Causal Inference I

Mixtape Session

Roadmap

- Introduction to course
- Potential outcomes
  - Naive causal inference
  - Potential outcomes notation
  - Selection bias
  - Independence
  - Example of physical experimentation: eBay advertising
  - Example of physical experimentation: HIV status
- Randomization inference
  - Lady tasting tea
  - Fisher's sharp null
  - Alternative test statistics

Welcome!

- Scott Cunningham, professor of economics at Baylor University, author of Causal Inference: the Mixtape, applied microeconomist
- Causal inference is a sub-category within econometrics most often associated with “treatment effects”, as opposed to economic theory
- Workshops can be helpful ways to plug into one’s methodological training as the field has become very impactful (arguably won the Nobel Prize last October)

What is Mixtape Sessions?

- Mixtape Sessions is my online platform created to “democratize causal inference” at all levels by helping connect people with material and teachers from beginner to advanced
- I started it because a strong conviction I have which is there is a particular approach to empirical work (which I tried to describe in my 3-part history) which did not universally spread into everyone’s methods training
- That’s because imo the stronger part of the growth in “causal inference” came from ordinary empiricists, and as such, it got passed around through professors in how they taught field courses, and not as broadly in how people taught econometrics

Class goals

1. **Confidence**: You will feel like you have a good understanding of causal inference so that by the end it doesn't feel all that mysterious or intimidating
2. **Comprehension**: You will have learned a lot both conceptually and in the specifics, particularly with regards to issues around identification and estimation
3. **Competency**: You will have more knowledge of programming syntax in Stata and R (and python!) so that later you can apply this in your own work

4-day Causal Inference Workshop

- We workshop together for 4-days, 8am to 5pm CST, with 15 min breaks on the hour and a 1-hour lunch break at 12:00PM CST
- I mix exposition, discussion of papers, coding exercises and discussion as best as I can
- I’m me, and I teach how I teach, with passion, enthusiasm, deep joy, but I’m not an econometrician so sometimes I take the long way

Causal Inference table of contents

Causal Inference I				
	Day 1	**Day 2**	**Day 3**	**Day 4**
Pre-Class	*Read Thornton AER*	*Read Dehejia and Wahba RESTAT*	*Read Card Econometrica*	*Read Hansen AER*
9am	Potential outcomes and counterfactuals	Causal graphs	Instrumental variables, intuition, and 2SLS	Introduction to regression discontinuity design
10am				
11am		**Lalonde Coding Lab Part 1**	Weak instruments and 2SLS bias	Nonlinearities and estimation
12pm	Lunch			
1pm	Randomization, selection bias	Matching	Heterogenous treatment effects & LATE	RD Shiny App
2pm	**Thornton Coding Lab Part 1**			Nonparametric estimation
3pm				
4pm	Randomization inference		**IV Coding Lab**	General tips (data visualization, density tests, etc)
5pm	RI Shiny App			**Hansen Coding Lab**
6pm	**Thornton Coding Lab Part 2**	**Lalonde Coding Lab Part 2**	Judge IV Design	

Workshop (Part 1) Topics

1. Foundations: Day 1
2. Graphs and Selection on Observables: Day 2
3. Instrumental Variables: Day 3
4. Regression Discontinuity Design: Day 4

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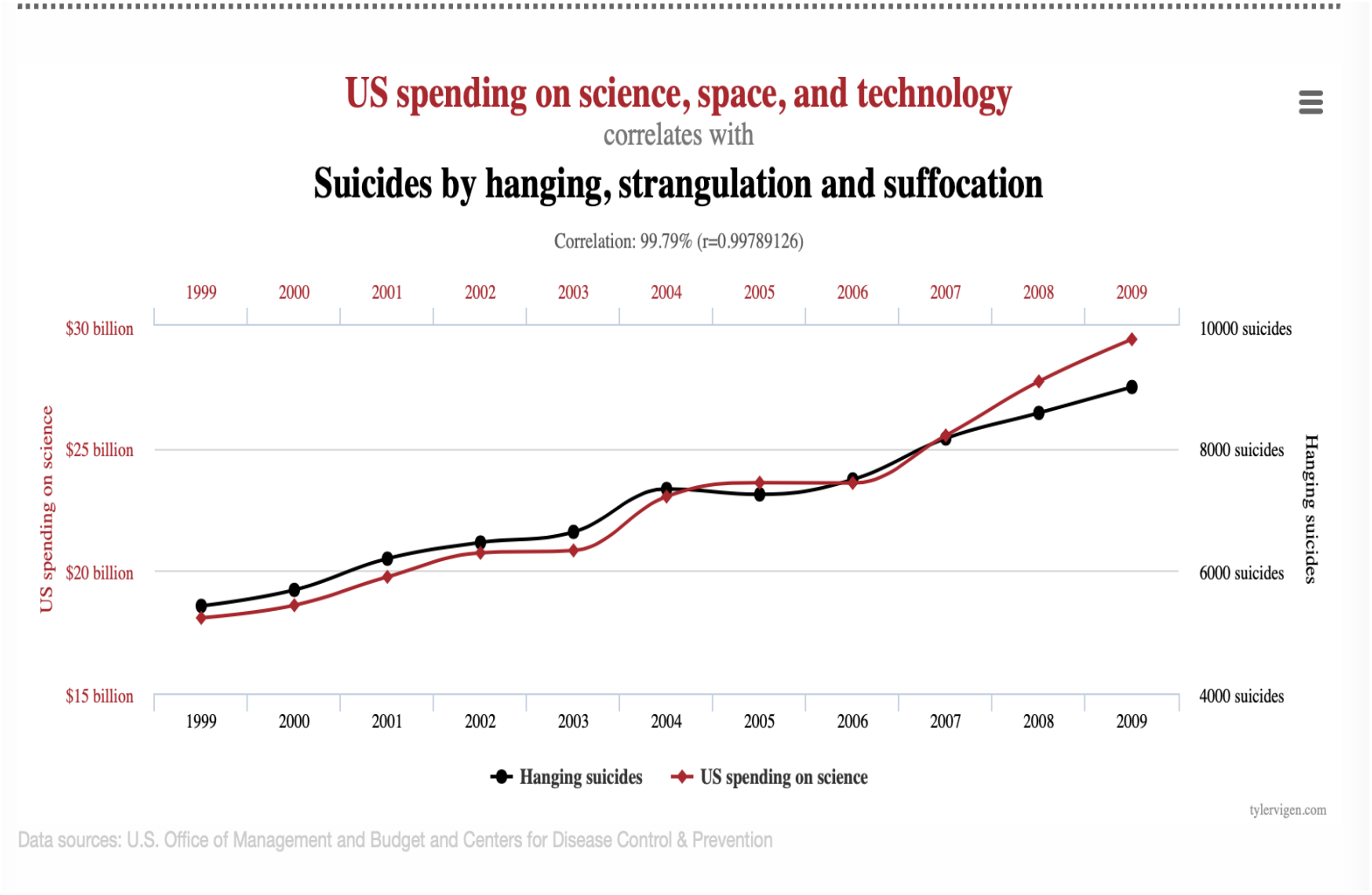
Coming to causal inference

- Many roads lead to this material – from computer science, to statistics, to economics, to philosophy, to epidemiology and so on and so forth
- Therefore any effort to teach it will always have a degree of subjectivity reflecting the presenter
- But even on something as narrow as what I'm going to discuss, there are even subtleties I want to take note of, and this reflects my own subjectivity

Sociological history of causal inference

- You can get a sense of my own perspective in my 3-part substack series on the sociological history of late 20th century quantitative causal inference (https://causalinf.substack.com/p/a-selected-history-of-quantitative)
- I'm not going to review it here, as it takes a long time, but I encourage you to read it as it can help you understand where this came from and why it looks the way it does, but also my own beliefs
- All I will say is in my opinion, causal inference in the applied social sciences has two parents – the statistician/econometricians and the "ordinary data workers" – and that gives it the shape it has taken on

Spurious correlations



Spurious correlations

- What is causality? **We need a definition**.
- Philosophers created a definition, but statisticians found a trick to make it tractable
- That trick combined notation (a definition) and a mechanism (treatment assignment) into “acceptable solutions”
- Let’s now dig into the issues around definitions and then the first major breakthrough – randomization

