# Causal Inference

## Mixtape Session

# Roadmap

Counterfactuals and causality
- Causality and models
- Potential outcomes
- Randomization and selection bias
- Randomization inference

Directed Acyclic Graphs
- Graph notation
- Backdoor criterion
- Collider bias
- Front door criterion
- Concluding remarks

# References

Material drawn from a number of sources

- Speech by Card on model and design based approaches to empirical micro
- Lewbel (2019), “Identification Zoo”
- Netto (2021), “Experiments in the Armchair: History of Microeconometrics and Program Evaluation”
- Nobel Prize 2021 scientific document, “Answering Causal Questions using Observational Data”

# Models

All models are wrong but some are useful – George Box

- Economists use models to reduce the infinitely complex world into something we hope helps us understand it better
- Economists hope their models will help us better understand labor and product markets better so that we can use policies (or not) to make more informed decisions about resource allocation

# Causality and model

- What is a theoretical model in economics?
- What role, if any, should "the model" play in causal identification?
- Empirical micro has been divided along two almost philosophical approaches to causal inference over the years
  - → **Model**: Causality is model-based. It only exists within the framework of a theory that says "X causes Y" (e.g., Heckman)
  - → **Design**: Causality is design-based. No causality without *physical* manipulation of a treatment $X$ (e.g., Rubin, Holland)

# Economist's models

Economics models typically contain the following

- Preference functions (e.g., quasi concave utility), objective function (e.g., profit)
- Constraints (e.g., time, budgets)
- Endogenous choice variables (e.g., bundles of goods, output)
- Equilibrium (e.g., first order conditions, Nash equilibrium)

# Three economic models within empirical micro

1. **Approximating models**: Consumer demand, labor supply models (e.g., Mincer 1958; 1974)
   - → Theory implies $y_i = f_i(x_i)$ with restrictions on $f_i$ (e.g., concavity)
   - → Researcher estimates a simpler version

$$y_i = \alpha + x_i\beta + \varepsilon_i$$

2. **Exact models**: Models gives us all causes ("complete DGP")
   - → Utility, heterogenous taste, complete demand
   - → Estimate model parameters and distribution of heterogeneity
   - → Functional form, useful for welfare analysis
3. **Working model**: Program evaluation (e.g., Princeton), non-market behavior (e.g., Levitt)
   - → No precise model was used or relied for estimation, though it may guide the questions
   - → Structural outcome model with signed coefficients

# Causality and labor economics

- **Mid-century**: Macro and linear systems of equations, identification problems
- **1970s**: Micro data shows up, McFadden's logit, Heckman's selection model
- **1980s**: Econometric critiques like the Lucas critique, Leamer "specification searching", LaLonde (1985) critiques program evaluation, Lewis dismisses IV and Heckit models
- **1980s/1990s**: Design emerges within the Princeton labor group, randomized instruments, "clever" natural experiments, "plausibly exogenous", RDD, difference-in-differences

# Princeton and design

- 1970s saw rising inequality and poverty
- Princeton becomes ground zero for design: Albert Rees brings in micro data; advises Orley Ashenfelter
- Chicago and others focus on model driven approaches (Heckman)
- Ashenfelter focuses on job trainings program, invents difference-in-differences (though John Snow did it 100 years before)
- Extensive mentoring: David Card, Bob LaLonde, Josh Angrist, Janet Currie, Philip Levine, Pischke, and on and on
- Other faculty: Alan Krueger, Guido Imbens, Don Rubin
- Adoption of potential outcomes (Krueger notes the NEJM and medical concepts)

# Credibility revolution wins

- Design approach tends to crowd out the model based approach in the applied micro fields like labor, health, development with exceptions (Wolpin, Rust)
- Nobel Prizes for experiments, RCTs and causal inference (Vernon Smith, Bannerjee, Duflo, Kremer, Card, Angrist, Imbens)
- Structural wins too though (Deaton) so the debate still rages

# 2021 Nobel Prize

This course very much is connected to the work of Card, Angrist, Imbens, Krueger, Rubin (three of whom win the Nobel Prize)

- David Card (1/2) for empirical labor
- Josh Angrist (1/4) for causal inference (specifically 1990s papers on IV)
- Guido Imbens (1/4) for causal inference (same as Angrist)

# Design contributions

What are the broad contributions of the design approach to causal inference?

- Counterfactuals and causality; research design outlines an “explicit counterfactual”, randomization is best, credible instruments are second best
- Substantive specification tests: randomization tests in RCTs like balance across covariates, pre-treatment comparisons, event studies, falsification
- Data quality, replication, data warehouses, journals requiring programs, pre-registration of RCTs

# Design limitations

Design approach tends to have limitations though

- Stable Unit Treatment Value Assumption (SUTVA)
- Partial equilibrium and marginal effects only
- Heterogenous treatment effects and LATE
- Short-run (“well-defined counterfactuals” break down)
- Straight-forward predictions disappear (e.g., minimum wage)

# Identification

Competing approaches between two schools of econometric thought

- **Design**: Emphasized credible identification with testing and evaluating of assumptions (e.g., pre-trends, smoothness using covariates, McCrary density test)
- **Model**: Functional form, exclusion, calibration

# Confidence differs

Schools of thoughts use their models in very different ways

- **Design**: Focus tends to be on the Popperian falsifiable predictions of theory (e.g., immigration reduces domestic employment)
- **Model**: Stipulate complete models with an accompanying goal of estimating parameters, do welfare analysis, counterfactual estimation from within the model

# Topics

Dependence on the model vs freed from the model for causal inference increases topics

- **Design**: Anything goes, “economics is what economists study”, happiness, fringe stuff (e.g., sex work) (opening up topics)
- **Model**: Neoclassical topics due to needing agreed upon models (limiting topics)

# Design vs Model *within* Design

Confusingly, within the broadly design tradition we will often now hear about “design vs. model” identification. What?

- **Design-design**: emphasizes randomization for identification which is inside RCTs, IV, even matching (i.e., conditional independence)
- **Design-model**: restricts the “behavior” of unobserved potential outcomes through appeals to parallel trends (DiD), smoothness (RDD), factor models (synthetic control)

# Introduction to Counterfactuals

- Aliens come and orbit earth, see sick people in hospitals and conclude “doctors are hurting people”
- They kill the doctors, unplug patients from machines, throw open the doors – many patients inexplicably die
- Ridiculous to us but only because we know what hospitals are – they don’t
- Consider this: aren’t we the aliens in our research?
- Three types of errors

# #1: Correlation and causality are different

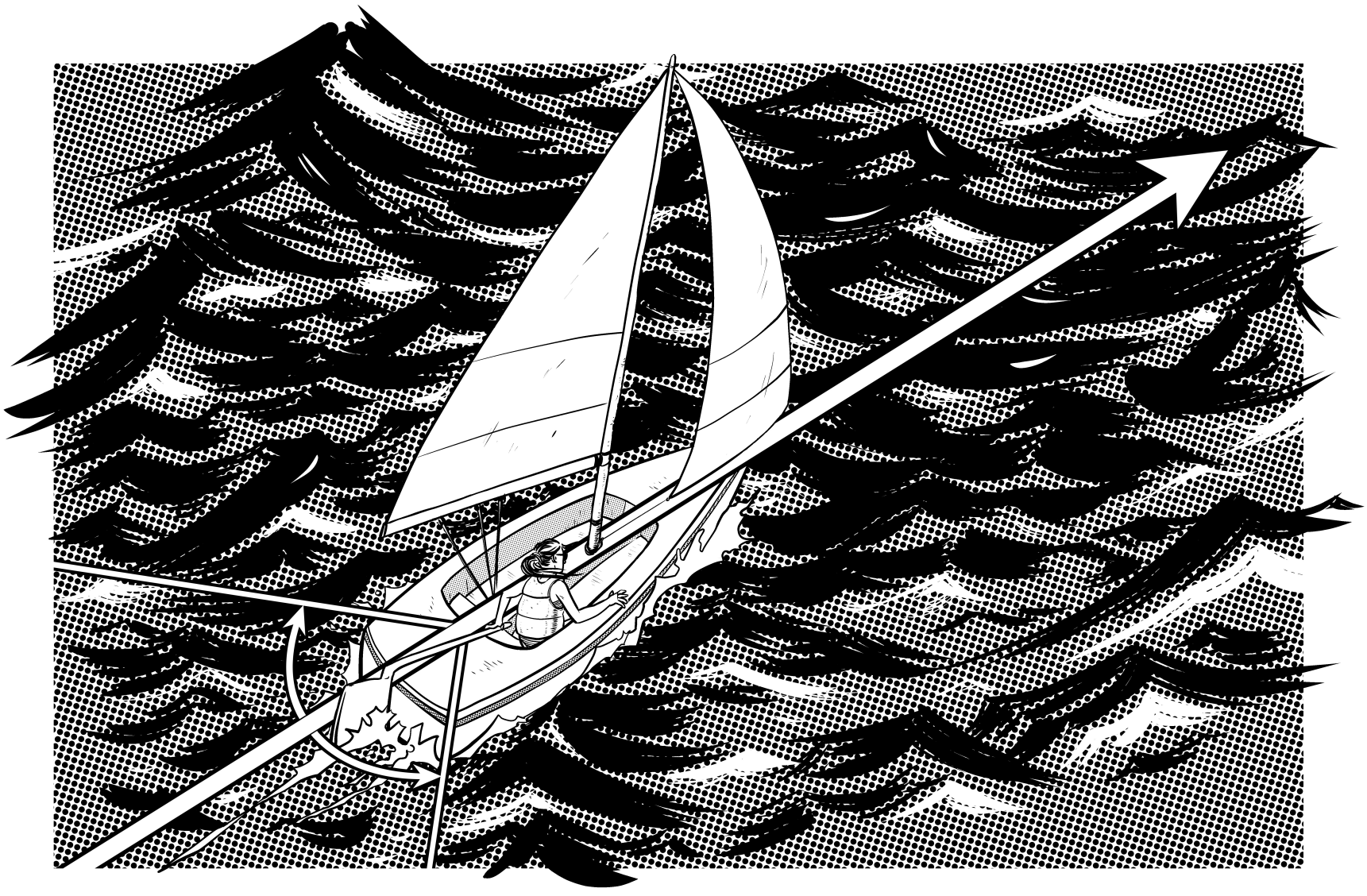
Causal is one unit, correlation is many units

- Causal question: “If a doctor puts a patient on a ventilator (D), will her covid symptoms (Y) improve?”
- Correlation question:

$$\frac{Cov(D,Y)}{\sqrt{Var_D}\sqrt{Var_Y}}$$

# #2: Coming first may not mean causality!

- Every morning the rooster crows and then the sun rises
- Did the rooster cause the sun to rise? Or did the sun cause the rooster to crow?
- What if cat killed the rooster?
- *Post hoc ergo propter hoc*: “after this, therefore, because of this”



# #3: No correlation does not mean no causality!

- A sailor sails her sailboat across a lake
- Wind blows, and she perfectly counters by turning the rudder
- The same aliens observe from space and say “Look at the way she’s moving that rudder back and forth but going in a straight line. That rudder is broken.” So they send her a new rudder
- They’re wrong but why are they wrong? There is, after all, no correlation
- Example: Fed and open market operations

# Potential outcomes notation

- Let the treatment be a binary variable:

$$D_{i,t} = \begin{cases} 1 \text{ if hospitalized at time } t \\ 0 \text{ if not hospitalized at time } t \end{cases}$$

  where $i$ indexes an individual observation, such as a person

- Potential outcomes:

$$Y^j_{i,t} = \begin{cases} 1 \text{ health if hospitalized at time } t \\ 0 \text{ health if not hospitalized at time } t \end{cases}$$

  where $j$ indexes a counterfactual state of the world

# Moving between worlds

- I'll drop $t$ subscript, but note – these are potential outcomes for the same person at the exact same moment in time
- A potential outcome $Y^1$ is not the historical outcome $Y$ either conceptually or notationally
- Potential outcomes are hypothetical states of the world but historical outcomes are ex post realizations
- Major philosophical move here: go from the potential worlds to the actual (historical) world based on your treatment assignment

# Important definitions

## Definition 1: Individual treatment effect

The individual treatment effect, $\delta_i$, equals $Y_i^1 - Y_i^0$

## Definition 2: Average treatment effect (ATE)

The average treatment effect is the population average of all $i$ individual treatment effects

$$\begin{aligned} E[\delta_i] &= E[Y_i^1 - Y_i^0] \\ &= E[Y_i^1] - E[Y_i^0] \end{aligned}$$

## Definition 3: Switching equation

An individual's observed health outcomes, $Y$, is determined by treatment assignment, $D_i$, and corresponding potential outcomes:

$$\begin{aligned} Y_i &= D_i Y_i^1 + (1 - D_i) Y_i^0 \\ Y_i &= \begin{cases} Y_i^1 \text{ if } D_i = 1 \\ Y_i^0 \text{ if } D_i = 0 \end{cases} \end{aligned}$$

# So what's the problem?

## Definition 4: Fundamental problem of causal inference

If you need both potential outcomes to know causality with certainty, then since it is impossible to observe both $Y_i^1$ and $Y_i^0$ for the same individual, $\delta_i$, is *unknowable*.

