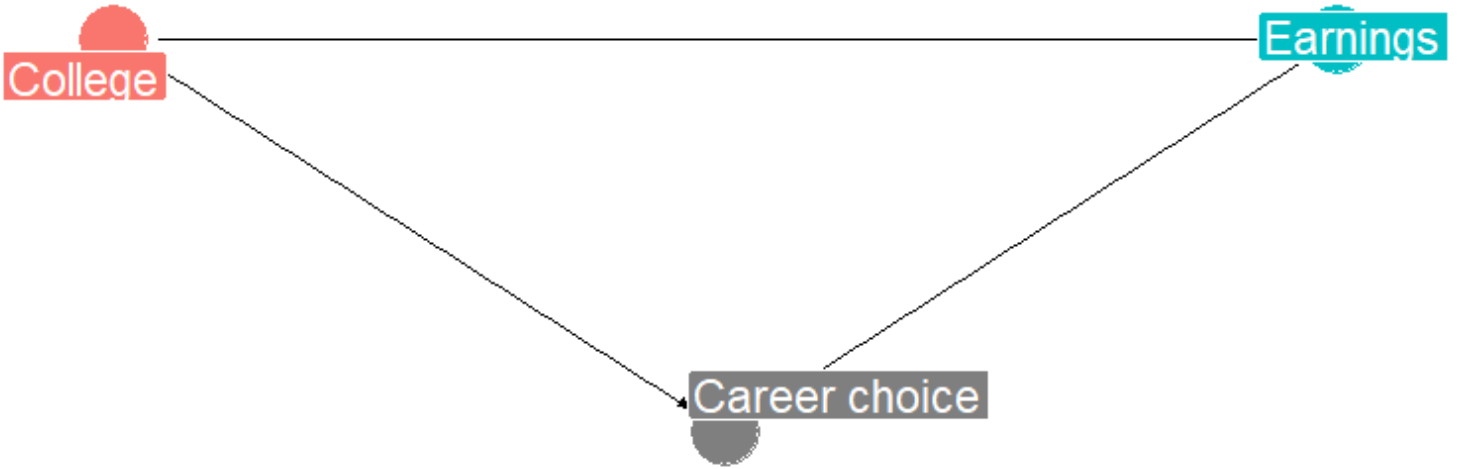
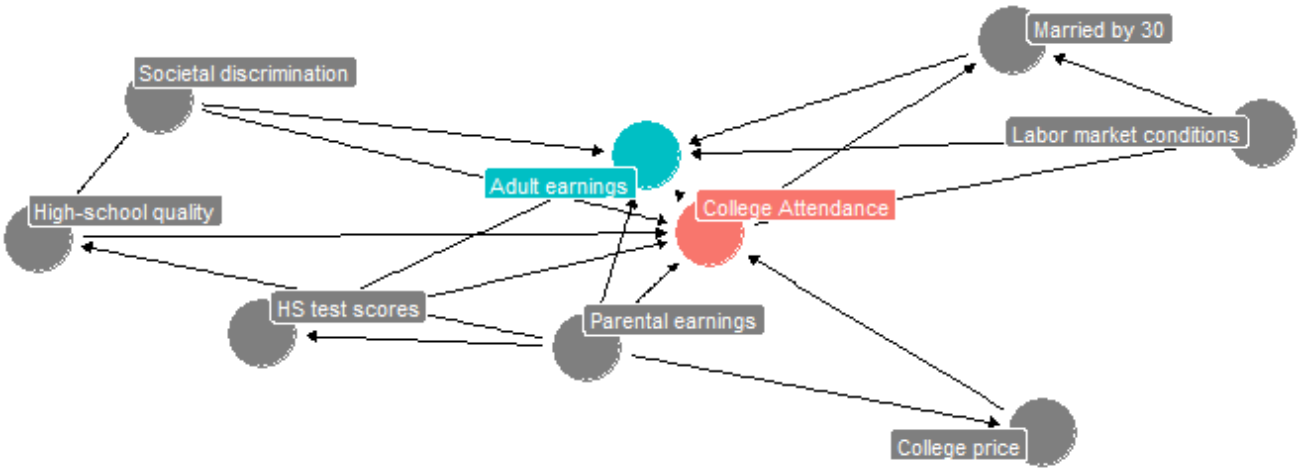
- $D \leftarrow X \rightarrow PE \rightarrow I \rightarrow Y$: unobserved background characteristics influence parental education, family income, college attendance and own earnings
- BUT is it true that family background has no direct effect on earnings?