Causality, causal inference

- Philosophers have been interrogating what causality is going back to antiquity
- It is one of the most important topics in both the fields of epistemology (how do I know if something causes something else?) and metaphysics (we will see why it involved metaphysics)
- But I want you to distinguish for this workshop between the topic of "causality" and the topic of "causal inference", because the former is a much broader topic that includes many differing views
- The latter is really a description of contemporary scientific methods bridging many sciences, including medicine, computer science, and much of the social sciences

Philosophical origins of causal inference concepts

“If a person eats of a particular dish, and dies in consequence, that is, would not have died if he had not eaten it, people would be apt to say that eating of that dish was the source of his death.” – John Stuart Mill (19th century moral philosopher and economist)

“Causation is something that makes a difference, and the difference it makes must be a difference from what would have happened without it.” – David Lewis (20th century metaphysical philosopher)

Ancient One explains it

Both people pinned the idea of causality in *comparisons* between (1) different actions taken and (2) different timelines

Let's start off and listen to the Ancient One explain to Bruce Banner (aka the Incredible Hulk) from Avengers: Endgame

https://youtu.be/1S3I0PqkooA?t=50

Counterfactuals

Philosophers and stories alike eventually settled on the idea that causality was rooted in a particular kind of comparison, like “treatment” versus “control” groups

But the comparison as you heard the Ancient One say was much weirder than comparing two groups – it was comparing two possible events (choices) across two possible timelines that may (or may not) trigger two different outcomes

The key idea in contemporary causal inference is the idea of the counterfactual. Counterfactuals are neither past nor future. They are alternative histories created by thought experiments but we use them as framing devices to decipher causality in our timeline

Statistical origins

"Yet, although the seeds of the idea that [causal effects are comparisons of potential outcomes] can be traced back at least to the 18th century [most likely he means David Hume], the formal notation for potential outcomes was not introduced until 1923 by Neyman." – Don Rubin (1990)

Jerzy Neyman introduces his definition

- Early 20th century statistician, one of the “fathers” of modern statistics
- 1923 article describes a field experiment with differing plots of land (imagine hundreds of square gardens) and many different “varieties” of fertilizer that farmers could apply to the land
- “U_{ik} is the yield of the ith variety on the kth plot...” (Neyman 1923)
- He calls U_{ik} “potential yield”, as opposed to the realized yield because i (the fertilizer type) described all possible fertilizers that could be assigned to each k square garden
- Though only one fertilizer will be assigned to the land, many possibilities exist in other words

Urn model

- For each fertilizer there is an associated “potential yield” that he collapses into U and each of them he considers to be “a priori fixed but unknown” (Rubin 1990)
- Farmers draw fertilizer from an urn, like a bingo ball from a bingo ball machine, and apply it to each square garden
- Once the fertilizer is assigned, we go from “all possible outcomes” to “realized outcome” terminology
- Interestingly – the urn model was a thought experiment, but it was stochastically identical to the completely randomized experiment, and he doesn’t notice it
- His arch-rival, Ronald Fisher, does notice it and publishes a book two years later recommending randomized experiments rooted in this paper

Treatment assignment mechanism

"Before the 20th century, there appears to have been only limited awareness of the concept of the assignment mechanism. Although by the 1930s, randomized experiments were firmly established in some areas of scientific investigation, notably in agricultural experiments, there was no formal statement for a general assignment mechanism and, moreover, not even formal arguments in favor of randomization until Fisher (1925)." (Imbens and Rubin 2015)

Progress is made and progress is not made

- Whereas the RCT takes off in medicine and agriculture, it is not adopted as universally in social sciences, like economics
- Economics ironically does have in its history early econometricians who thought about causality like Neyman and Fisher, but listen to Guido Imbens describe the transition towards modeling causality in terms of “realized outcomes

https://www.youtube.com/watch?v=drGkRy53bB4

Prediction vs causal inference

- Statistics was not merely interested in causal inference
- It was also interested in prediction, as are many of the social sciences
- But causal inference appeared to progress from Neyman-Fisher until Rubin's 1970s work without explicit references to Neyman's original ideas
- I think sometimes therefore the "clarity" as Imbens said was lost and as such, prediction and causal inference did not have sharp lines separating them

Different types of prediction

Traditional prediction

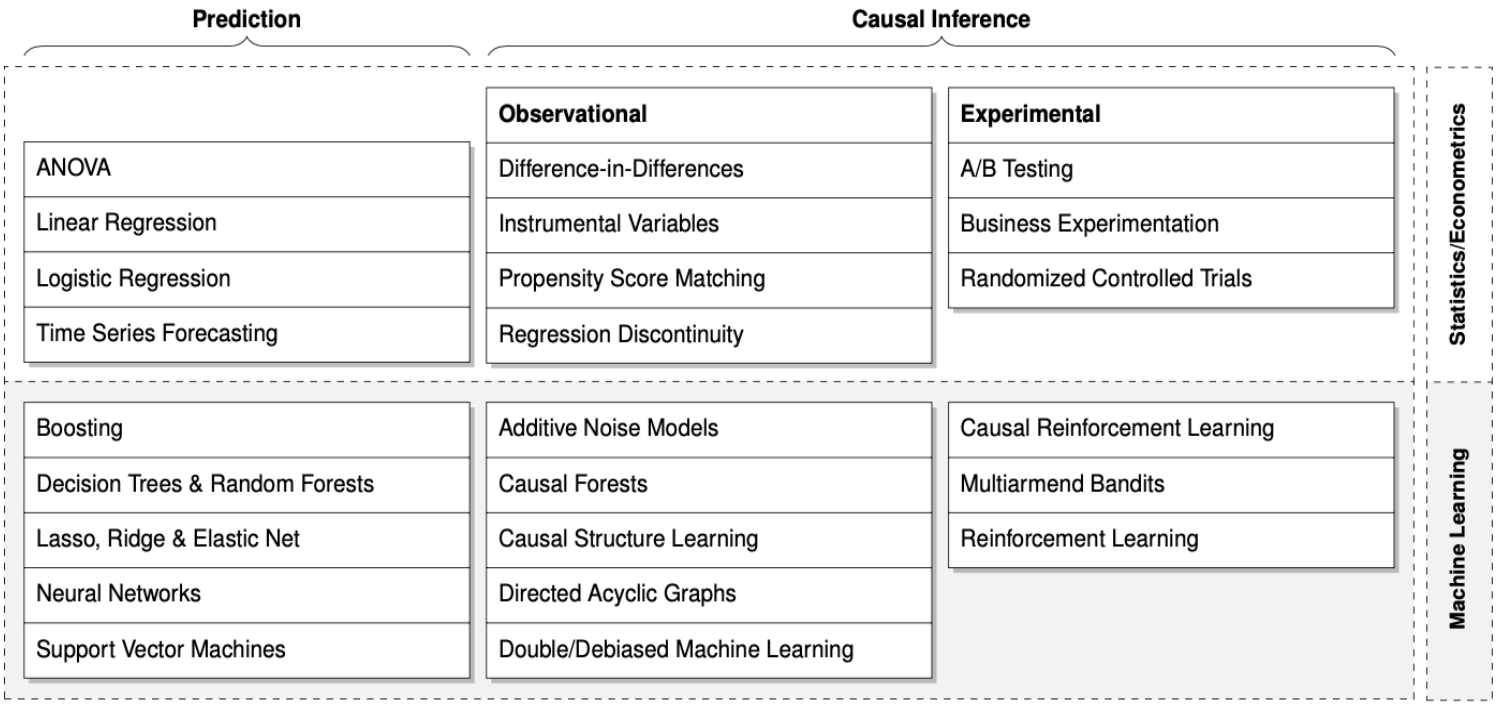
- Traditional prediction seeks to detect patterns in data and fit functional relationships between variables with a high degree of accuracy
- “Does this person have heart disease?”, “How many books will I sell?”
- It is not predictions of what effect a choice will have, though

Causal inference

- Causal inference is also a type of prediction, but it’s a prediction of a *counterfactual* associated with a particular *choice taken*
- Causal inference takes that predicted (or imputed) counterfactual and constructs a causal effect that we hope tells us about a future in the event of a similar choice taken

Identification problem

Figure 1: Examples of popular data analysis algorithms in statistics and econometrics, as well as machine learning and artificial intelligence, classified according to prediction and causal inference methods. Causal inference methods are further differentiated according to observational (based on ex-post observed data) and experimental approaches.