# Conditional Average Treatment Effects

## Definition 5: Average Treatment Effect on the Treated (ATT)

The average treatment effect on the treatment group is equal to the average treatment effect conditional on being a treatment group member:

$$\begin{aligned} E[\delta|D=1] &= E[Y^1 - Y^0|D=1] \\ &= E[Y^1|D=1] - E[Y^0|D=1] \end{aligned}$$

## Definition 6: Average Treatment Effect on the Untreated (ATU)

The average treatment effect on the untreated group is equal to the average treatment effect conditional on being untreated:

$$\begin{aligned} E[\delta|D=0] &= E[Y^1 - Y^0|D=0] \\ &= E[Y^1|D=0] - E[Y^0|D=0] \end{aligned}$$

# Causality and comparisons

- What will we do? We will make comparisons using groups of observations with quantitative data
- But not all comparisons are equal
  - → Aliens compared patients to non-patients
  - → Does the ventilator make someone have severe COVID symptoms? Or are they sick with COVID symptoms, and that's why they are on a ventilator?
- What are we actually measuring if we compare average health outcomes for those on vents (treatment) versus those who aren't (control)?

## Definition 7: Simple difference in mean outcomes (SDO)

A simple difference in mean outcomes (SDO) can be approximated by the sample averages:

$$\begin{aligned} SDO &= E[Y^1|D=1] - E[Y^0|D=0] \\ &= E[Y|D=1] - E[Y|D=0] \end{aligned}$$

I tend to use expectation operators $E[.]$ but note we are using samples $E_N(.)$

# SDO and the ATE

- SDO is calculated with data
- ATE cannot be calculated with data because it is missing counterfactuals (missing data problem)
- SDO is an *estimate*, whereas ATE is a *parameter*
- SDO is an estimate of the ATE but can be biased
- When is SDO biased and when is it unbiased?

# Potentially biased comparisons

## Decomposition of the SDO

The SDO can be decomposed into the sum of three parts:

$$
\begin{aligned}
E[Y^1|D=1] - E[Y^0|D=0] \quad &= \quad ATE \\
&\quad + E[Y^0|D=1] - E[Y^0|D=0] \\
&\quad + (1-\pi)(ATT - ATU)
\end{aligned}
$$

Seeing is believing so let's work through this identity!

## Use LIE to decompose ATE into the sum of four conditional average expectations

$$\begin{aligned}
\text{ATE} &= E[Y^1] - E[Y^0] \\
&= \{\pi E[Y^1|D=1] + (1-\pi)E[Y^1|D=0]\} \\
&\quad -\{\pi E[Y^0|D=1] + (1-\pi)E[Y^0|D=0]\}
\end{aligned}$$

## Substitute letters for expectations

$$\begin{aligned}
E[Y^1|D=1] &= a \\
E[Y^1|D=0] &= b \\
E[Y^0|D=1] &= c \\
E[Y^0|D=0] &= d \\
\text{ATE} &= e
\end{aligned}$$

## Rewrite ATE

$$e = \{\pi a + (1-\pi)b\} - \{\pi c + (1-\pi)d\}$$

## Move SDO terms to LHS

$$
\begin{aligned}
e &= \{\pi a + (1-\pi)b\} - \{\pi c + (1-\pi)d\} \\
e &= \pi a + b - \pi b - \pi c - d + \pi d \\
e &= \pi a + b - \pi b - \pi c - d + \pi d + (\mathbf{a} - \mathbf{a}) + (\mathbf{c} - \mathbf{c}) + (\mathbf{d} - \mathbf{d}) \\
0 &= e - \pi a - b + \pi b + \pi c + d - \pi d - \mathbf{a} + \mathbf{a} - \mathbf{c} + \mathbf{c} - \mathbf{d} + \mathbf{d} \\
\mathbf{a} - \mathbf{d} &= e - \pi a - b + \pi b + \pi c + d - \pi d + \mathbf{a} - \mathbf{c} + \mathbf{c} - \mathbf{d} \\
\mathbf{a} - \mathbf{d} &= e + (\mathbf{c} - \mathbf{d}) + \mathbf{a} - \pi a - b + \pi b - \mathbf{c} + \pi c + d - \pi d \\
\mathbf{a} - \mathbf{d} &= e + (\mathbf{c} - \mathbf{d}) + (1-\pi)a - (1-\pi)b + (1-\pi)d - (1-\pi)c \\
\mathbf{a} - \mathbf{d} &= e + (\mathbf{c} - \mathbf{d}) + (1-\pi)(a-c) - (1-\pi)(b-d)
\end{aligned}
$$

## Substitute conditional means

$$
\begin{aligned}
E[Y^1|D=1] - E[Y^0|D=0] &= \text{ATE} \\
&\quad + (E[Y^0|D=1] - E[Y^0|D=0]) \\
&\quad + (1-\pi)(\{E[Y^1|D=1] - E[Y^0|D=1]\} \\
&\quad - (1-\pi)\{E[Y^1|D=0] - E[Y^0|D=0]\}) \\
E[Y^1|D=1] - E[Y^0|D=0] &= ATE \\
&\quad + (E[Y^0|D=1] - E[Y^0|D=0]) \\
&\quad + (1-\pi)(ATT - ATU)
\end{aligned}
$$

# Decomposition of difference in means

$$\underbrace{E_N[y_i|d_i=1] - E_N[y_i|d_i=0]}_{\text{SDO}} = \underbrace{E[Y^1] - E[Y^0]}_{\text{Average Treatment Effect}} + \underbrace{E[Y^0|D=1] - E[Y^0|D=0]}_{\text{Selection bias}} + \underbrace{(1-\pi)(ATT - ATU)}_{\text{Heterogenous treatment effect bias}}$$

where $E_N[Y|D=1] \rightarrow E[Y^1|D=1]$, $E_N[Y|D=0] \rightarrow E[Y^0|D=0]$ and $(1-\pi)$ is the share of the population in the control group.

# Independence

## Independence assumption

Treatment is assigned to a population independent of that population's potential outcomes

$$(Y^0, Y^1) \perp\!\!\!\perp D$$

This is random or quasi-random assignment and ensures mean potential outcomes for the treatment group and control group are the same. Also ensures other variables are distributed the same for a large sample.

$$\begin{aligned} E[Y^0|D=1] &= E[Y^0|D=0] \\ E[Y^1|D=1] &= E[Y^1|D=0] \end{aligned}$$

# Random Assignment Solves the Selection Problem

$$\underbrace{E_N[y_i|d_i=1] - E_N[y_i|d_i=0]}_{\text{SDO}} = \underbrace{E[Y^1] - E[Y^0]}_{\text{Average Treatment Effect}} + \underbrace{E[Y^0|D=1] - E[Y^0|D=0]}_{\text{Selection bias}} + \underbrace{(1-\pi)(ATT - ATU)}_{\text{Heterogenous treatment effect bias}}$$

- If treatment is independent of potential outcomes, then swap out equations and **selection bias** zeroes out:

$$E[Y^0|D=1] - E[Y^0|D=0] = 0$$

## Random Assignment Solves the Heterogenous Treatment Effects

- How does randomization affect heterogeneity treatment effects bias from the third line? Rewrite definitions for ATT and ATU:

$$\text{ATT} = E[Y^1|D=1] - E[Y^0|D=1]$$
$$\text{ATU} = E[Y^1|D=0] - E[Y^0|D=0]$$

- Rewrite the third row bias after $1-\pi$:

$$\begin{aligned} ATT - ATU &= \textbf{E[Y}^1 \mid \textbf{D=1]} - E[Y^0|D=1] \\ &\quad -\textbf{E[Y}^1 \mid \textbf{D=0]} + E[Y^0|D=0] \\ &= 0 \end{aligned}$$

- If treatment is independent of potential outcomes, then:

$$\begin{aligned} E_N[y_i|d_i=1] - E_N[y_i|d_i=0] &= E[Y^1] - E[Y^0] \\ SDO &= ATE \end{aligned}$$

# SUTVA

- Potential outcomes model places a limit on what we can measure: the “stable unit-treatment value assumption” . Horrible acronym.
    1. **S**: ***s****table*
    2. **U**: across all ***u****nits*, or the population
    3. **TV**: ***t****reatment-value* (“treatment effect”, “causal effect”)
    4. **A**: ***a****ssumption*
- As this is a bit of a pregnant concept, let’s go slow

# SUTVA: Unit-level assignment only

- Most people, if they know of SUTVA, tend to associate with one of its elements not its core definition
- It's core definition is actually the switching equation:

$$Y_{i,t} = D_{i,t}Y^1_{i,t} + (1 - D_{i,t})Y^0_{i,t}$$

- Notice now the $i$ and $t$ subscripts; think of what that means

# SUTVA: No Anticipation

- A particular unit $i$ at some point in time $t$ assigns potential outcome for unit $i$ at time $t$ to outcome based on *its* contemporanous treatment assignment for the same $i$ unit at the same $t$ time
- Outcomes are **not** someone else's (spillovers), nor on future assignment (anticipation)
- Example: I increase spending based on a future raise I haven't yet gotten

# SUTVA: No Hidden Variation in Treatment

- SUTVA requires each unit receive the same treatment dosage; this is what it means by “stable”
- If we are estimating the effect of vents on covid symptoms, we assume everyone is getting the same kinds of vents more or less.
- Easy to imagine violations if hospital quality, staffing or even the vents themselves vary across treatment group
- Be careful what we are and are not defining as *the treatment*

# SUTVA: No spillovers to other units

- What if putting someone on a ventilator causes someone else to be more or less likely to develop severe covid symptoms?
- Have to think hard about externalities, particularly with transmissible diseases
- SUTVA means that you don't have a problem like this.
- If there are no externalities from treatment, then $\delta_i$ is stable for each $i$ unit regardless of whether someone else receives the treatment too, but herd immunity must be considered when it comes to cures

# SUTVA: Partial equilibrium only

Easier to imagine this with a different example.

- Let's say we estimate a causal effect of early childhood intervention in Texas
- Now President Biden wants to roll it out for the whole United States – will it have the same effect as we found?
- Scaling up a policy can be challenging to predict if there are rising costs of production
- What if expansion requires hiring lower quality teachers just to make classes?
- That's a general equilibrium effect; we only estimated a partial equilibrium effect (external versus internal validity)

# Demand for Learning HIV Status

- Rebecca Thornton implemented an RCT in rural Malawi for her job market paper at Harvard in mid-2000s
- At the time, it was an article of faith that you could fight the HIV epidemic in Africa by encouraging people to get tested; but Thornton wanted to see if this was true
- She randomly assigned cash incentives to people to incentivize learning their HIV status
- Also examined whether learning changed sexual behavior.

# Experimental design

- Respondents were offered a free door-to-door HIV test
- Treatment is randomized vouchers worth between zero and three dollars
- These vouchers were redeemable once they visited a nearby voluntary counseling and testing center (VCT)
- Estimates her models using OLS with controls

# Why Include Control Variables?

- To evaluate experimental data, one may want to add additional controls in the multivariate regression model. So, instead of estimating the prior equation, we might estimate:

$$Y_i = \alpha + \delta D_i + \gamma X_i + \eta_i$$

- There are 2 main reasons for including additional controls in the regression models:
  1. Conditional random assignment. Sometimes randomization is done *conditional* on some observable (e.g., gender, school, districts)
  2. Exogenous controls increase precision. Although control variables $X_i$ are uncorrelated with $D_i$, they may have substantial explanatory power for $Y_i$. Including controls thus reduces variance in the residuals which lowers the standard errors of the regression estimates.