Colliders



- Career choice is a collider.
- No need to condition on it as the backdoor path is already closed
- Leave colliders alone! Beware of conditioning on them and thereby opening backdoors or (worse) introducing bias.
  - Here, doing so might underestimate the effect of going to college

Where DAGs get tricky (for me)



DAGs can be an intuitive and careful way of thinking through causal research design (see Pearl, 2009). They also risk encouraging the researcher to believe she can solve by analysis what is broke by design (see Imbens, 2020).

In this class, we'll use the **potential outcomes framework** and rely on research designs in which we can credibly argue that **assignment to treatment is exogenous or based on observable characteristics**, but concepts such as confounders, colliders and controlling backdoors are valuable parts of your toolkits. You can learn much more about DAGs than I have presented here in our SEM sequence (EDLD 633/634)!

Break

Nested Data

What is nested data?

Recall the Success for All evaluation from *Methods Matter*[1]

```
ch7_sfa <- read_dta(here("data/ch7_sfa.dta"))
```

```
...
#>   schid    stuid wattack sfa    ppvt sch_ppvt
#>   <fct>    <dbl>   <dbl> <fct> <dbl>    <dbl>
#> 1 1     10158087     469 1        89     90.6
#> 2 1     10217961     486 1        83     90.6
#> 3 1     10486718     501 1        90     90.6
...
```

```
...
#>   schid    stuid wattack sfa    ppvt sch_ppvt
#>   <fct>    <dbl>   <dbl> <fct> <dbl>    <dbl>
#> 1 41    31979390     473 0        78     83.6
#> 2 41    31989400     485 0        65     83.6
...
```

[1] Most datasets from *MM* available from UCLA stats site.

Modeling nested data

Physical nesting

- Our data can be nested in multiple units: students inside classrooms, classrooms inside schools, schools inside districts, districts inside states, etc.

Conceptual nesting

- If we observe students across multiple years, we will have multiple observations nested inside students
- If we administer assessments multiple times, we will have tests nested inside students

Each of these forms of nesting have implications for how we model treatment effects (and on our standard errors).

In the SfA example, we want to capture the effect of receiving the SfA treatment, **independent of the effect of the unobserved and observed qualities of the school the student attends**.

Two common approaches

Random intercepts (aka random effects)

$$WATTACK_{ij} = \gamma_0 + \gamma_1 SFA_j + (\varepsilon_{ij} + \nu_j)$$

You may also have seen this written as:

$$WATTACK_{ij} = \gamma_{00} + \gamma_{01} SFA_j + (\varepsilon_{ij} + \nu_{0j})$$

THESE ARE IDENTICAL!

Two common approaches

Random intercepts (aka random effects)

$$WATTACK_{ij} = \gamma_0 + \gamma_1 SFA_j + (\varepsilon_{ij} + \nu_j)$$

You may also have seen this written as:

$$WATTACK_{ij} = \gamma_{00} + \gamma_{01} SFA_j + (\varepsilon_{ij} + \nu_{0j})$$