Naive causal inference

- Aliens come and orbit earth, see people dying in hospitals and conclude “doctors are hurting people”
- They kill the doctors, unplug patients from machines, throw open the doors – many patients inexplicably die
- *We are the aliens in our research*

#1: Correlation and causality are different concepts

Causal is one unit, correlation is many units

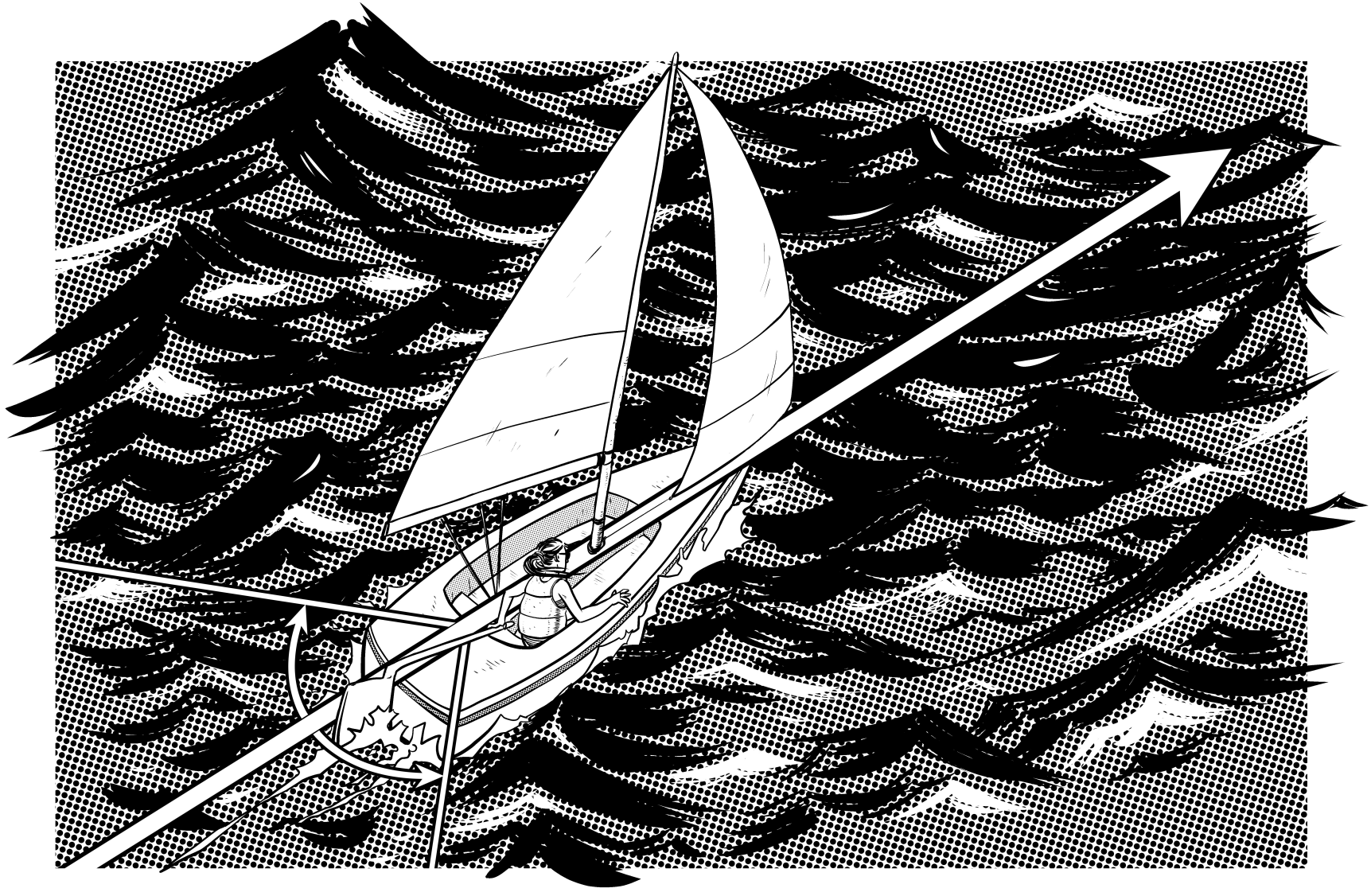
- Causal question: “If a doctor puts a patient on a ventilator (D), will her covid symptoms (Y) improve?”
- Correlation question:

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

#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: Causality may mask correlations!



Potential outcomes notation

- Let the treatment be a binary variable:

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

where i indexes an individual observation, such as a person

Potential outcomes notation

- Potential outcomes:

$$Y_{i,t}^{j} = \begin{cases} 1 \text{ health if placed on ventilator at time } t \\ 0 \text{ health if not placed on ventilator at time } t \end{cases}$$

where j indexes a potential treatment status for the same i person at the same t point in time

Realized vs potential outcomes

- Potential outcomes Y^1 and realized outcomes Y are not the same ideas or notation
- Potential outcomes refer to the “a priori fixed but unknown” outcomes associated with different possible treatment assignments
- Realized outcomes refer to the “posterior and known” outcome associated with a specific treatment assignment
- This distinction is more subtle than I can emphasize, and so we have to spend time on the front end in spreadsheets and code

Important definitions

Definition 1: Individual treatment effect

The individual treatment effect, δ_i, associated with a ventilator is equal to $Y_i^1 - Y_i^0$.

Important definitions

Definition 2: Switching equation

An individual's realized health outcome, Y_i, is determined by treatment assignment, D_i which selects one of the potential outcomes:

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

Missing data problem

Definition 3: 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, δ_i, is *unknowable*.

- Fundamental problem of causal inference is a consequence of what the switching equation does and cannot be fixed with more data – always missing one of the potential outcomes
- Causal inference is a missing data problem requiring imputation of missing counterfactuals (sometimes explicitly such as with nearest neighbor or synthetic control, sometimes implicitly as with RDD)

Average Treatment Effects

Definition 4: Average treatment effect (ATE)

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

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

Aggregate parameters based on individual treatment effects are *summaries* of individual treatment effects

Cannot be calculated because Y_i^1 and Y_i^0 do not exist *for the same unit i* due to switching equation

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:

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

Cannot be calculated because Y_i^1 and Y_i^0 do not exist *for the same unit i* due to switching equation.

Conditional Average Treatment Effects

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:

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

Cannot be calculated because Y_i^1 and Y_i^0 do not exist *for the same unit i* due to switching equation

Any collection of treatment effects

- Notice how in all three of these, all we did was take the defined treatment effect at the individual and aggregate
- The aggregate causal parameters are *definitions* of summaries but cannot be calculated directly bc of missing data problem
- But they can be estimated, which is probably a distinction in epistemology as it's knowledge but of a different type ("warranted belief")

Naive causal inference and selection bias

- Naive causal inference is often caused by confusing prediction or description with causal inference
- It is naive if it does not directly address, in a reasonable way, the problem of *selection bias*
- This is better seen with a story and a decomposition

Definition 7: Simple difference in mean outcomes (SDO)

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

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

Notice how I moved between potential outcomes (Y^1) to realized outcomes (Y for $D=1$) using the switching equation

Simple difference in mean outcomes

- Simple difference in mean outcomes may or may not be “naive”
- It can be calculated manually by differencing averages, or with a regression

$$Y_i = \alpha + \delta D_i + \varepsilon_i$$

  where $\widehat{\delta}$ is the SDO from the previous slide
- SDO creates a number (i.e., it’s a calculation), but what does that number mean in terms of causality and bias?

Decomposition of the SDO

Decomposition of the SDO

The SDO is made up of three things:

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

We need to see this as everything hinges on getting it

Begin with ATE definition

Law of iterated expectations

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

Change notation

Substitute letters for expectations to go easy on the eyes

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

Rewrite ATE definition

Rewrite ATE

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

Simple manipulation of ATE definition

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

Carry forward from previous slide

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

Replace letters with original terms

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

Decomposition of the SDO

Decomposition of the SDO

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

Note: this is a *rewritten* formula for the definition of the ATE and so is *always* true. Also, notice that we started with π but in the end we weight by $1-\pi$.

Estimate SDO with sample averages

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

Using the switching equation and sample averages, we can calculate $E_N[Y|D=1] \rightarrow E[Y^1|D=1]$, $E_N[Y|D=0] \rightarrow E[Y^0|D=0]$ and $(1-\pi)$ is the share of the population in the control group.