*Table:* Impact of Monetary Incentives and Distance on Learning HIV Results

| | **1** | **2** | **3** | **4** | **5** |
|---|---|---|---|---|---|
| Any incentive | 0.431*** | 0.309*** | 0.219*** | 0.220*** | 0.219 *** |
| | (0.023) | (0.026) | (0.029) | (0.029) | (0.029) |
| Amount of incentive | | 0.091*** | 0.274*** | 0.274*** | 0.273*** |
| | | (0.012) | (0.036) | (0.035) | (0.036) |
| Amount of incentive$^2$ | | | $-0.063$*** | $-0.063$*** | $-0.063$*** |
| | | | (0.011) | (0.011) | (0.011) |
| HIV | $-0.055$* | $-0.052$ | $-0.05$ | $-0.058$* | $-0.055$* |
| | (0.031) | (0.032) | (0.032) | (0.031) | (0.031) |
| Distance (km) | | | | $-0.076$*** | |
| | | | | (0.027) | |
| Distance$^2$ | | | | 0.010** | |
| | | | | (0.005) | |
| Controls | Yes | Yes | Yes | Yes | Yes |
| Sample size | 2,812 | 2,812 | 2,812 | 2,812 | 2,812 |
| Average attendance | 0.69 | 0.69 | 0.69 | 0.69 | 0.69 |

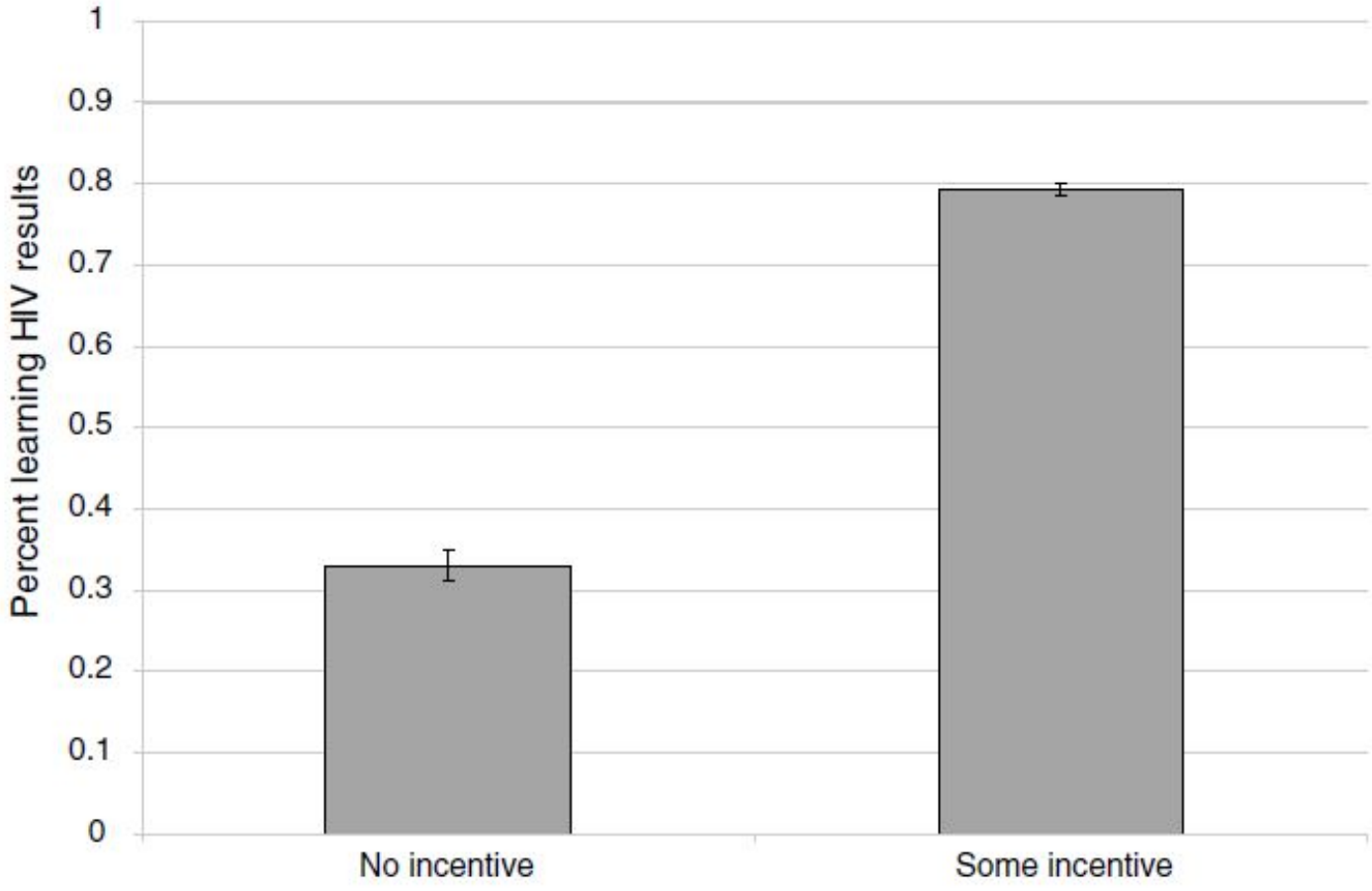



*Figure:* Visual representation of cash transfers on learning HIV test results.

# Results

- Even small incentives were effective
- Any incentive increases learning HIV status by 43% compared to the control (mean 34%)
- Next she looks at the effect that learning HIV status has on risky sexual behavior

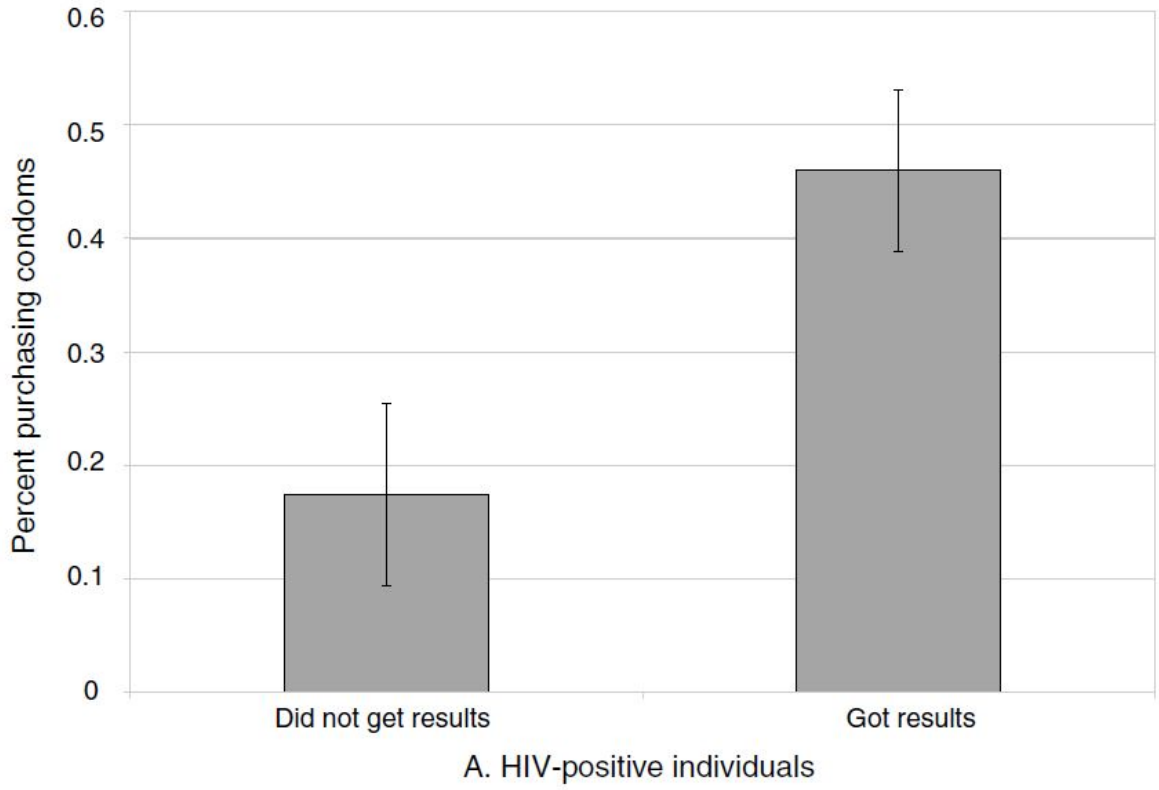



*Figure:* Visual representation of cash transfers on condom purchases for HIV positive individuals.

*Table:* Reactions to Learning HIV Results among Sexually Active at Baseline

| **Dependent variables:** | **Bought condoms** | | **Number of condoms bought** | |
|---|---|---|---|---|
| | **OLS** | **IV** | **OLS** | **IV** |
| Got results | −0.022 | −0.069 | −0.193 | −0.303 |
| | (0.025) | (0.062) | (0.148) | (0.285) |
| Got results × HIV | 0.418*** | 0.248 | 1.778*** | 1.689** |
| | (0.143) | (0.169) | (0.564) | (0.784) |
| HIV | −0.175** | −0.073 | −0.873 | −0.831 |
| | (0.085) | (0.123) | (0.275) | (0.375) |
| Controls | Yes | Yes | Yes | Yes |
| Sample size | 1,008 | 1,008 | 1,008 | 1,008 |
| Mean | 0.26 | 0.26 | 0.95 | 0.95 |

# Results

- For those who were HIV+ and got their test results, 42% more likely to buy condoms (but shrinks and becomes insignificant at conventional levels with IV).
- Number of condoms bought – very small. HIV+ respondents who learned their status bought 2 more condoms

# Randomization inference and causal inference

- "In randomization-based inference, uncertainty in estimates arises naturally from the random assignment of the treatments, rather than from hypothesized sampling from a large population." (Athey and Imbens 2017)
- Athey and Imbens is part of growing trend of economists using randomization-based methods for doing causal inference
- Unclear (to me) why we are hearing more and more about randomization inference, but we are.
- Could be due to improved computational power and/or the availability of large data instead of samples?

# Lady tasting tea experiment

- Ronald Aylmer Fisher (1890-1962)
  - → Two classic books on statistics: *Statistical Methods for Research Workers* (1925) and *The Design of Experiments* (1935), as well as a famous work in genetics, *The Genetical Theory of Natural Science*
  - → Developed many fundamental notions of modern statistics including the theory of randomized experimental design.

# Lady tasting tea

- Muriel Bristol (1888-1950)
  - → A PhD scientist back in the days when women weren't PhD scientists
  - → Worked with Fisher at the Rothamsted Experiment Station (which she established) in 1919
  - → During afternoon tea, Muriel claimed she could tell from taste whether the milk was added to the cup before or after the tea
  - → Scientists were incredulous, but Fisher was inspired by her strong claim
  - → He devised a way to test her claim which she passed using randomization inference

# Description of the tea-tasting experiment

- Original claim: Given a cup of tea with milk, Bristol claims she can discriminate the order in which the milk and tea were added to the cup
- Experiment: To test her claim, Fisher prepares 8 cups of tea – 4 **milk then tea** and 4 **tea then milk** – and presents each cup to Bristol for a taste test
- Question: How many cups must Bristol correctly identify to convince us of her unusual ability to identify the order in which the milk was poured?
- Fisher's sharp null: Assume she can't discriminate. Then what's the likelihood that random chance was responsible for her answers?

# Choosing subsets

- The lady performs the experiment by selecting $4$ cups, say, the ones she claims to have had the tea poured first.

$$\binom{n}{k} = \frac{n!}{k!\,(n-k)!}$$

- "8 choose 4" – $\binom{8}{4}$ – ways to choose 4 cups out of 8
  - → Numerator is $8 \times 7 \times 6 \times 5 = 1,680$ ways to choose a first cup, a second cup, a third cup, and a fourth cup, in order.
  - → Denominator is $4 \times 3 \times 2 \times 1 = 24$ ways to order $4$ cups.

# Choosing subsets

- There are $70$ ways to choose $4$ cups out of $8$, and therefore a 1.4% probability of producing the correct answer by chance

$$\frac{24}{1680} = 1/70 = 0.014.$$

- For example, the probability that she would correctly identify all $4$ cups is $\frac{1}{70}$

# Statistical significance

- Suppose the lady correctly identifies all 4 cups. Then . . .
  1. Either she has no ability, and has chosen the correct 4 cups purely by chance, or
  2. She has the discriminatory ability she claims.
- Since choosing correctly is highly unlikely in the first case (one chance in $70$), the second seems plausible.
- Bristol actually got all four correct
- I wonder if seeing this, any of the scientists present changed their mind

# Null hypothesis

- In this example, the null hypothesis is the hypothesis that the lady has no special ability to discriminate between the cups of tea.
- We can never prove the null hypothesis, but the data may provide evidence to reject it.
- In most situations, rejecting the null hypothesis is what we hope to do.