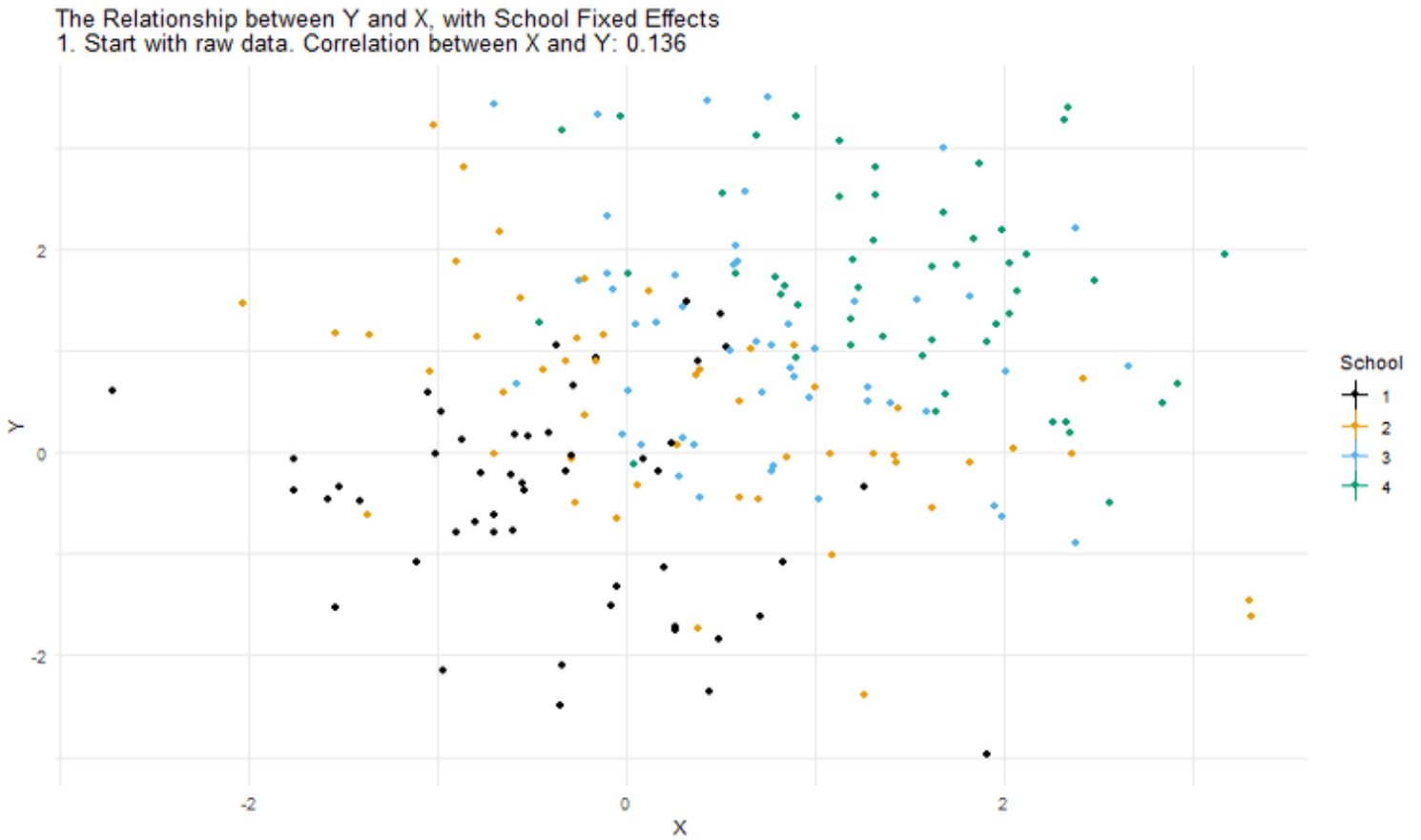
Fixed intercepts (aka fixed effects)

$$WATTACK_{ij} = \sum_{1}^{J} \alpha_j S_{ij} + \gamma_1 SFA_{ij} + \varepsilon_{ij}$$

Notice the within-school variation in treatment in this hypothetical example

> A note on notation: fixed effects are often represented with capital Greek letters $(e.g., \Gamma_j, \Pi_t, \Delta_k)$. Vectors of covariates are often represented with vector notation $(e.g., \mathbf{X}_{ij})$

What is a fixed effect doing?



h/t @nickchk

Random v. Fixed Effects

	Random effects	Fixed effects
Strengths	- Minimal loss of power - Preserves (almost all of) outcome variance	- Accounts for observed and unobserved, time-invariant, within-group differences - Reduces outcome variance to only that relevant to estimating treatment effect
Limitations	- Introduces bias if any correlation between predictors and group-level residuals - Less transparent (more complex) interpretation	- Sacrifices degrees of freedom - Cannot have hierarchically nested fixed effects - Cannot have fixed effect collinear with level of treatment - Cannot include adjustments ("controls") that are invariant within unit

Random v. Fixed Effects

Some guidelines:

- Preference should be informed by data structure, analytic strategy and context[1]
- In both cases, need to pay attention to how you calculate standard errors
- Often disciplinary preferences
- Generally, with long panels (many w/in grouping unit observations) and in non-experimental settings where we seek to estimate treatment effects, fixed effects are preferable

[1] See Clark & Linzer (PSRM, 2015) for a short, minimally technical, summary. *Note*: mixed models with both fixed- and random-intercepts are possible as well as are many other multi-level models (random slopes, random slopes and intercepts, etc.). Consider taking our multi-level modeling sequence (EDLD 628/629) to learn more.