Selection bias

- For many of us, we have heard the word “selection bias” before but it was with respect to “non-random samples”
- In causal inference, that isn’t what we mean. We mean mean potential outcomes differ for two groups.
- We cannot observe this though because one of the comparisons is counterfactual and the other is realized

Bias #1: Selection bias

- Look very closely at the selection bias terms on their left and right hand sides

$$E[Y^0|D=1] \neq E[Y^0|D=0]$$

- Ask yourself: do you think that the people placed on vents would've had the same mean health outcomes as the people not on vents had they not been on vents? Why do you think that?

Bias #1: Selection bias

Probably not. Had they not been on vents, many would've died.

$$\underbrace{E[Y^0|D=1]}_{\text{Worse off vents}} < \underbrace{E[Y^0|D=0]}_{\text{Better off vents}}$$

Bias was caused by *the doctors* being good at their jobs!

Bias was caused by the *treatment assignment mechanism*, and Imbens and Rubin said that was not really emphasized until Fisher (1925)

Humans cause selection bias, not statistical model

- People cause the bias bc people choose treatments to make their lives better which means choosing $D = 1$ if $Y^1 - Y^0 > 0$.
    1. I chose to get a PhD because I thought I would be less happy without it – i.e., Y^0 maybe was lower for me than others
    2. I chose to get a PhD because I thought it would make me happier – i.e., Y^1 maybe was higher for me than others
- Selection bias is associated with the first; heterogenous treatment effect bias with the second

Illustrating selection bias with spreadsheets

- Chronic PTSD has historically been treated with cognitive behavior therapies like mindfulness, but recent work shows therapist assisted MDMA (street name: ecstasy), are effective too
- Ongoing work in psychopharmacology has begun experimenting with long dormant approaches in the psychedelics and empathogens for treating mental illness, including PTSD
- Several states have legalized it, Australia just legalized it this week, and FDA is expected to “reschedule” it soon
- MAPS organization has been funding RCTs in compliance with FDA trials to study MDMA’s effect on PTSD

  https://www.nature.com/articles/s41591-021-01336-3

Illustrating selection bias with spreadsheets

- Perfect Doctor can accurately determine whether mindfulness practices or MDMA is more beneficial for treating a patient's chronic PTSD ($Y^1 - Y^0$ is positive or negative), and makes treatment assignments ($D = 1$ or 0) depending on its impact
- We will go through an exercise together (copy this google sheet) analyzing the implications of the perfect doctor's choices on a range of statistics, followed by discussion

  https://docs.google.com/spreadsheets/d/10DuQqGtH_Ewea7zQoLTFYHbnvqaTVDhn2GDzq3Oa6EQ/edit?usp=sharing

Summarizing the goals of causal inference

Our goal in causal inference is to estimate aggregate causal parameters with data by exploiting what is known about the treatment assignment mechanism

Depending on the treatment assignment mechanism, certain procedures are allowed and others are prohibited

Let's look what happens in an RCT *and why* this addresses selection bias term $E[Y^0|D=1]$ and $E[Y^0|D=0]$ to see why Fisher (1925) recommended it

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:

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

Bad Doctor and Perfect Doctor Lab

Now let’s spent a half hour on a lab in code in R and Stata to try and illustrate this again

Interference when aggregating units

- While treatment effects are defined at individual level, aggregate parameters combine units
- This therefore means that for the aggregate parameters to be stable, there cannot be “interference” between one unit’s treatment choice and another unit’s potential outcome
- Creates challenges for definitions and estimation that are probably huge headaches, even in the RCT

SUTVA

- SUTVA stands for “stable unit-treatment value assumption”
  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*
- Largely about interference when aggregating but also poorly defined treatments and scale

SUTVA: No spillovers to other units

- What if we impose a treatment at one neighborhood but not a contiguous one?
- Treatment may spill over causing $Y = Y^1$ even for the control units because of spillovers from treatment group
- Can be mitigated with careful delineation of treatment and control units so that interference is impossible, may even require aggregation (e.g., classroom becomes the unit, not students)

SUTVA: No Hidden Variation in Treatment

- SUTVA requires each unit receive the same treatment dosage; this is what it means by “stable” (i.e., notice that the super scripts contain either 0 or 1, not 0.55, 0.27)
- If we are estimating the effect of aspirin on headaches, we assume treatment is 200mg per person in the treatment
- 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*; you may have to think of it as multiple arms

SUTVA: Scale can affect stability of treatment effects

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)

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CONSUMER HETEROGENEITY AND PAID SEARCH EFFECTIVENESS: A LARGE-SCALE FIELD EXPERIMENT

BY THOMAS BLAKE, CHRIS NOSKO, AND STEVEN TADELIS[1]

Internet advertising has been the fastest growing advertising channel in recent years, with paid search ads comprising the bulk of this revenue. We present results from a series of large-scale field experiments done at eBay that were designed to measure the causal effectiveness of paid search ads. Because search clicks and purchase intent are correlated, we show that returns from paid search are a fraction of non-experimental estimates. As an extreme case, we show that brand keyword ads have no measurable short-term benefits. For non-brand keywords, we find that new and infrequent users are positively influenced by ads but that more frequent users whose purchasing behavior is not influenced by ads account for most of the advertising expenses, resulting in average returns that are negative.

KEYWORDS: Advertising, field experiments, causal inference, electronic commerce, return on investment, information.

1. INTRODUCTION

ADVERTISING EXPENSES ACCOUNT for a sizable portion of costs for many companies across the globe. In recent years, the Internet advertising industry has grown disproportionately, with revenues in the United States alone totaling $36.6 billion for 2012, up 15.2 percent from 2011. Of the different forms of Internet advertising, paid search advertising, also known in industry as "search engine marketing" (SEM), remains the largest advertising format by revenue, accounting for 46.3 percent of 2012 revenues, or $16.9 billion, up 14.5 percent from $14.8 billion in 2010. Google Inc., the leading SEM provider, registered $46 billion in global revenues in 2012, of which $43.7 billion, or 95 percent, were attributed to advertising.[2]

Internet advertising facts

- In 2012, revenues from Internet advertising was $36.6 billion and has only grown since
- Paid search (“search engine marketing”) is the largest format by revenue (46.3% of 2012 revenues, or $16.9 billion)
- Google is leading provider (registered $46 billion in global revenues in 2012 of which 95% was attributed to advertising)

Selection bias

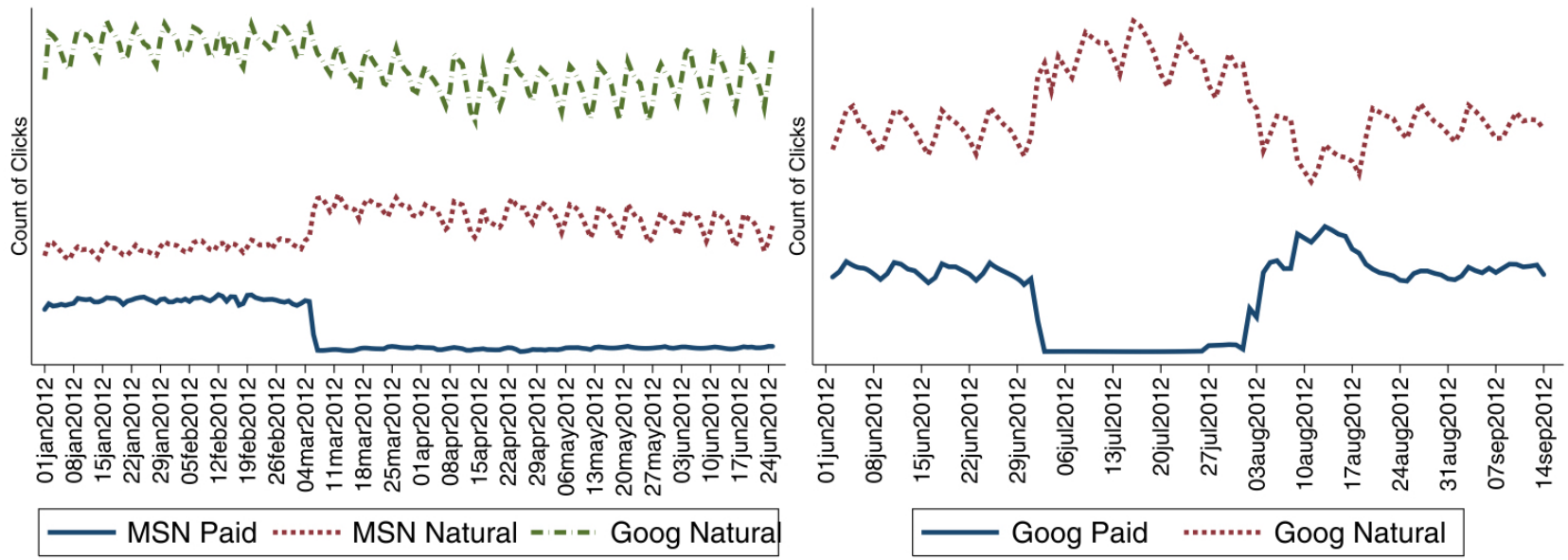
- Treatment was targeted ads at particular people conducting particular types of keyword search
- Consumers who choose to click on ads are loyal and already informed about products with high likelihood to buy already
- Problem is ads are targeting people at the end of their search, so the question is whether they would've found it already (i.e., $E[Y^0|D=1] \neq E[Y^0|D=0]$)