# Null hypothesis of no effect

- Randomization inference allows us to make probability calculations revealing whether the treatment assignment was “unusual”
- Fisher’s sharp null is when entertain the possibility that no unit has a treatment effect
- This allows us to make “exact” p-values which do not depend on large sample approximations
- It also means the inference is not dependent on any particular distribution (e.g., Gaussian); sometimes called nonparametric

# Sidebar: bootstrapping is different

- Sometimes people confuse randomization inference with bootstrapping
- Bootstrapping randomly draws a percent of the total observations for estimation; “uncertainty over the sample”
- Randomization inference randomly reassigns the treatment; “uncertainty over treatment assignment”

(Thanks to Jason Kerwin for helping frame the two against each other)

# 6-step guide to randomization inference

The following is from Imbens and Rubin's textbook on causal inference, as well as Matthew Blackwell's helpful lectures

1. Choose a sharp null hypothesis (e.g., no treatment effects)
2. Calculate a test statistic ($T$ is a scalar based on $D$ and $Y$)
3. Then pick a randomized treatment vector $\tilde{D}_1$
4. Calculate the test statistic associated with $(\tilde{D}, Y)$
5. Repeat steps 3 and 4 for all possible combinations to get $\tilde{T} = \{\tilde{T}_1, \ldots, \tilde{T}_K\}$
6. Calculate exact p-value as $p = \frac{1}{K} \sum_{k=1}^{K} I(\tilde{T}_k \geq T)$

# Pretend experiment

*Table:* Pretend DBT intervention for some homeless population

| Name | D | Y | $Y^0$ | $Y^1$ |
|---|---|---|---|---|
| Andy | 1 | 10 | . | 10 |
| Ben | 1 | 5 | . | 5 |
| Chad | 1 | 16 | . | 16 |
| Daniel | 1 | 3 | . | 3 |
| Edith | 0 | 5 | 5 | . |
| Frank | 0 | 7 | 7 | . |
| George | 0 | 8 | 8 | . |
| Hank | 0 | 10 | 10 | . |

For concreteness, assume a program where we pay homeless people $15 to take dialectical behavioral therapy (DBT). Outcomes are some measure of mental health 0-20 with higher scores being improvements in mental health symptoms.

# Step 1: Sharp null of no effect

## Fisher's Sharp Null Hypothesis

$$H_0 : \delta_i = Y_i^1 - Y_i^0 = 0 \; \forall i$$

- Assuming no effect means any test statistic is due to chance
- Neyman and Fisher test statistics were different – Fisher was exact, Neyman was not
- Neyman's null was no average treatment effect (ATE=0). If you have a treatment effect of 5 and I have a treatment effect of -5, our ATE is zero. This is not the sharp null even though it also implies a zero ATE

# More sharp null

- Since under the Fisher sharp null $\delta_i = 0$, it means each unit's potential outcomes under both states of the world are the same
- We therefore know each unit's missing counterfactual
- The randomization we will perform will cycle through all treatment assignments under a null well treatment assignment doesn't matter because all treatment assignments are associated with a null of zero unit treatment effects
- We are looking for evidence *against* the null

# Step 1: Fisher's sharp null and missing potential outcomes

*Table:* Missing potential outcomes are no longer missing

| Name | D | Y | $Y^0$ | $Y^1$ |
|---|---|---|---|---|
| Andy | 1 | 10 | **10** | 10 |
| Ben | 1 | 5 | **5** | 5 |
| Chad | 1 | 16 | **16** | 16 |
| Daniel | 1 | 3 | **3** | 3 |
| Edith | 0 | 5 | 5 | **5** |
| Frank | 0 | 7 | 7 | **7** |
| George | 0 | 8 | 8 | **8** |
| Hank | 0 | 10 | 10 | **10** |

Fisher sharp null allows us to **fill in** the missing counterfactuals bc under the null there's zero treatment effect at the unit level. This guarantees zero ATE, but is different in formulation than Neyman's null

# Step 2: Choosing a test statistic

## Test Statistic

A test statistic $T(D, Y)$ is a scalar quantity calculated from the treatment assignments $D$ and the observed outcomes $Y$

- By scalar, I just mean it's a number (vs. a function) measuring some relationship between $D$ and $Y$
- Ultimately there are many tests to choose from; I'll review a few later
- If you want a test statistic with high statistical power, you need large values when the null is false, and small values when the null is true (i.e., *extreme*)

# Simple difference in means

- Consider the absolute SDO from earlier

$$\delta_{SDO} = \left| \frac{1}{N_T} \sum_{i=1}^{N} D_i Y_i - \frac{1}{N_C} \sum_{i=1}^{N} (1 - D_i) Y_i \right|$$

- Larger values of $\delta_{SDO}$ are evidence *against* the sharp null
- Good estimator for constant, additive treatment effects and relatively few outliers in the potential outcomes

# Step 2: Calculate test statistic, $T(D, Y)$

*Table:* Calculate $T$ using $D$ and $Y$

| Name | D | Y | $Y^0$ | $Y^1$ | $\delta_i$ |
|---|---|---|---|---|---|
| Andy | **1** | **10** | 10 | 10 | 0 |
| Ben | **1** | **5** | 5 | 5 | 0 |
| Chad | **1** | **16** | 16 | 16 | 0 |
| Daniel | **1** | **3** | 3 | 3 | 0 |
| Edith | **0** | **5** | 5 | 5 | 0 |
| Frank | **0** | **7** | 7 | 7 | 0 |
| George | **0** | **8** | 8 | 8 | 0 |
| Hank | **0** | **10** | 10 | 10 | 0 |

We'll start with this simple the simple difference in means test statistic, $T(D, Y)$: $\delta_{SDO} = 34/4 - 30/4 = 1$

# Steps 3-5: Null randomization distribution

- Randomization steps reassign treatment assignment for every combination, calculating test statistics each time, to obtain the entire distribution of counterfactual test statistics
- The key insight of randomization inference is that under Fisher's sharp null, the treatment assignment shouldn't matter
- Ask yourself:
  - → if there is no unit level treatment effect, can you picture a distribution of counterfactual test statistics?
  - → and if there is no unit level treatment effect, what must average counterfactual test statistics equal?

# Step 6: Calculate “exact” p-values

- Question: how often would we get a test statistic as big or bigger as our “real” one if Fisher’s sharp null was true?
- This can be calculated “easily” (sometimes) once we have the randomization distribution from steps 3-5
  - → The number of test statistics ($t(D, Y)$) bigger than the observed divided by total number of randomizations

$$Pr(T(D, Y) \geq T(\tilde{D}, Y|\delta = 0)) = \frac{\sum_{D \in \Omega} I(T(D, Y) \leq T(\tilde{D}, Y)}{K}$$

# First permutation (holding $N_T$ fixed)

| Name | $\tilde{D}_2$ | Y | $Y^0$ | $Y^1$ |
|---|---|---|---|---|
| Andy | 1 | 10 | 10 | 10 |
| Ben | 0 | 5 | 5 | 5 |
| Chad | 1 | 16 | 16 | 16 |
| Daniel | 1 | 3 | 3 | 3 |
| Edith | 0 | 5 | 5 | 5 |
| Frank | 1 | 7 | 7 | 7 |
| George | 0 | 8 | 8 | 8 |
| Hank | 0 | 10 | 10 | 10 |

$$\tilde{T}_1 = |36/4 - 28/4| = 9 - 7 = 2$$

# Second permutation (again holding $N_T$ fixed)

| Name | $\tilde{D}_3$ | Y | $Y^0$ | $Y^1$ |
|---|---|---|---|---|
| Andy | 1 | 10 | 10 | 10 |
| Ben | 0 | 5 | 5 | 5 |
| Chad | 1 | 16 | 16 | 16 |
| Daniel | 1 | 3 | 3 | 3 |
| Edith | 0 | 5 | 5 | 5 |
| Frank | 0 | 7 | 7 | 7 |
| George | 1 | 8 | 8 | 8 |
| Hank | 0 | 10 | 10 | 10 |

$$T_{rank} = |36/4 - 27/4| = 9 - 6.75 = 2.25$$

# Sidebar: Should it be 4 treatment groups each time?

- In this experiment, I've been using the same $N_T$ *under the assumption* that $N_T$ had been fixed when the experiment was drawn.
- But if the original treatment assignment had been generated by something like a Bernoulli distribution (e.g., coin flips over every unit), then you should be doing a complete permutation that is also random in this way
- This means that for 8 units, sometimes you'd have 1 treated, or even 8
- Correct inference requires you know the original data generating process

# Randomization distribution

| Assignment | $D_1$ | $D_2$ | $D_3$ | $D_4$ | $D_5$ | $D_6$ | $D_7$ | $D_8$ | $\lvert T_i \rvert$ |
|---|---|---|---|---|---|---|---|---|---|
| True $D$ | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 1 |
| $\tilde{D}_2$ | 1 | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 2 |
| $\tilde{D}_3$ | 1 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 2.25 |
| ... | | | | | | | | | |

# Step 2: Other test statistics

- The simple difference in means is fine when effects are additive, and there are few outliers in the data
- But outliers create more variation in the randomization distribution
- A good test statistic is the one that best fits your data.
- Some test statistics will have weird properties in the randomization as we'll see in synthetic control.
- What are some alternative test statistics?

# Transformations

- What if there was a constant multiplicative effect: $Y_i^1 / Y_i^0 = C$?
- Difference in means will have low power to detect this alternative hypothesis
- So we transform the observed outcome using the natural log:

$$T_{log} = \left| \frac{1}{N_T} \sum_{i=1}^{N} D_i ln(Y_i) - \frac{1}{N_C} \sum_{i=1}^{N} (1 - D_i) ln(Y_i) \right|$$

- This is useful for skewed distributions of outcomes

# Difference in medians/quantiles

- We can protect against outliers using other test statistics such as the difference in quantiles
- Difference in medians:

$$T_{median} = |median(Y_T) - median(Y_C)|$$

- We could also estimate the difference in quantiles at any point in the distribution (e.g., 25th or 75th quantile)

# Rank test statistics

- Basic idea is rank the outcomes (higher values of $Y_i$ are assigned higher ranks)
- Then calculate a test statistic based on the transformed ranked outcome (e.g., mean rank)
- Useful with continuous outcomes, small datasets and/or many outliers

# Rank statistics formally

- Rank is the domination of others (including oneself):

$$\tilde{R} = \tilde{R}_i(Y_1, \ldots, Y_N) = \sum_{j=1}^{N} I(Y_j \leq Y_i)$$

- Normalize the ranks to have mean 0

$$\tilde{R}_i = \tilde{R}_i(Y_1, \ldots, Y_N) = \sum_{j=1}^{N} I(Y_j \leq Y_i) - \frac{N+1}{2}$$

- Calculate the absolute difference in average ranks:

$$T_{rank} = |\overline{R}_T - \overline{R}_C| = \left| \frac{\sum_{i:D_i=1} R_i}{N_T} - \frac{\sum_{i:D_i=0} R_i}{N_C} \right|$$

- Minor adjustment (averages) for ties

# Randomization distribution

| Name | D | Y | $Y^0$ | $Y^1$ | Rank | $R_i$ |
|---|---|---|---|---|---|---|
| Andy | 1 | 10 | **10** | 10 | 6.5 | 2 |
| Ben | 1 | 5 | **5** | 5 | 2.5 | -2 |
| Chad | 1 | 16 | **16** | 16 | 8 | 3.5 |
| Daniel | 1 | 3 | **3** | 3 | 1 | -3.5 |
| Edith | 0 | 5 | 5 | **5** | 2.5 | -2 |
| Frank | 0 | 7 | 7 | **7** | 4 | -0.5 |
| George | 0 | 8 | 8 | **8** | 5 | 0.5 |
| Hank | 0 | 10 | 10 | **10** | 6.5 | 2 |

$$T_{rank} = |0 - 0| = 0$$

# Effects on outcome distributions

- Focused so far on “average” differences between groups.
- Kolmogorov-Smirnov test statistics is based on the difference in the distribution of outcomes
- Empirical cumulative distribution function (eCDF):