Random intercepts application

```
sfa <- lme4::lmer(wattack ~ sfa + (1 | schid), data=ch7_sfa)
summary(sfa)
```

```
...
#> Random effects:
#>  Groups   Name        Variance Std.Dev.
#>  schid    (Intercept)  75.69    8.70
#>  Residual             314.23   17.73
#> Number of obs: 2334, groups:  schid, 41
#>
#> Fixed effects:
#>             Estimate Std. Error t value
#> (Intercept)  475.302      2.035 233.616
#> sfa1           4.366      2.844   1.535
#>
#> Correlation of Fixed Effects:
#>      (Intr)
#> sfa1 -0.715
...
```

Random intercepts application

```
...
#> Random effects:
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#>
#> Correlation of Fixed Effects:
#>      (Intr)
#> sfa1 -0.715
...
```

Compare the intra-class correlation (ICC) $(\hat{\rho})$ w/ Table 7.1 in *MM* (p. 114):

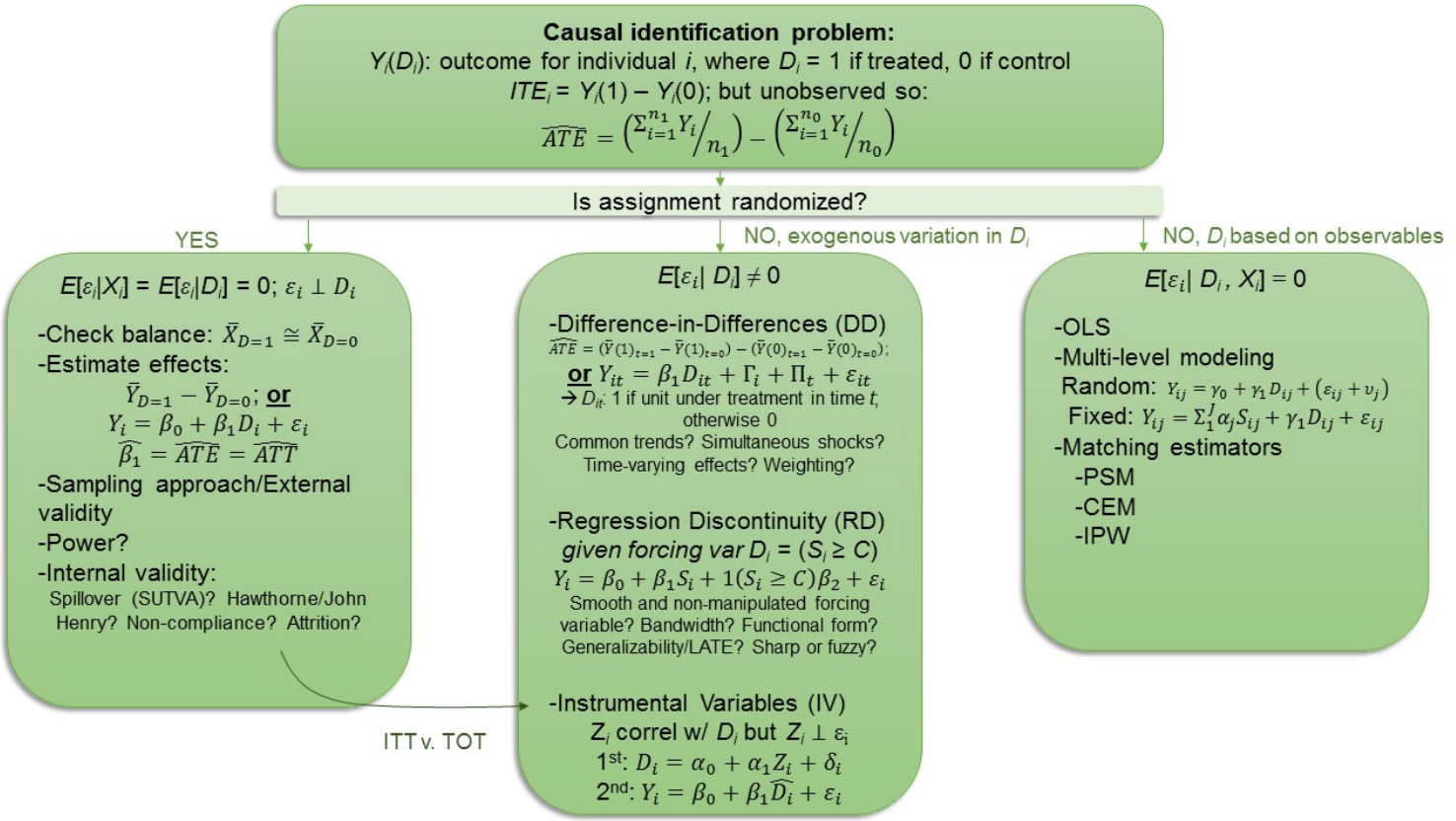
$$\hat{\rho} = \frac{75.69}{75.69 + 314.23} = 0.194$$