Selection bias

- Estimated return on investment using OLS found ROI of over 1600%
- Compared this to experimental methods and found ROI of -63% with a 95% CI of $[-124\%, -3\%]$, rejecting the hypothesis that the channel yielded short-run positive returns
- Think back to perfect doctor – Even without the treatment (Y^0), the treated group observationally would've still found a way

Natural experiment

- Study began with a naturally occurring and somewhat fortuitous event at eBay
- eBay halted SEM queries for brand words (i.e., queries that included the term eBay) on Yahoo! and Microsoft but continued to pay for these terms on Google
- Blake, Nosky and Tadelis (2015) showed almost all of the foregone click traffic and attributed sales were captured by natural search
- Substitution between paid and unpaid traffic was nearly one to one complete



(a) MSN Test

(b) Google Test

FIGURE 2.—Brand keyword click substitution. MSN and Google click-traffic counts to eBay on searches for 'ebay' terms are shown for two experiments where paid search was suspended (panel (a)) and suspended and resumed (panel (b)).

Interpretation of natural experiment

"The evidence strongly supports the intuitive notion that for brand keywords, natural search is close to a perfect substitute for paid search, making brand keyword SEM ineffective for short-term sales. After all, the users who type the brand keyword in the search query intend to reach the company's website, and most likely will execute on their intent regardless of the appearance of a paid search ad."

Selection bias

Observational data masked causal effect (recall the decomposition of the any non-designed estimation strategy)

> *“Advertising may appear to attract these consumers, when in reality they would have found other channels to visit the company’s web-site. We overcome this endogeneity challenge with our controlled experiments.”*

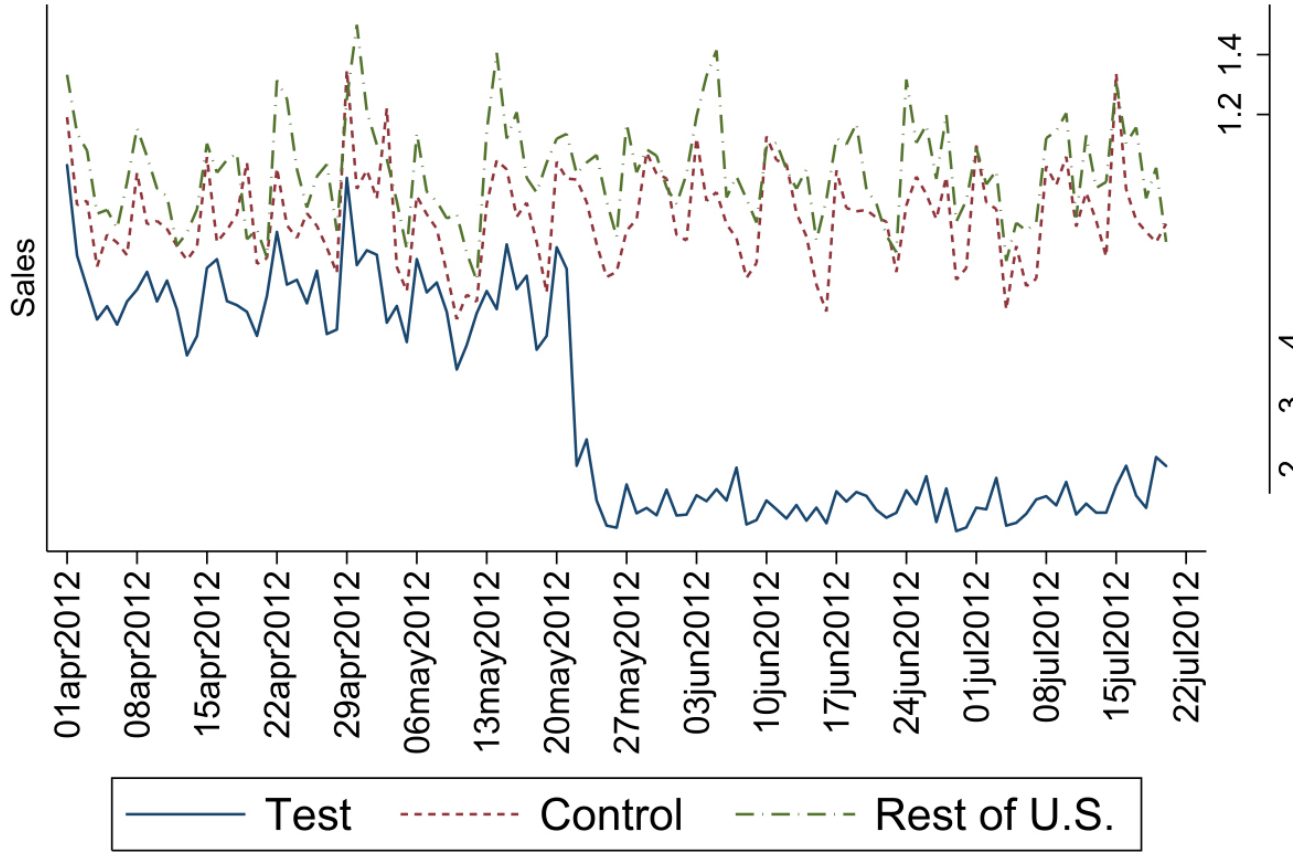
RCT

Natural experiment was valuable, but eBay could run a large scale RCT.

Use this finding of a nearly one-to-one substitution once paid search was dropped to convince eBay to field a large scale RCT discontinuing non-band key words