$$\widehat{F}_C(Y) = \frac{1}{N_C} \sum_{i:D_i=0} 1(Y_i \leq Y)$$

$$\widehat{F}_T(Y) = \frac{1}{N_T} \sum_{i:D_i=1} 1(Y_i \leq Y)$$

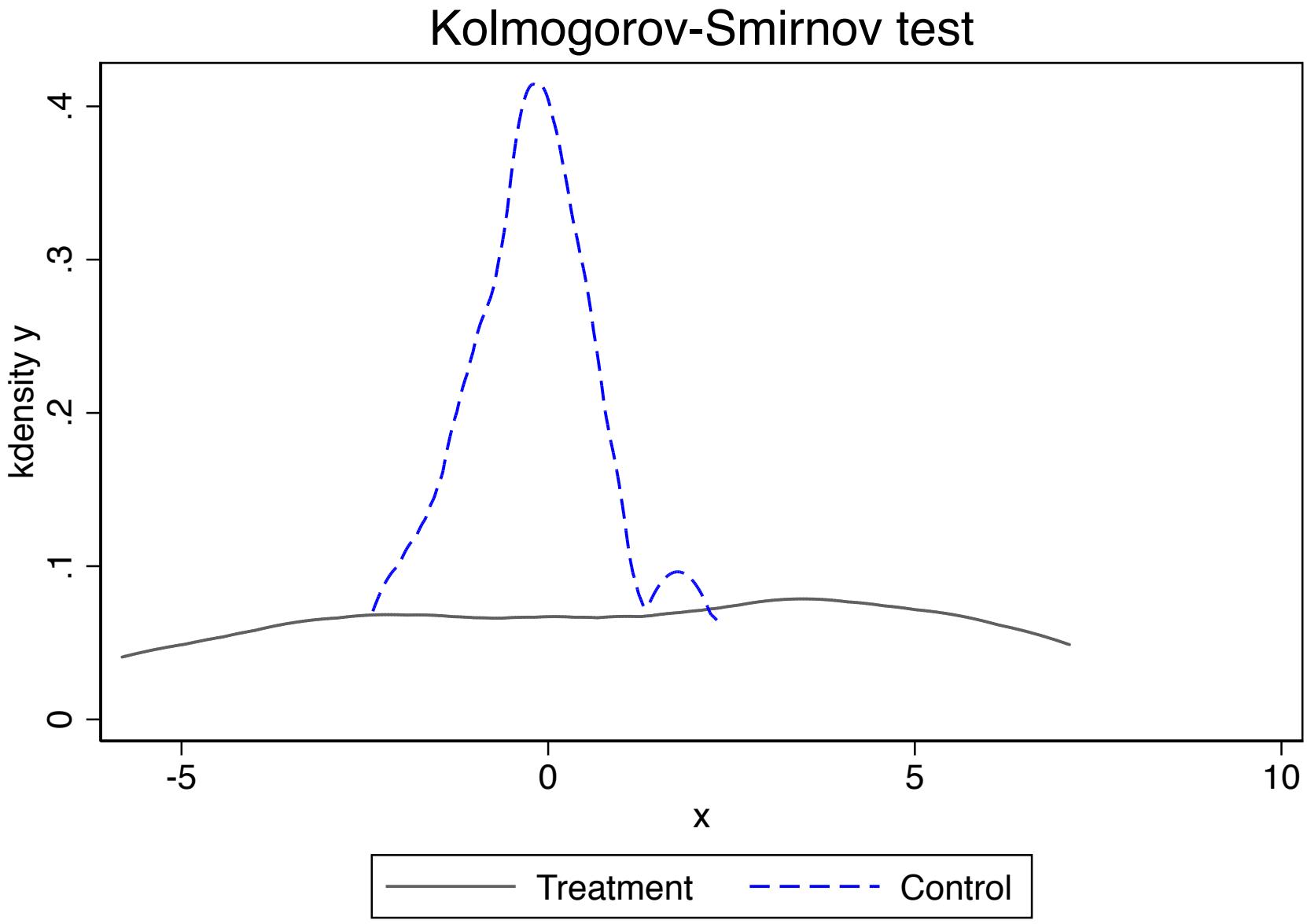
- Proportion of observed outcomes below a chosen value for treated and control separately
- If two distributions are the same, then $\widehat{F}_C(Y) = \widehat{F}_T(Y)$

# Kolmogorov-Smirnov statistic

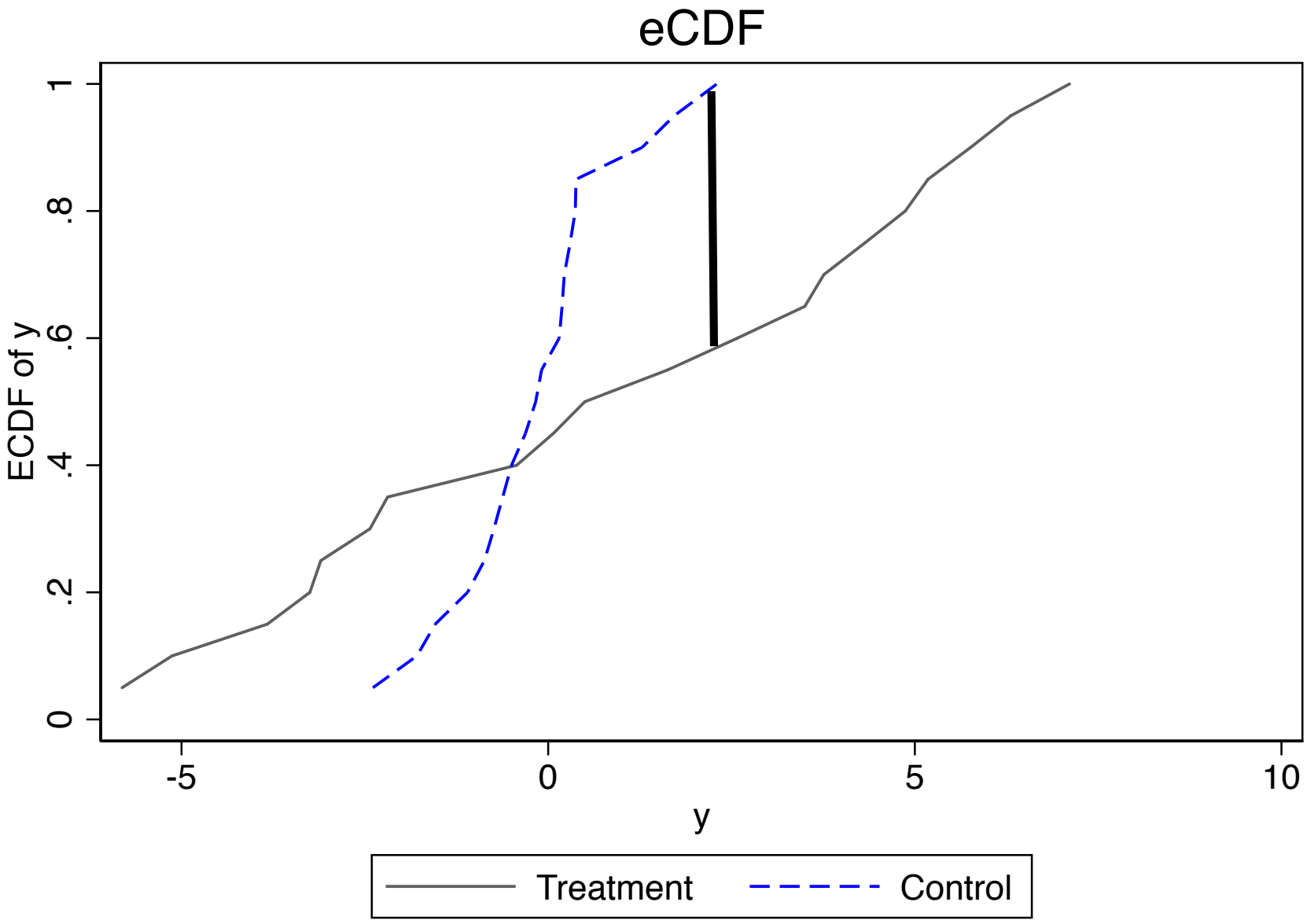
- Test statistics are scalars not functions
- eCDFs are functions, not scalars
- Solution: use the maximum discrepancy between the two eCDFs:

$$T_{KS} = max|\widehat{F}_T(Y_i) - \widehat{F}_C(Y_i)|$$

# Kernel density by group status



# eCDFs by treatment status and test statistic



# KS Test Statistic

| Treatment | D | Exact P-value |
|---|---|---|
| K-S | 0.4500 | 0.034 |

Max distance is 0.45. Exact $p$ is 0.034.

# One-sided or two-sided?

- So far, we have defined all test statistics as absolute values
- We are testing against a two-sided alternative hypothesis

$$
\begin{aligned}
H_0 &: \delta_i = 0 \ \forall i \\
H_1 &: \delta_i \neq 0 \ \texttt{for some} \ i
\end{aligned}
$$

- What about a one-sided alternative

$$
\begin{aligned}
H_0 &: \delta_i = 0 \ \forall i \\
H_1 &: \delta_i > 0 \ \texttt{for some} \ i
\end{aligned}
$$

- For these, use a test statistic that is bigger under the alternative:

$$T_{diff}* = \overline{Y}_T - \overline{Y}_C$$

# Small vs. Modest Sample Sizes are non-trivial

Computing the exact randomization distribution is not always feasible (Wolfram Alpha)

- $N = 6$ and $N_T = 3$ gives us 20 assignment vectors
- $N = 8$ and $N_T = 4$ gives us 70 assignment vectors
- $N = 10$ and $N_T = 5$ gives us 252 assignment vectors
- $N = 20$ and $N_T = 10$ gives us 184,756 assignment vectors
- $N = 50$ and $N_T = 25$ gives us $1.2641061 \times 10^{14}$ assignment vectors

Exact $p$ calculations are not realistic bc the number of assignments explodes at even modest size

# Approximate p-values

These have been “exact” tests when they use every possible combination of $D$

- When you can’t use every combination, then you can get *approximate* p-values from a simulation (TBD)
- With a rejection threshold of $\alpha$ (e.g., 0.05), randomization inference test will falsely reject less than $100 \times \alpha\%$ of the time

# Approximate $p$ values

- Use simulation to get approximate $p$-values
  - $\rightarrow$ Take $K$ samples from the treatment assignment space
  - $\rightarrow$ Calculate the randomization distribution in the $K$ samples
  - $\rightarrow$ Tests no longer exact, but bias is under your control (increase $K$)
- Imbens and Rubin show that $p$ values converge to stable $p$ values pretty quickly (in their example after 1000 replications)

# Sample dataset

Let’s do this now with Thornton’s data. You can replicate that using thorton_ri.do or thornton_ri.R

# Thornton's experiment

| ATE | Iteration | Rank | $p$ | no. trials |
|---|---|---|---|---|
| 0.45 | 1 | 1 | 0.01 | 100 |
| 0.45 | 1 | 1 | 0.002 | 500 |
| 0.45 | 1 | 1 | 0.001 | 1000 |

*Table:* Estimated $p$-value using different number of trials.

# Including covariate information

- Let $X_i$ be a pretreatment measure of the outcome
- One way is to use this as a gain score: $Y^{d'} = Y_i^d - X_i$
- Causal effects are the same $Y^{1i} - Y^{0i} = Y_i^1 - Y_i^0$
- But the test statistic is different:

$$T_{gain} = \left| (\overline{Y}_T - \overline{Y}_C) - (\overline{X}_T - \overline{X}_C) \right|$$

- If $X_i$ is strongly predictive of $Y_i^0$, then this could have higher power
  - $\rightarrow$ $Y_{gain}$ will have lower variance under the null
  - $\rightarrow$ This makes it easier to detect smaller effects

# Regression in RI

- We can extend this to use covariates in more complicated ways
- For instance, we can use an OLS regression:

$$Y_i = \alpha + \delta D_i + \beta X_i + \varepsilon$$

- Then our test statistic could be $T_{OLS} = \widehat{\delta}$
- RI is justified even if the model is wrong
  - → OLS is just another way to generate a test statistic
  - → The more the model is "right" (read: predictive of $Y_i^0$), the higher the power $T_{OLS}$ will have
- See if you can do this in Thornton's dataset using the loops and saving the OLS coefficient (or just use `ritest`)

# Roadmap

Counterfactuals and causality
- Causality and models
- Potential outcomes
- Randomization and selection bias
- Randomization inference

Directed Acyclic Graphs
- Graph notation
- Backdoor criterion
- Collider bias
- Front door criterion
- Concluding remarks

## Judea Pearl and DAGs

- Judea Pearl and colleagues in Artificial Intelligence at UCLA developed DAG modeling to create a formalized causal inference methodology
- They causality concepts extremely clear, they provide a map to the estimation strategy, and maybe best of all, they communicate to others what must be true about the data generating process to recover the causal effect

Judea Pearl, 2011 Turing Award winner, drinking his first IPA

# Further reading

1. Pearl (2018) The Book of Why: The New Science of Cause and Effect, Basic Books (*popular*)
2. Morgan and Winship (2014) Counterfactuals and Causal Inference: Methods and Principles for Social Research, Cambridge University Press, 2nd edition (*excellent*)
3. Pearl, Glymour and Jewell (2016) Causal Inference In Statistics: A Primer, Wiley Books (*accessible*)
4. Pearl (2009) Causality: Models, Reasoning and Inference, Cambridge, 2nd edition (*difficult*)
5. Cunningham (2021) Causal Inference: The Mixtape, Yale, 1st edition (*best choice, no question*)

# Design vs. Model

- DAGs tend to be focused more on the theory of treatment assignment in the world
- As such it’s compatible with design-based approaches
- But assumptions in design based approaches tend to emphasize selection into treatment which is not exactly what is meant here

# Causal model

- The causal model is sometimes called the structural model, but for us, I prefer the former as it's less alienating
- Think of this as more connected to the model-based approach discussed earlier
- It's the system of equations describing the relevant aspects of the world
- It necessarily is filled with causal effects associated with some particular comparative statics
- To illustrate, I will assume a Beckerian human capital model

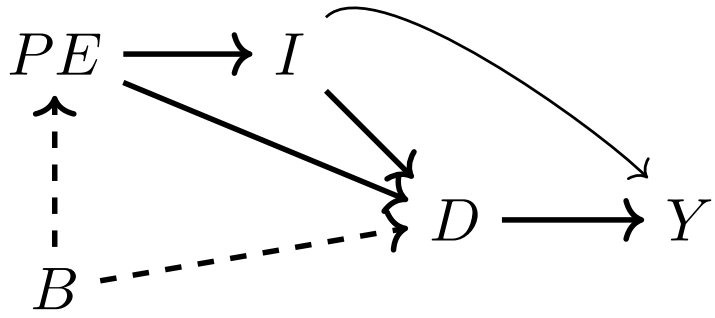
# Human capital model: statements *and* graphs

Let's describe my simplified Beckerian human capital model.