Class 1 Discussion Questions

Section III

1. Review your answers to Section III
2. Revise any of your answers based on the information from the past slides
3. What is still unclear? Turn-and-talk with neighbor to see if you can gain clarity
4. We will share out any outstanding questions for the group to answer

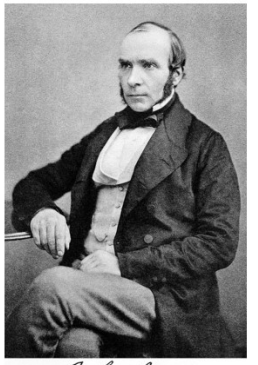
Roadmap

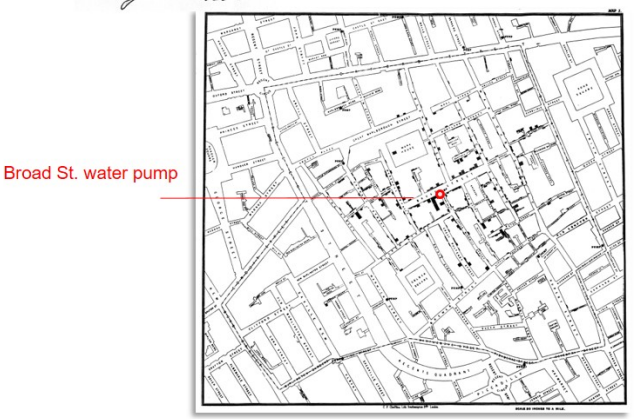

Causal identification problem:
$Y_i(D_i)$: outcome for individual i, where D_i = 1 if treated, 0 if control
$ITE_i = Y_i(1) - Y_i(0)$; but unobserved so:
$\widehat{ATE} = \left(\Sigma_{i=1}^{n_1} Y_i / n_1\right) - \left(\Sigma_{i=1}^{n_0} Y_i / n_0\right)$
Is assignment randomized?
YES
NO, exogenous variation in D_i
NO, D_i based on observables
$E[\varepsilon_i|X_i] = E[\varepsilon_i|D_i] = 0$; $\varepsilon_i \perp D_i$
-Check balance: $\bar{X}_{D=1} \cong \bar{X}_{D=0}$
-Estimate effects:
$\bar{Y}_{D=1} - \bar{Y}_{D=0}$; or
$Y_i = \beta_0 + \beta_1 D_i + \varepsilon_i$
$\widehat{\beta_1} = \widehat{ATE} = \widehat{ATT}$
-Sampling approach/External validity
-Power?
-Internal validity:
Spillover (SUTVA)? Hawthorne/John Henry? Non-compliance? Attrition?
ITT v. TOT
$E[\varepsilon_i|D_i] \neq 0$
-Difference-in-Differences (DD)
$\widehat{ATE} = (\bar{Y}(1)_{t=1} - \bar{Y}(1)_{t=0}) - (\bar{Y}(0)_{t=1} - \bar{Y}(0)_{t=0})$;
or $Y_{it} = \beta_1 D_{it} + \Gamma_i + \Pi_t + \varepsilon_{it}$
→ D_{it}: 1 if unit under treatment in time t, otherwise 0
Common trends? Simultaneous shocks? Time-varying effects? Weighting?
-Regression Discontinuity (RD)
given forcing var $D_i = (S_i \geq C)$
$Y_i = \beta_0 + \beta_1 S_i + 1(S_i \geq C)\beta_2 + \varepsilon_i$
Smooth and non-manipulated forcing variable? Bandwidth? Functional form? Generalizability/LATE? Sharp or fuzzy?
-Instrumental Variables (IV)
Z_i correl w/ D_i but $Z_i \perp \varepsilon_i$
1st: $D_i = \alpha_0 + \alpha_1 Z_i + \delta_i$
2nd: $Y_i = \beta_0 + \beta_1 \widehat{D_i} + \varepsilon_i$
$E[\varepsilon_i|D_i, X_i] = 0$
-OLS
-Multi-level modeling
Random: $Y_{ij} = \gamma_0 + \gamma_1 D_{ij} + (\varepsilon_{ij} + \upsilon_j)$
Fixed: $Y_{ij} = \Sigma_1^J \alpha_j S_{ij} + \gamma_1 D_{ij} + \varepsilon_{ij}$
-Matching estimators
-PSM
-CEM
-IPW