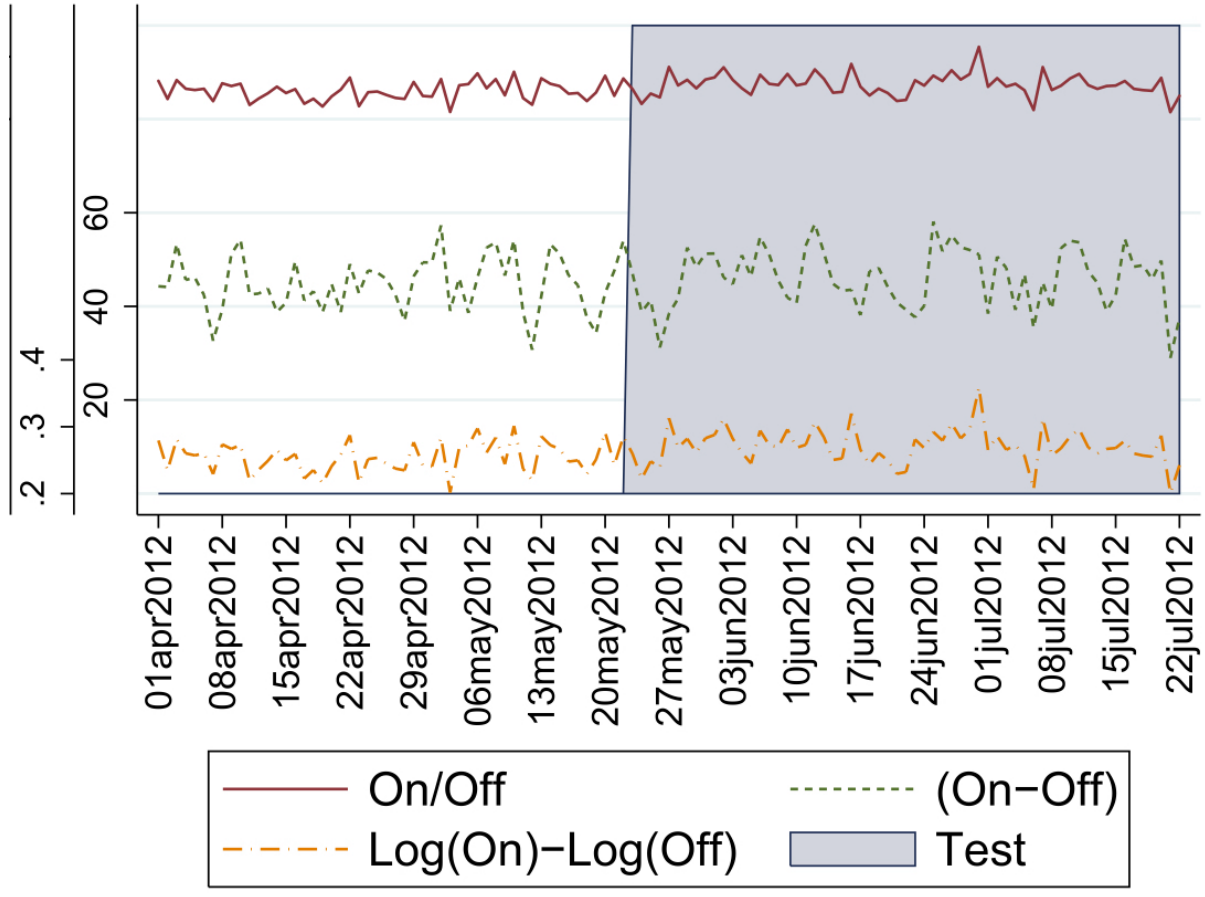
Design of the experiment

- Randomly assigned 30 percent of eBay's US traffic to stop all bidding for all non-brand keywords for 60 days
- Some random group of users, in other words, were exposed to ads; a control group did not see the ads
- Used Google's geographic bid feature that can accurately identify geographic market of the user conducting the search
- Ads were suspended in 30 percent of markets to reduce the scope of the test and minimize the potential cost and impact to the business



(a) Attributed Sales by Region

Figure: Attributed sales due to clicking on a Google link (treatment group)



(b) Differences in Total Sales

Figure: Differences in total sales by market (treatment to control)

	OLS	
	(1)	(2)
Estimated Coefficient	0.88500	0.12600
(Std Err)	(0.0143)	(0.0404)
DMA Fixed Effects		Yes
Date Fixed Effects		Yes
N	10,500	10,500
$\Delta \ln(Spend)$ Adjustment	3.51	3.51
$\Delta \ln(Rev)$ (β)	3.10635	0.44226
Spend (Millions of $)	$51.00	$51.00
Gross Revenue (R′)	2,880.64	2,880.64
ROI	4,173%	1,632%
ROI Lower Bound	4,139%	697%
ROI Upper Bound	4,205%	2,265%

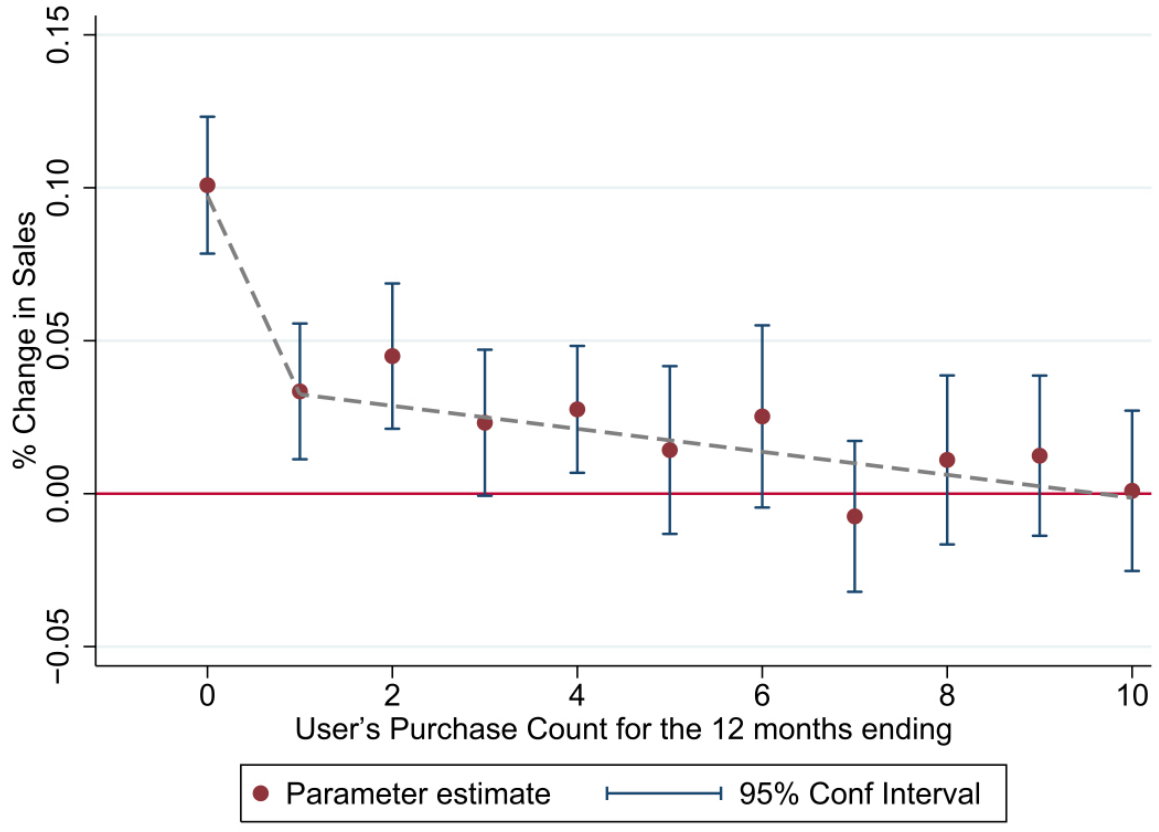
Figure: Spending effect on revenue using OLS but not the randomization. Effects are gigantic.

	(5)
Estimated Coefficient	0.00659
(Std Err)	(0.0056)
DMA Fixed Effects	Yes
Date Fixed Effects	Yes
N	23,730
$\Delta \ln(Spend)$ Adjustment	1
$\Delta \ln(Rev)$ (β)	0.00659
Spend (Millions of $)	$51.00
Gross Revenue (R′)	2,880.64
ROI	−63%
ROI Lower Bound	−124%
ROI Upper Bound	−3%

Figure: Spending effect on revenue using the randomization. Effects are negative.