- Individuals maximize utility by choosing consumption and schooling (D) subject to multi-period budget constraint
- Education has current costs but longterm returns
- But people choose different levels of schooling based on a number of things we will call "background" (B) which won't be in the dataset ("unobserved")
- And own-schooling will also be because of parental schooling (PE)
- Finally, wages (Y) are a function of parental schooling

# Becker's human capital causal model

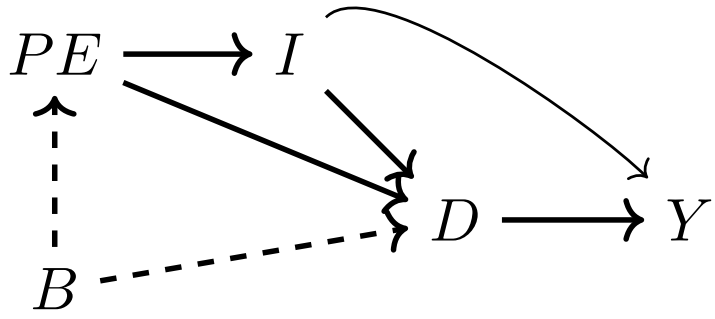
We can represent that causal model visually



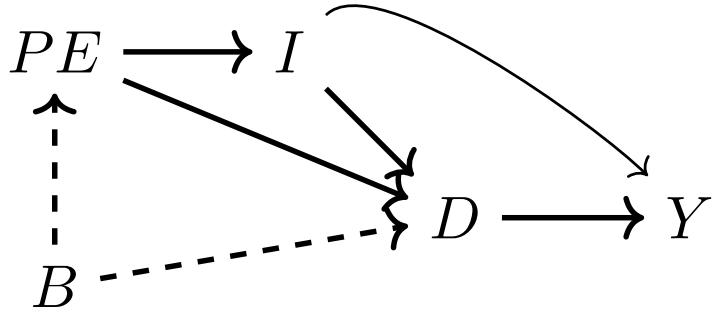

$PE$ is parental education, $B$ is "unobserved background factors (i.e., "ability")", $I$ is family income, $D$ is college education and $Y$ is log wages. The DAG is an approximation of Becker's underlying (causal) human capital model.

# Arrows, but also *missing arrows*

Before we dive into all this notation, couple of things

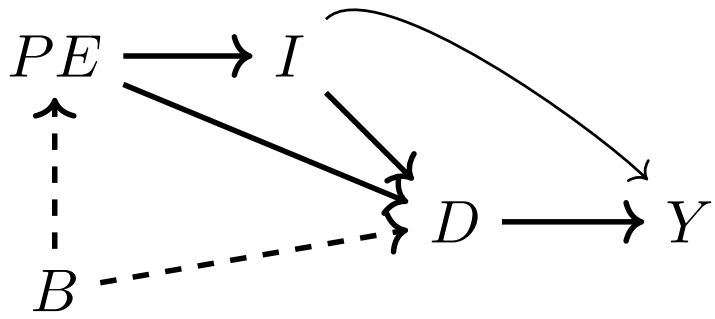



$PE$ and $D$ are caused by $B$. But why doesn't $B$ cause $Y$?? Do you believe this? Why/why not? We can dispute this, but notice – we can see the assumption, which is transparent and communicates the author's beliefs, as well as the needed assumptions in their forthcoming *empirical* model. Every empirical strategy makes assumptions, but oftentimes they are not as transparent to us as this is.




- $B$ is a **parent** of $PE$ and $D$
- $PE$ and $D$ are **descendants** of $B$
- There is a **direct (causal) path** from $D$ to $Y$
- There is a **mediated (causal) path** from $B$ to $Y$ through $D$
- There are four **paths** from $PE$ to $Y$ but none are direct, and one is unlike the others

# Colliders




Notice anything different with this DAG? Look closely.

- $D$ is a **collider** along the path $B \rightarrow D \leftarrow I$ (i.e., "colliding" at $D$)
- $D$ is a **noncollider** along the path $B \rightarrow D \rightarrow Y$

# Summarizing Value of DAGs imo

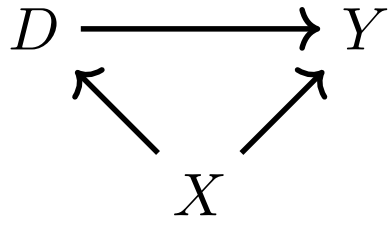
1. Facilitates the task of designing identification strategy for estimating average causal effects
2. Facilitates the task of testing compatibility of the model with your data
3. Visualizes the identifying assumptions which opens up the model to critical scrutiny

# Creating DAGs

- The DAG is a *relevant* causal relationships describing the relationship between $D$ and $Y$
- It will include:
  - → All direct causal effects among the *relevant* variables in the graph
  - → All common causes of any pair of *relevant* variables in the graph
- No need to model a dinosaur stepping on a bug causing in a million years some evolved created that impacted your decision to go to college
- We get ideas for DAGs from theory, models, observation, experience, prior studies, intuition
- Sometimes called the data generating process.

# Confounding

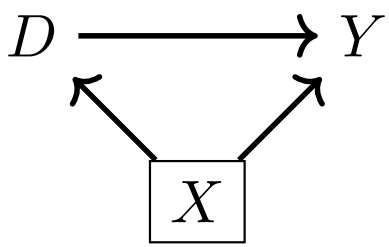
- Omitted variable bias has a name in DAGs: “confounding”
- Confounding occurs when when the treatment and the outcomes have a common cause or parent which creates spurious correlation between $D$ and $Y$




The *correlation* between $D$ and $Y$ no longer reflects the causal effect of $D$ on $Y$

# Backdoor Paths

- Confounding creates **backdoor paths** between treatment and outcome ($D \leftarrow X \rightarrow Y$) – i.e., spurious correlations
- Not the same as mediation ($D \rightarrow X \rightarrow Y$)
- We can "block" backdoor paths by conditioning on the common cause $X$
- Once we condition on $X$, the correlation between $D$ and $Y$ estimates the causal effect of $D$ on $Y$
- Conditioning means calculating $E[Y|D=1,X] - E[Y|D=0,X]$ for each value of $X$ then combining (e.g., integrating)



# Blocked backdoor paths

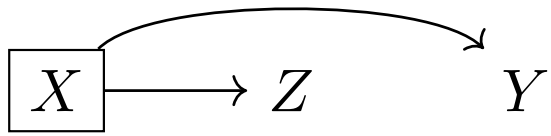
A backdoor path is blocked if and only if:

- It contains a noncollider that has been conditioned on
- Or it contains a collider that has not been conditioned on

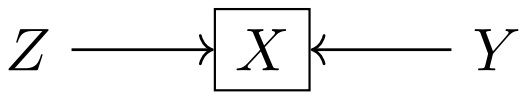
# Examples of blocked paths

Examples:

1. Conditioning on a noncollider blocks a path:




2. Conditioning on a collider opens a path (i.e., creates spurious correlations):




3. *Not* conditioning on a collider blocks a path:

# Backdoor criterion

## Backdoor criterion

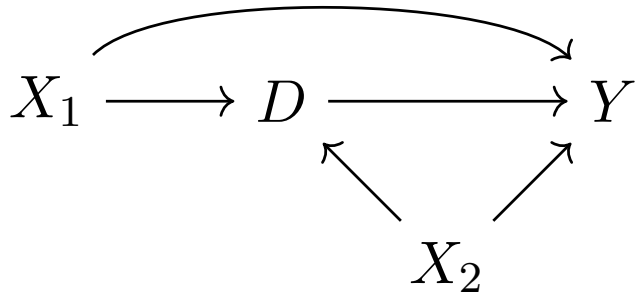
Conditioning on $X$ satisfies the backdoor criterion with respect to $(D, Y)$ directed path if:

1. All backdoor paths are blocked by $X$
2. No element of $X$ is a collider

In words: If $X$ satisfies the backdoor criterion with respect to $(D, Y)$, then controlling for or matching on $X$ identifies the causal effect of $D$ on $Y$

# What control strategy meets the backdoor criterion?

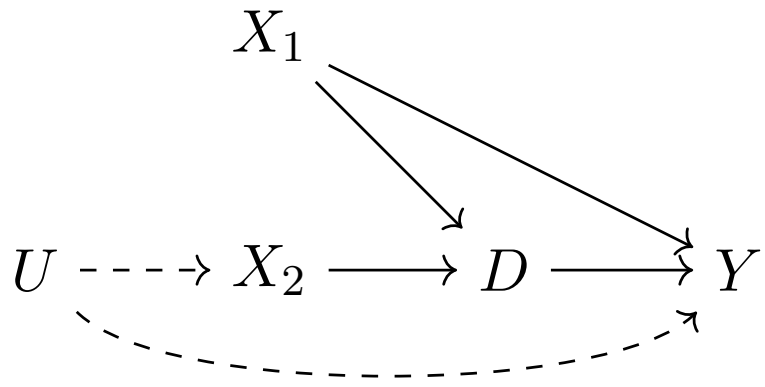
- List all backdoor paths from $D$ to $Y$. I'll wait.




- What are the necessary and sufficient set of controls which will satisfy the backdoor criterion?

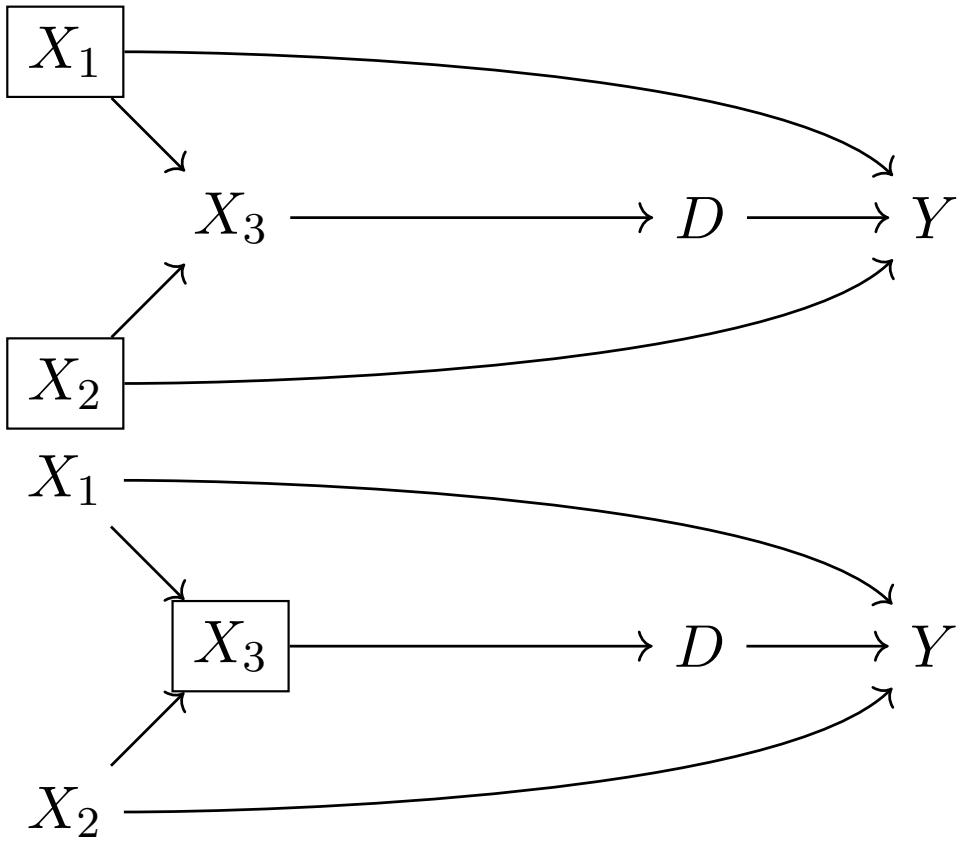
# What if you have an unobservable?

- List all the backdoor paths from $D$ to $Y$.




- What are the necessary and sufficient set of controls which will satisfy the backdoor criterion?
- What about the unobserved variable, $U$?