Difference-in-differences (DD)

S. London cholera outbreak 1854

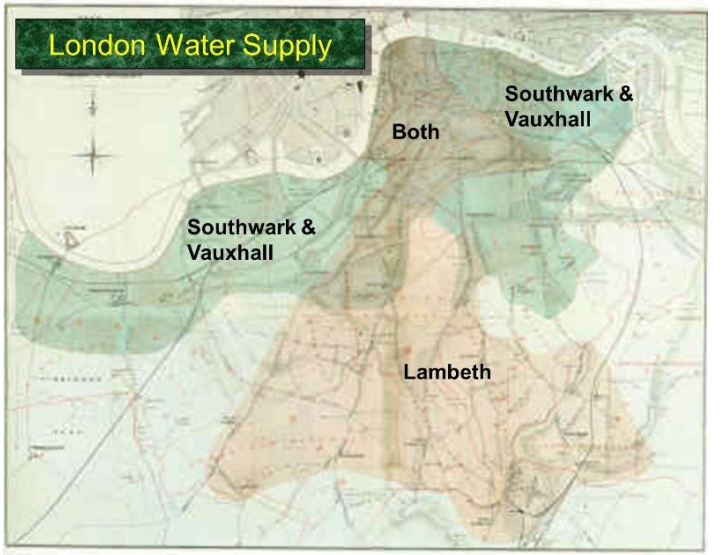
- Londed was a crowded, dirty city w/ waste disposed directly in Thames River
- Disease poorly understood; cholera widely believed to be caused by miasma & contagion
- Outbreak in S. London in summer of 1854 killing over 5,000
    - Followed an earlier outbreak in 1849 that had killed >6,000
- Physician John Snow had developed a theory that these illnesses were water-borne and set out to prove it





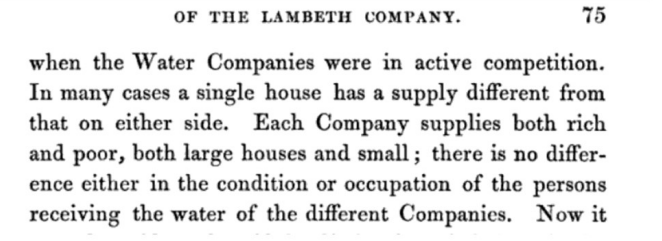
The "Grand Experiment" (I)

- Water is supplied to households be competing private companies:
    1. Southwark & Vauxhall
    2. Lambeth
- Southwark & Vauxhall water from Thames
- Lambeth from Thames until 1852, then from Ditton (22 miles upstream)
- Some portions of the city receive water from only one of companies; others from both



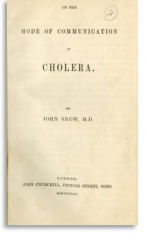
The "Grand Experiment" (II)

- When companies supply to same area, distributed quasi-randomly
- Snow tallies the deaths in all districts supplied by one, the other, or both companies as well as the deaths in the 1849 outbreak



OF THE LAMBETH COMPANY. 75

when the Water Companies were in active competition. In many cases a single house has a supply different from that on either side. Each Company supplies both rich and poor, both large houses and small; there is no difference either in the condition or occupation of the persons receiving the water of the different Companies. Now it



MODE OF COMMUNICATION

OF

CHOLERA.

BY

JOHN SNOW, M.D.

Snow, J. (1855) *On the Mode of Communication of Cholera*. London: Churchill.

We will now pause this history lesson for a short methodological break

So many differences!!!

What is one approach by which we might estimate the effects of a policy change or intervention?

	Treatment group
Before	Y_0
After	Y_1

Could just subtract the mean value of "before" levels of the outcome from mean value of "after":

$$\Delta Y = \bar{Y_1} - \bar{Y_0}$$

BUT, there could be lots of other things going on in between those two times!