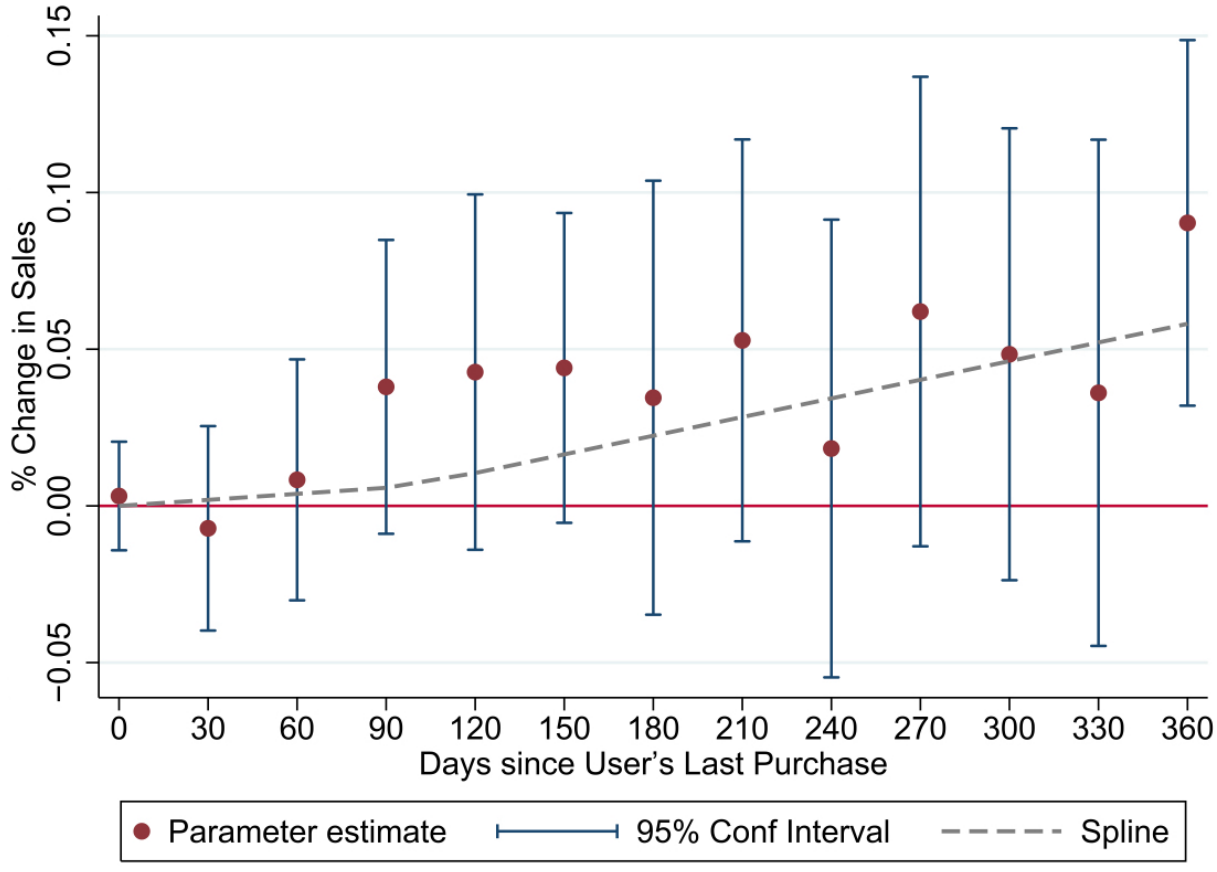
Heterogenous treatment effects

- Recall how the potential outcomes model explicitly models individual treatment effects could be unique and that the perfect doctor showed selection on gains masked treatment effects, perhaps even reversing sign
- Search advertising in this RCT only worked if the consumer had no idea that the company had the desired product
- Large firms like eBay with powerful brands will see little benefit from paid search advertising because most consumers already know that they exist, as well as what they have to offer



(a) User Frequency

Figure: Effects on new users are positive and large, but not others.



(b) User Recency

Figure: Effects are largest for "least active" customers.

Why are causal effects small?

- They suggest that the brand query tests found small causal returns because users simply substituted from the paid search clicks to the natural search clicks
- If that's the case, then it's explicitly a selection bias story

$$E[Y^0|D=1] \neq E[Y^0|D=0]$$

where D is being shown the branded advertisement based on search (i.e., they were already going there)

- They weren't using branded search for information; they were using to *navigate*

Self selection based on gains

- Potential outcomes is the foundation of the physical experiment because the physical experiment assigns units to treatments *independent* of potential outcomes, Y^0, Y^1
- This is important because outside of the physical experiment, we expect people select those important treatments based on whether, subjectively, they think $Y^1 > Y^0$ or $Y^1 \leq Y^0$.
- Rational actors almost by definition are thought to “self-select into treatment” making non-designed comparisons potentially misleading – sometimes by a little, sometimes by a lot

Comments

- Natural experiments are valuable, but they don't always have the same certainty the way an RCT does
- We use natural experiments when people won't let us run the RCTs we want to run!
- Findings from natural experiments often push others to run RCTs – like at eBay

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 SDO, we might estimate:

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

Why Control Variables?

- 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.
- Ongoing work by econometricians is investigating this more carefully

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

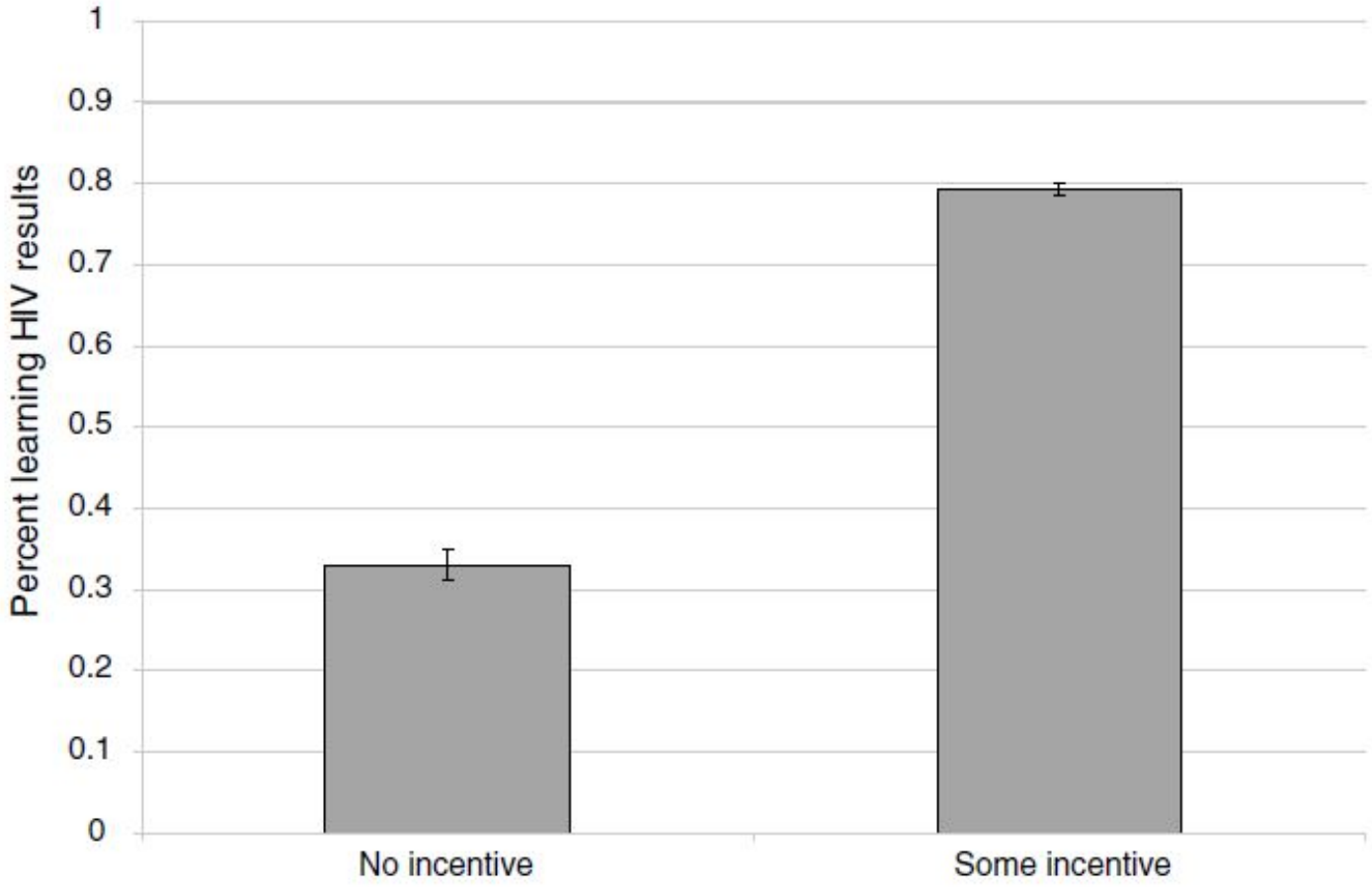


Figure: Visual representation of cash transfers on learning HIV test results.