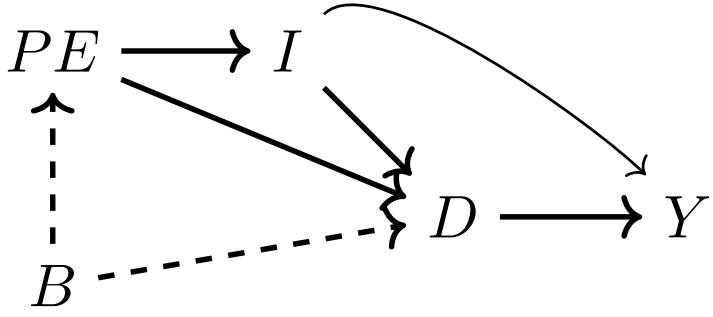
# Multiple strategies

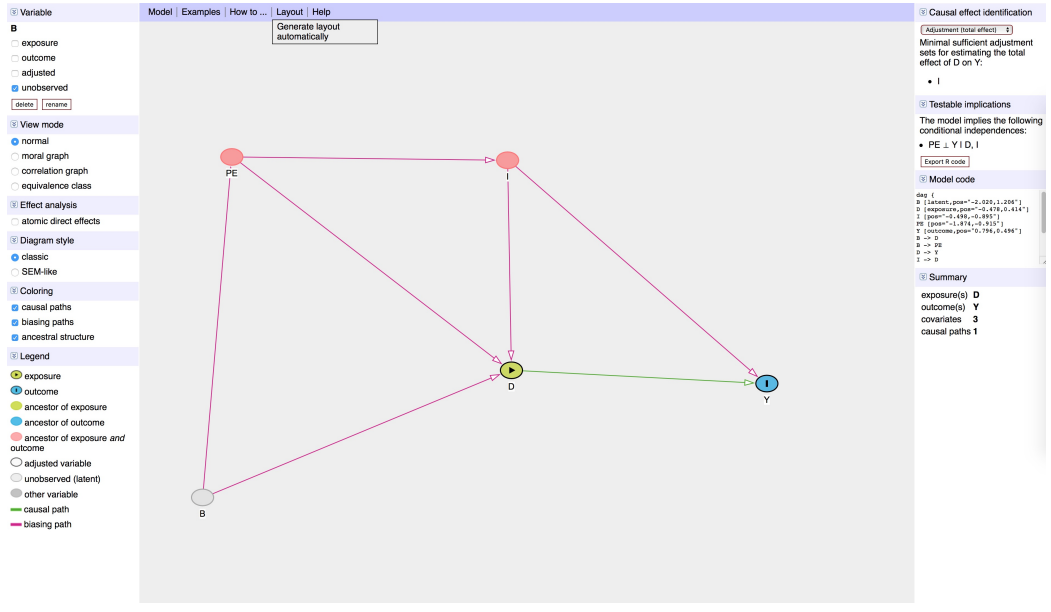



- Conditioning on the common causes, $X_1$ and $X_2$, is sufficient
- . . . but so is conditioning on $X_3$

# Testing the Validity of the DAG

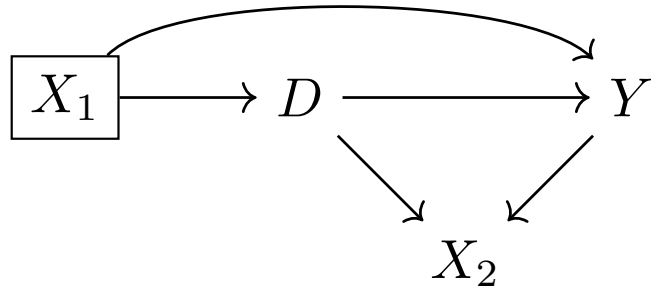
- The DAG makes testable predictions
- Conditional on $D$ and $I$, parental education ($PE$) should no longer be correlated with $Y$
- Can be hard to figure this out by hand, but software can help (e.g., Daggity.net is browser based, Causal Fusion is more advanced)
- Causal algorithms tend to be DAG based and are becoming popular in industry



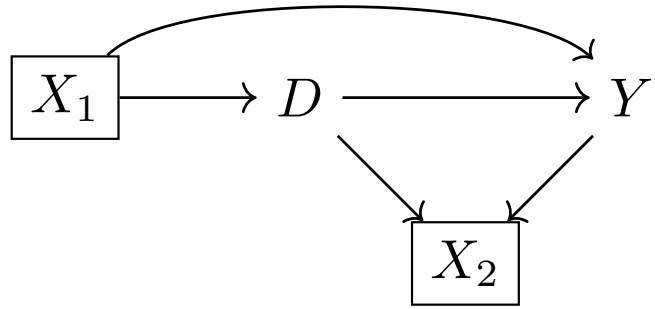

Variable
B
exposure
outcome
adjusted
unobserved
delete
rename
View mode
normal
moral graph
correlation graph
equivalence class
Effect analysis
atomic direct effects
Diagram style
classic
SEM-like
Coloring
causal paths
biasing paths
ancestral structure
Legend
exposure
outcome
ancestor of exposure
ancestor of outcome
ancestor of exposure and outcome
adjusted variable
unobserved (latent)
other variable
causal path
biasing path
Model | Examples | How to ... | Layout | Help
Generate layout automatically
PE
I
D
Y
B
Causal effect identification
Adjustment (total effect)
Minimal sufficient adjustment sets for estimating the total effect of D on Y:
I
Testable implications
The model implies the following conditional independences:
PE ⊥ Y | D, I
Export R code
Model code
dag {
B [latent,pos="-2.026,1.206"]
D [exposure,pos="-0.478,0.414"]
I [pos="-0.498,-0.895"]
PE [pos="-1.874,-0.915"]
Y [outcome,pos="0.796,0.496"]
B -> D
B -> PE
D -> Y
I -> D
Summary
exposure(s) D
outcome(s) Y
covariates 3
causal paths 1

# Collider bias

- **Conditioning on a collider introduces spurious correlations; can even mask causal directions**
  - → There is only one backdoor path from $D$ to $Y$

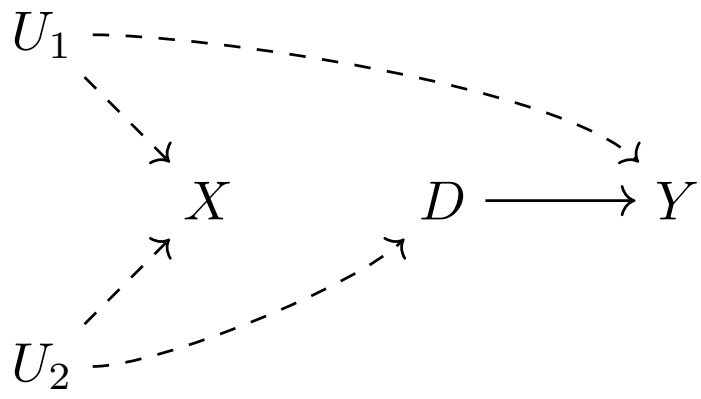



  - → Conditioning on $X_1$ blocks the backdoor path
  - → But what if we also condition on $X_2$?

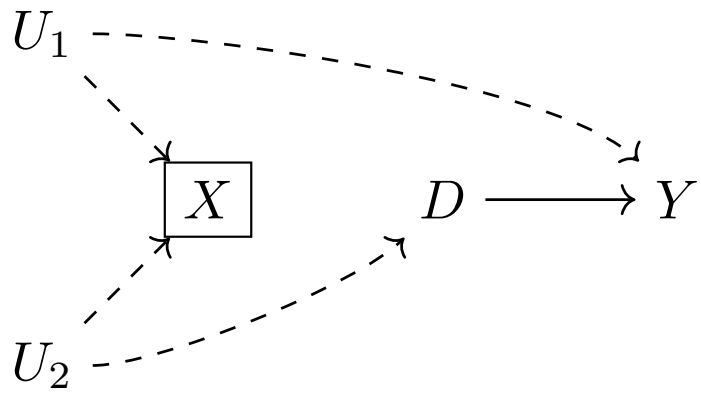



  - → Conditioning on $X_2$ opens up a new path, creating new spurious correlations between $D$ and $Y$

- **Even controlling for pretreatment covariates can create bias**
  - → Name the backdoor paths. Is it open or closed?




  - → But what if we condition on $X$?



# Living in reality - he doesn't love you

- **Fact #1**: We can't know if we have a collider bias (confounder) problem without making assumptions about the causal model (i.e. not in the codebook)
- **Fact # 2**: You can't just haphazardly throw in a bunch of controls on the RHS (i.e., "the kitchen sink") bc you may inadvertently be conditioning on a collider which can lead to massive biases
- **Fact # 3**: You have no choice but to leverage economic theory, intuition, intimate familiarity with institutional details and background knowledge for research designs.
- **Fact #4**: You can only estimate causal effects with **data** and **assumptions**.

# Examples of collider bias

# Bad controls

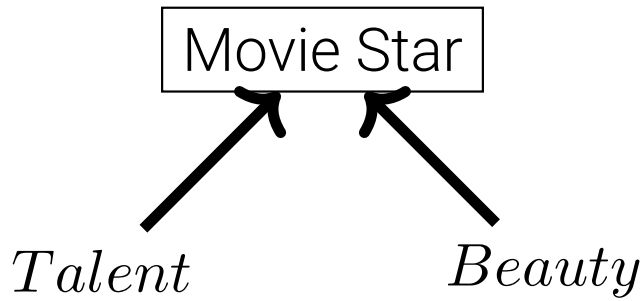
- Angrist and Pischke in MHE talk about a specific type of danger associated with controlling for an outcome – “bad controls”
- The problem is not controlling for an outcome;
- The problem is controlling for a collider and don’t correct for *that*
- This has implications for when you work with non-random administrative data, too

# Sample selection example of collider bias

Important: Since unconditioned colliders block back-door paths, what exactly does conditioning on a collider do? Let's illustrate with a fun example and some made-up data

- CNN.com headline: Megan Fox voted worst – but sexiest – actress of 2009 (link)
- Are these two things actually negatively correlated in the world?
- Assume talent and beauty are independent, but each causes someone to become a movie star. What's the correlation between talent and beauty for a sample of movie stars compared to the population as a whole (stars and non-stars)?

- What if the sample consists *only* of movie stars?



# Stata code

```
clear all
set seed 3444

* 2500 independent draws from standard normal distribution
set obs 2500
generate beauty=rnormal()
generate talent=rnormal()

* Creating the collider variable (star)
gen score=(beauty+talent)
egen c85=pctile(score), p(85)
gen star=(score>=c85)
label variable star "Movie star"

* Conditioning on the top 15%
twoway (scatter beauty talent, mcolor(black) msize(small) msymbol(smx)),
ytitle(Beauty) xtitle(Talent) subtitle(Aspiring actors and actresses) by(star,
total)
```


*Figure:* Top left figure: Non-star sample scatter plot of beauty (vertical axis) and talent (horizontal axis). Top right right figure: Star sample scatter plot of beauty and talent. Bottom left figure: Entire (stars and non-stars combined) sample scatter plot of beauty and talent.

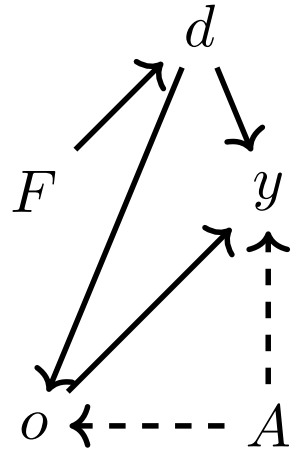
# Stata

- Run Stata file star.do

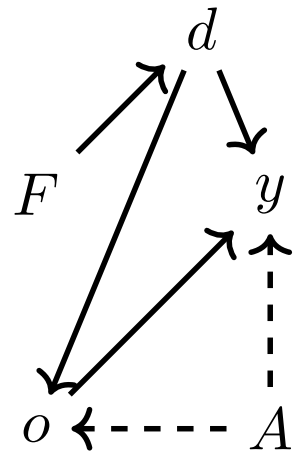
# Occupational sorting and discrimination example of collider bias

- Let's look at another example: very common for think tanks and journalists to say that the gender gap in earnings disappears once you control for occupation.
- But what if occupation is a collider, which it could be in a model with occupational sorting
- Then controlling for occupation in a wage regression searching for discrimination can lead to all kinds of crazy results *even in a simulation where we explicitly design there to be discrimination*

# DAG




$F$ is female, $d$ is discrimination, $o$ is occupation, $y$ is earnings and $A$ is ability. Dashed lines mean the variable cannot be observed. Note, by design, being a female has no effect on earnings or occupation, and has no relationship with ability. So earnings is coming through discrimination, occupation, and ability.