The "difference" in DD

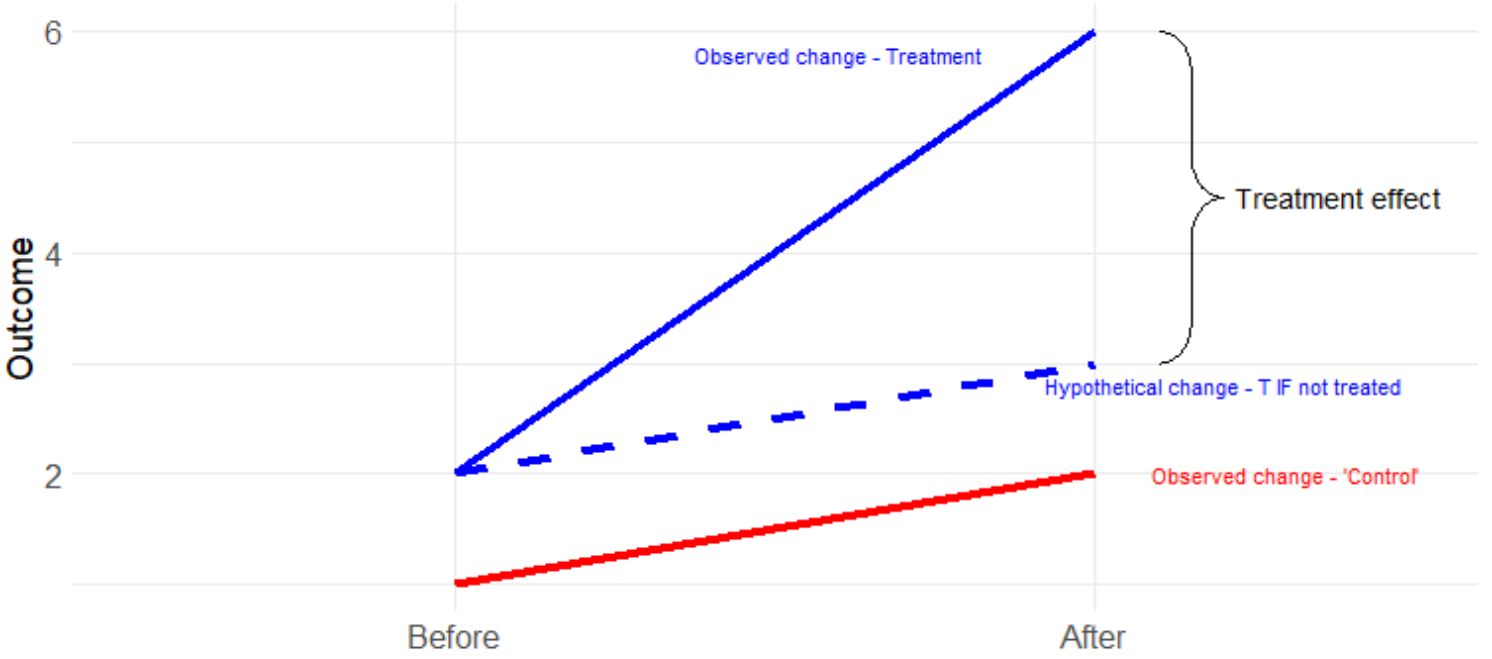
What is one approach by which we might estimate the effects of a policy change or intervention?

	Treatment group	"Control" group
Before	$Y_0^{D=1}$	$Y_0^{D=0}$
After	$Y_1^{D=1}$	$Y_1^{D=0}$

Difference-in-difference (DD) estimates are the difference of two differences:

$$\hat{ATE} = (Y_1^{D=1} - Y_0^{D=1}) - (Y_1^{D=0} - Y_0^{D=0})$$

Graphical DD



John Snow's DD

Table XII. Deaths per 10,000 in homes served by Lambeth and Southwark & Vauxhall, 1849 and 1854

	Treatment = Lambeth	Control = S&V	Diff-in-Diff
Before = 1849	85	135	
After = 1854	19	147	
Difference	-66	12	-78