Results

- Any incentive increases learning HIV status by 43 percentage points compared to the control (34% of controls learned HIV status)
- Next she looks at the effect that learning HIV status has on risky sexual behavior
- She had to do a lot of planning by creating two sources of randomization – the voucher and the distance to clinics – which required using instruments (we discuss it next week)

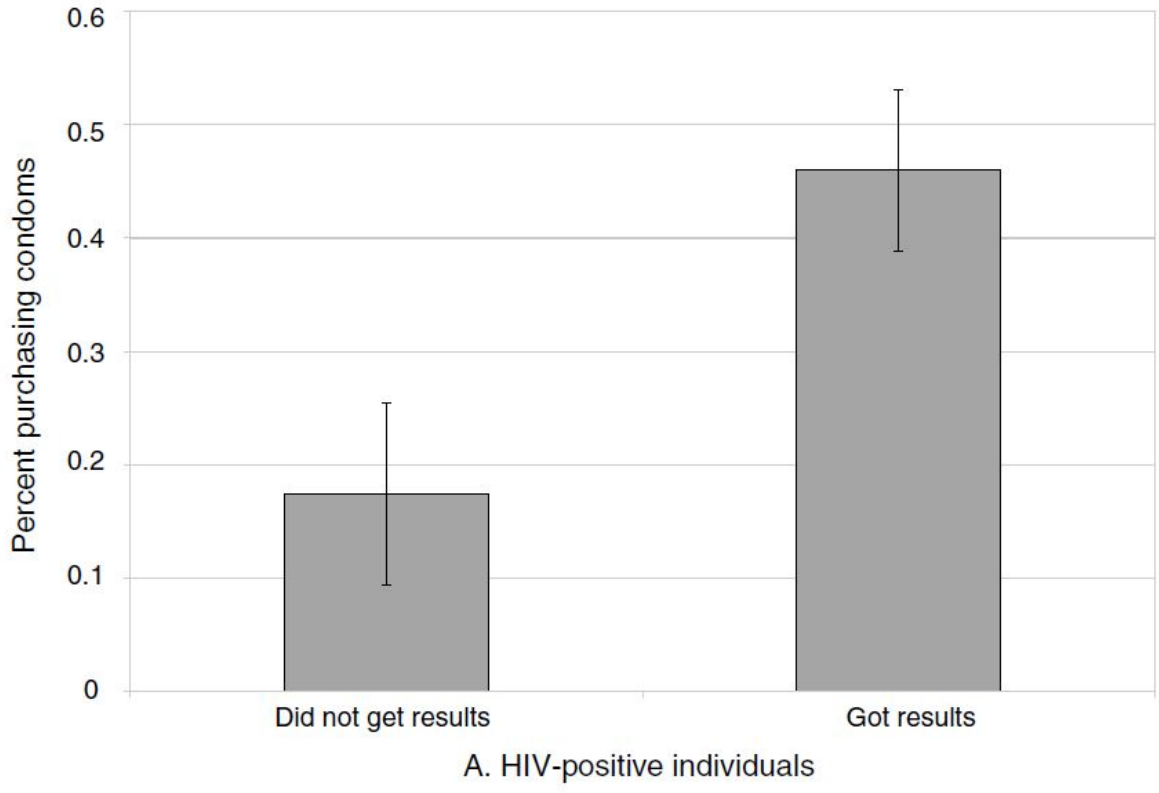


Figure: Visual representation of cash transfers on condom purchases for HIV positive individuals.

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

Thoughts you want to keep in mind

- Describe the way you would conduct the RCT by explaining the following:
  - → What's the treatment? Who will be treated? Who will not?
  - → Write down a regression with a binary variable as sometimes that alone will clarify it
  - → What is the outcome you are interested in?
  - → How will you assign this so that SUTVA holds and independence is achieved?
- Describe the steps you would take to do this if you had all the money in the world

Comment

- Methods do not drive the question
- Questions drive the methods
- Don’t lose sight of the ball – the importance of the questions should be what motivate you

Roadmap

- Introduction to course
- Potential outcomes
  - Naive causal inference
  - Potential outcomes notation
  - Selection bias
  - Independence
  - Example of physical experimentation: eBay advertising
  - Example of physical experimentation: HIV status
- Randomization inference
  - Lady tasting tea
  - Fisher’s sharp null
  - Alternative test statistics

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

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

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

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 δ_{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	δ_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):

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

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

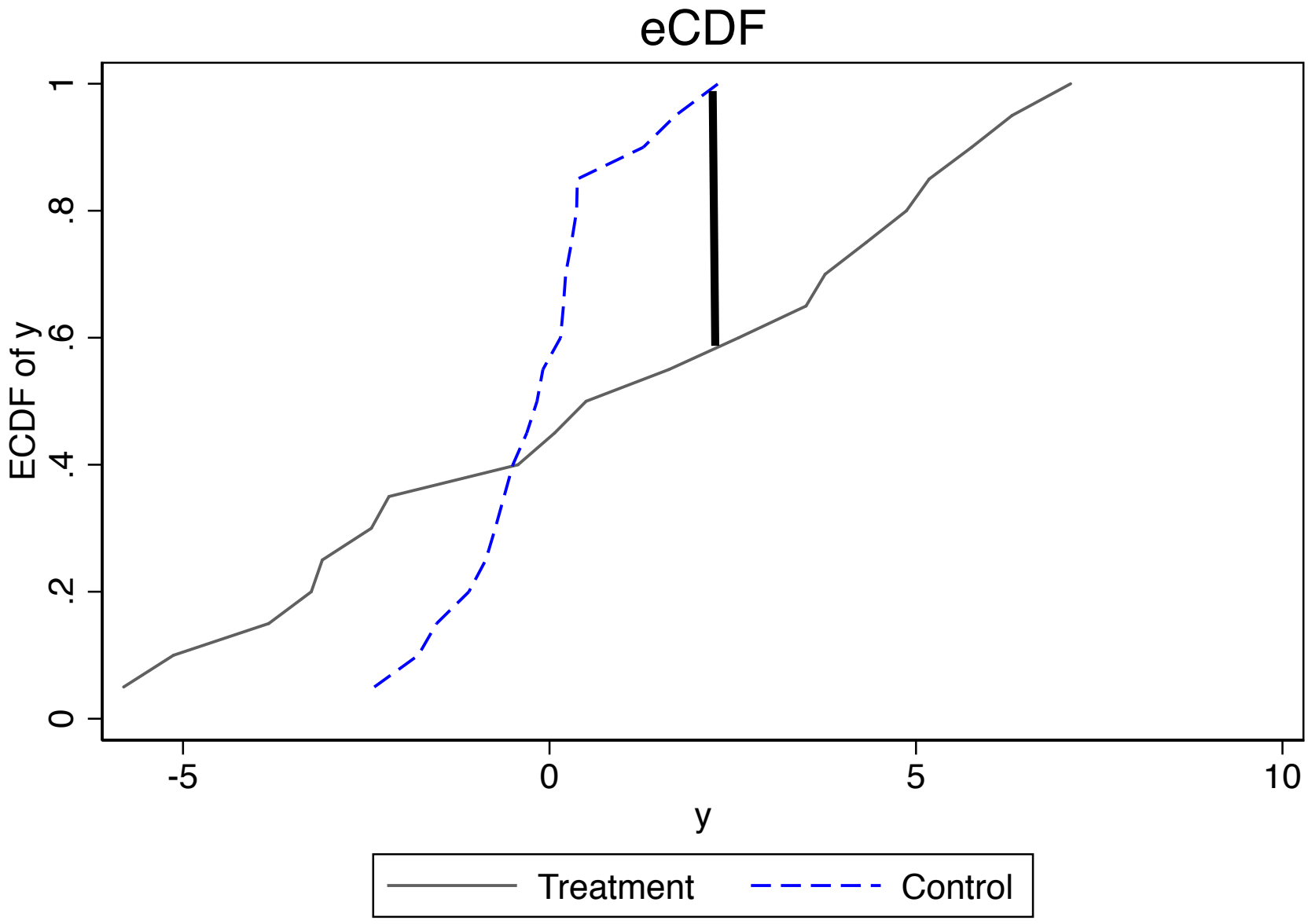
- Proportion of observed outcomes below a chosen value for treated and control separately
- If two distributions are the same, then $\widehat{F}_C(Y) = \widehat{F}_T(Y)$

Kolmogorov-Smirnov statistic

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

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

eCDFs by treatment status and test statistic



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 1.2641061×10^{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 α (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
  - → Take K samples from the treatment assignment space
  - → Calculate the randomization distribution in the K samples
  - → 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)

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`)

Concluding remarks

- Randomization inference is very common, particularly useful you don't want to make strong assumptions (parametric free)
- It's an area of continual examination by statisticians and econometricians, both in the experimental design and the quasi-experimental design
- We will use it primarily in my workshops with synthetic control, but it's going to be one you encounter and valued because of the non-parametric nature of it