Mediation and Backdoor paths

1. $d \rightarrow o \rightarrow y$
2. $d \rightarrow o \leftarrow A \rightarrow y$

# Stata model (Erin Hengel)

- Erin Hengel (`www.erinhengel.com`) and I worked out this code and she gave me permission to put in my Mixtape
- Let's look at collider_discrimination.do or collider_discrimination.R together

*Table:* Regressions illustrating collider bias with simulated gender disparity

| Covariates: | **Unbiased combined effect** | **Biased** | **Unbiased wage effect only** |
|---|---|---|---|
| Female | -3.074*** | 0.601*** | -0.994*** |
| | (0.000) | (0.000) | (0.000) |
| Occupation | | 1.793*** | 0.991*** |
| | | (0.000) | (0.000) |
| Ability | | | 2.017*** |
| | | | (0.000) |
| N | 10,000 | 10,000 | 10,000 |
| Mean of dependent variable | 0.45 | 0.45 | 0.45 |

- Recall we designed there to be a discrimination coefficient of -1
- If we do not control for occupation, then we get the combined effect of $d \to o \to y$ and $d \to y$
- Because it seems intuitive to control for occupation, notice column 2 - the sign flips!
- We are only able to isolate the direct causal effect by conditioning on ability and occupation, but ability is unobserved

# Administrative data

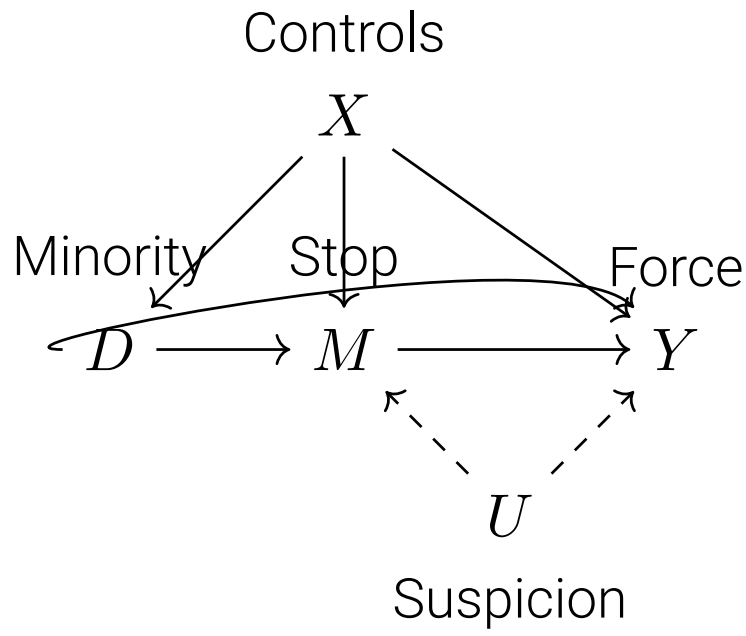
- Admin data has become extremely common, if not absolutely necessary
- But naive use of admin data can be dangerous if the drawing of the sample is itself a collider problem (Heckman 1979; Elwert and Winship 2014)
- Let's look at a new paper by Fryer (2019) and a critique by Knox, et al. (2019)

# Collider bias and police use of force

- Claims of excessive and discriminator use of police force against minorities (e.g., Black Lives Matter, Trayvon Martin, Michael Brown, Eric Garner)
- Challenging to identify
  - → Police-citizen interactions are conditional on interactions having already been triggered
  - → That initial interaction is unobserved
- Fryer (2019) is a monumental study for its data collection and analysis: Stop and Frisk, Police-Public Contact Survey, and admin data from two jurisdictions
- Codes up almost 300 variables from arrest narratives which range from 2-100 pages in length – shoeleather!

# Initial interaction

- Fryer finds that blacks and Hispanics were more than 50% more likely to have an interaction with the policy in NYC Stop and Frisk as well as Police-Public Contact survey
- It survives extensive controls – magnitudes fall, but still very large (21%)
- Moves to admin data
- Conditional on police interaction, *no* racial differences in officer-related shootings
- Fryer calls it one of the most surprising findings in his career
- Lots of eyes on this study as a result of the counter intuitive results; published in JPE
- Knox, et al (202) claim his data is itself a collider. What?



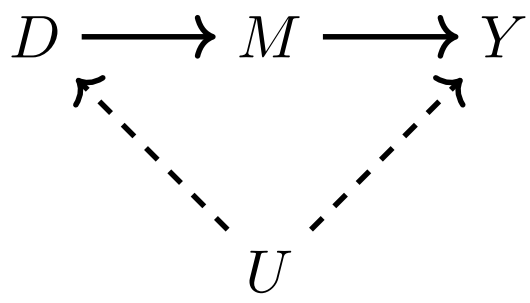

Fryer told us $D \to M$ exists from both Stop and Frisk and Police-Public. But note: admin data is instances of $M$ stops, which is itself a collider. If this DAG is true, then spurious correlations enter between $M$ and $Y$ which may dilute our ability to estimate causal effects.

# Knox, et al (2020)

- Move from DAG to more contemporary potential outcomes notation to design relevant parameters
- Use potential outcomes and bounds
- Even with lower bound estimates of the incidence of police violence against civilians is more than 5x higher than what Fryer (2019) finds
- Heckman (1979) – we *cannot* afford to ignore sample selection

# Mechanisms

- Rarely does an intervention operate directly on an outcome
  - → Parental substance abuse causes foster care removals not because foster care witness substance abuse, but because parents abuse and neglect their children when they abuse drugs
- The presence of mechanisms, it turns out, is valuable because of their policy relevance, but also because we can use them *sometimes* for identification




- $D$ is confounded by $U$; therefore we cannot identify the causal effect of $D$ on $Y$ using the backdoor criterion bc $D \leftarrow U \rightarrow Y$ cannot be blocked
- Pearl (2009) showed that this DAG actually does allow us to recover the effect of $D$ on $Y$, though – just not via the backdoor criterion
- We'll now look at a lesser known method of identification called the frontdoor criterion

# Front door criterion

*If one or more unblocked back door paths connect a causal variable to an outcome variable, the causal effect is identified by conditioning on a set of observed variables $M$ that make up the identifying mechanism if and only if: 1) the variables in $M$ intercept all directed paths from the causal variable to the outcome ("exhaustiveness"); 2) No unblocked back-door paths connecting the causal variable to the variables in the set $M$ and all back door paths from the variables in $M$ to the outcome can be blocked by conditioning on $D$ ("isolation")*

# Exhaustiveness

- Exhaustiveness means the variables $M$ are the only paths through which $D$ impacts $Y$.
- In other words, rules out direct effects that bypass $M$ altogether
- "only through $M$" in place of exhaustiveness and you get the idea

# Isolation

- Mechanism itself is not confounded with respect to $Y$
- There does not exist some additional unobservable creating a back door path between $M$ and $Y$
- It's a truly closed system, and as such, you're going to be making a strong argument so good luck

# Three step method

1. Estimate the effect of $D$ on $M$. Consider a regression of $M$ on $D$ or simple difference in mean $D$ with respect to $M$

$$D = \alpha_0 + \beta M + \epsilon$$

   → $M$ is isolated, so it is not confounded
   → $D \leftarrow U \rightarrow Y \leftarrow M$ which is blocked bc $Y$ is a collider
   → Therefore $\widehat{\beta}$ identifies $\beta$
2. Estimate the effect of $M$ on $Y$ conditional on $X$
   → Gets you an unbiased estimate of $M$ effect on $Y$ bc only backdoor path from $M$ to $Y$ is $M \leftarrow D \leftarrow U \rightarrow Y$
   → So long as we condition on $D$ this path is blocked

$$Y = \alpha_1 + \gamma M + \psi D + \varepsilon$$

3. Multiply $\widehat{\gamma} \times \widehat{\beta}$ and you get the causal effect of $D$ on $Y$

# Examples have been elusive

- Pearl has suggested smoking as an example of this
- Smoking causes tar build-up, tar build-up causes lung cancer, smoking is endogenous to confounders
- Requires smoking to not have a direct effect on lung cancer, which is incorrect
- But a new paper by Bellemare, et al. (2021) provides a plausible example involving tipping and Uber

# DGP

We will define a few error terms. Let $U$ be our unobserved confounder variable drawn from the standard normal distribution. And let $Z$ be drawn from the uniform distribution from 0 to 1. And let the following three error terms, $\varepsilon_D$, $\varepsilon_M$ and $\varepsilon_Y$ be normally distributed as well. Then let our variables of interest come from the following linear system:

$$
\begin{aligned}
D_i &= 0.5U_i + \varepsilon_D \\
M_i &= Z_i D_i + \varepsilon_M \\
Y_i &= 0.5M_i + 0.5U_i + \varepsilon_Y
\end{aligned}
$$

Code is available (I may run it for you now), but let's look at the results

*Table:* Simulation results Bellemare, et al. (2021)

| Variables: | **Benchmark** | **Naive** | **Front Door** | | **Direct effect** |
|---|---|---|---|---|---|
| | **Y** | **Y** | **M** | **Y** | **Y** |
| Treatment $D$ | 0.257*** | 0.451*** | 0.505*** | 0.198*** | 0.005 |
| | (0.004) | (0.003) | (0.001) | (0.004) | (0.003) |
| Mechanism $M$ | | | | 0.500*** | 0.500*** |
| | | | | (0.003) | (0.003) |
| Confounder $U$ | 0.492*** | | | | 0.491*** |
| | (0.004) | | | | (0.004) |
| Estimated causal effect ($\widehat{\delta}$) | 0.257*** | 0.451*** | | 0.252*** | – |
| | (0.004) | (0.003) | | (0.002) | |
| N | 100,000 | 100,000 | 100,000 | 100,000 | 100,000 |

# Real world data

- Harrington (2019) nots shared rides typically result in lower tips for Uber drives: “on average, about 17% of rideshares end up with the driver getting tipped. For strips where a shared trip was authorized, that number is halved to a measly 8.6%.”
- Drivers experiencing such declines probably think it’s caused by sharing rides (e.g., bystander effects, freeriding, etc.)
- But maybe it’s selection – cheapskates share rides
- Let’s use the front door criterion to check

# Assumed Uber Tipping DAG

- Let $D$ here be authorizing a shared ride (regardless of whether a shared ride occurred), $M$ be a dummy measuring one if sharing did occur, $Y$ be the amount the passengers tipped and $U$ be the unobserved covariates.
- Use the front door criterion, conditional on a series of geographic and time fixed effects using data on over 95 million Uber and Lyft rides in Chicago in 2019.
- Estimate the effect of authorization on both whether a passenger tips as well as how much, what they call the extensive and intensive margin of tipping, respectively.
- These data come from a data portal maintained by Chicago's Department of Business aAffairs and Consumer Protection's Transportation Network Providers and is freely available for download from the City of Chicago's website.

# Assumptions

- It’s the same DAG as before so I won’t redraw it
- Key to this DAG is to consider that once the authorization to share a ride is initiated (the treatment), then when the ride is shared (the mechanism), the authors argue that their extensive set of fixed effects will yield plausible conditions for isolation and exhaustiveness are guaranteed.
- This means there is no direct effect of authorization on tipping, nor does there exist an unblocked backdoor path from sharing a ride and tipping itself.

# Estimation

- Using the logic of the front door criterion, the authors estimate the same two step procedure as shown in the previous simulation with the caveat that they include extensive fixed effects so as to create conditional conditions for isolation and exhaustiveness.
- For illustrative purposes, I will only focus on the effect at the extensive margin (i.e., on whether a passenger tipped at all).

*Table:* Estimation results for tipping at the extensive margin from **?**

| Variables: | **Naive** | **Front Door** | |
|---|---|---|---|
| | **Tipped** | **Shared Trip** | **Tipped** |
| Sharing authorized $D$ | -0.0628*** | 0.6769*** | -0.0550*** |
| | (0.0001) | (0.0002) | (0.0002) |
| Shared trip $M$ | | | -0.0115*** |
| | | | (0.0002) |
| Full fare | 0.0050*** | -0.0064*** | 0.0049*** |
| | (0.00001) | (0.00001) | (0.00003) |
| Estimated causal effect ($\widehat{\delta}$) | -0.0628*** | | -0.0078** |
| | (0.0001) | | (0.0001) |
| N | 95,670,449 | 95,670,449 | 95,670,449 |

# Interpretation

- Column 1: naive regression simply compares tipping between authorized and non-authorized sharing (6.3pp reduction in tipping)
- Front door criterion: 1pp reduction
- Not surprising drivers don't want ride shares, but authors argue it's caused by selection (i.e., the people using ride shares) not ride share itself
- Unclear if you banned it whether it would increase driver earnings in other words
- Cool paper but it took me forever to download 95 million observations

# Summarizing all of this

- Your dataset will not come with a codebook flagging some variables as “confounders” and other variables as “colliders” because those terms are always context specific
- Except for some unique situations that aren’t generally applicable, you also don’t always know statistically you have an omitted variable bias problem; but both of these are fatal for any application
- You only know to do what you’re doing based on *knowledge about data generating process*.
- All identification must be guided by theory, experience, observation, common sense and knowledge of institutions
- DAGs absorb that information and can be then used to write out the explicit identifying model

# DAGs are not panacea

- DAGs cannot handle, though, reverse causality or simultaneity
- So there are limitations. “All models are wrong but some are useful”
- They are also not popular (see Twitter ongoing debates which have descended into light hearted jokes as well as aggressive debates)
- But I think they are helpful and while not *necessary*, showcase what is necessary – assumptions
- Heckman (1979) can maybe provide some justification at times

**Temporary page!**

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was some unprocessed data that should have been added to
page this extra page has been added to receive it.
If you rerun the document (without altering it) this surplus pag
away, because LaTeX now knows how many pages to expect fo
document.