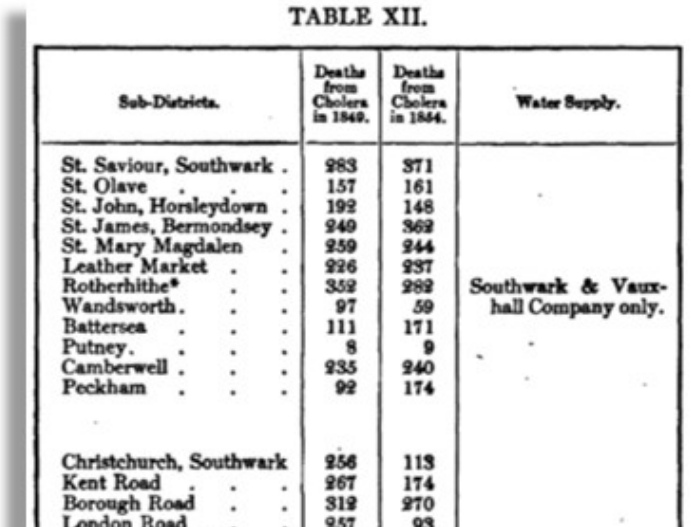


TABLE XII.

Sub-Districts.	Deaths from Cholera in 1849.	Deaths from Cholera in 1854.	Water Supply.
St. Saviour, Southwark	283	371	
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	Southwark & Vauxhall Company only.
Wandsworth	97	59	
Battersea	111	171	
Putney	8	9	
Camberwell	235	240	
Peckham	92	174	
Christchurch, Southwark	256	113	
Kent Road	267	174	
Borough Road	312	270	
London Road	257	93	

Clients of Southwark & Vauxhall experienced more deaths per 10,000 in the 1854 cholera outbreak than in the 1849 one. The Lambeth clients, therefore, might have expected to have more also, but they had MANY fewer. The only thing that changed was the source of the Lambeth water. From this evidence, Snow claimed that **the only possible cause was the water!**

DD by regression

We can get the same results for a two-period DD in a regression framework, which allows us to:

- Add statistical adjustments (see previous discussion on value in experiments)
- Model various functional forms, and more!

$$Y_{it} = \beta_0 + \beta_1 TREAT_{it} + \beta_2 AFTER_{it} + \beta_3 TREAT \times AFTER_{it} + \varepsilon_{it}$$

where, *TREAT* = 1 if in treatment and = 0 if in control and ...

AFTER = if after the treatment occurred (even if you didn't experience the treatment) and *AFTER* = 0 if before treatment; **OR**

$$CHOLERA_{it} = \beta_0 + \beta_1 LAMBETH_{it} + \beta_2 1854_{it} + \beta_3 LAMBETH \times 1854_{it} + \varepsilon_{it}$$

Here, β_3 is our causal parameter of interest. We can interpret it as the causal effect of living in a home that was served water from the Thames on the death rate of residents of those homes.

Synthesis and wrap-up

Goals for today

1. Articulate in words and simple graphical representations challenges in identifying causal relationships in quantitative data
2. Articulate in words and using simple mathematical terms a framework for identifying causal relationships in quantitative data
3. Describe (conceptually) unit fixed effects and their strengths (and limitations) in research designs seeking to identify causal relationships
4. Describe the conceptual approach to identifying causal effects using the difference-in-differences framework

Key logistics

- Review syllabus carefully
- Prepare questions in advance (partner work encouraged)
- Class canceled Jan. 15 for MLK Jr. Day; online video and submission of written responses to class questions
- Review session
    - Review DD details
    - What else? (multi-level models? residuals/standard errors? notation?)
    - When?
- Data Analysis and Replication Exercises (DAREs)
- Project proposal by January 28
    - Meet w/ teaching staff to discuss at least once
    - In class scholarly presentation (March 11)
    - Written final research project (March 20; *optional feedback by March 13*)

To-dos

Week 2: Difference-in-differences

Readings for next week:

- Murnane & Willet, Chapter 8
- Dynarski (2003), Does aid matter?
- Further, MHE: Ch. 5; 'Metrics: Ch. 5, Mixtape: Chs. 8 & 9

Assignments Due

- Complete student survey on Canvas (Jan. 10)
- Watch recorded video and submit written responses to Class 2 questions (Jan. 16)
- DARE #1 due: 11:59pm January 21

Feedback

Plus/Deltas

- What worked about today's class?
- What could be improved or changed about the pedagogical process of today's class?

Clear/Murky

- What substantively is most clear to you or got clarified during class today?
- What is the muddiest substantive topic for you?
- *For today only, could you please indicate (a) what times you are available next week for a review session; (b) what topics would you like to see included in the review?*