

Causal Inference I

MIXTAPE SESSION



Roadmap

Introduction to course

- What is Mixtape Sessions?

- Managing your expectations

- Future Tracks

Potential outcomes

- Naive causal inference

- Potential outcomes notation

- Selection bias

- Independence

- Industry example of RCT: eBay advertising

- Policy example of RCT: HIV status

Welcome!

- Scott Cunningham, professor of economics at Baylor University, author of Causal Inference: the Mixtape
- I run workshops on causal inference all over the world, but I am not an econometrician – I'm a run of the mill applied microeconomist who studies severe mental illness who just happens to love learning and teaching
- Workshops can be helpful ways to plug into one's methodological training, and online workshops are very helpful because of the recordings, the coding together, and bunch of bells and whistles (e.g., github repositories)
- Causal inference is an old field but which has increasingly drawn people to it (Nobel Prize two years ago maybe helped)

What is Mixtape Sessions?

- Mixtape Sessions is my online platform started in November 2022 to “democratize causal inference” by helping connect people, from beginner to advanced, with material and teachers that for various reasons may not be accessible otherwise
- I became obsessed with teaching and writing about causal inference because of a strong conviction that (a) it is important and (b) not everyone had the same chances to learn it
- I wrote a 3-part sociological history of it on my substack that I encourage you to read so you can better understand the context for why I say that
- I tend to emphasize intuition, mechanics, narrow calculations, meaning, assumptions, code including actually taking time to code, advocate for data visualization – in other words the art and the science

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 to get there when an econometrician would do it much faster

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

Github repo

- We will communicate with one another regularly in the Discord channel and I will always be monitoring it
- Encourage you to talk to each other there, help one another, network with one another, coauthor with one another!
- I will be distributing things to you, like code and slides, via the github repo: github.com/Mixtape-Sessions/Causal-Inference-1
- Each lecture will be recorded and then uploaded to Vimeo as a password protected file that you'll have access to into perpetuity
- Kyle Butts and I are committed to over time making the Github Repository like an open public library where the only club goods are (a) recordings, (b) Discord and (c) live lectures

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

Today: Day 1

1. Potential outcomes
2. Randomization inference
3. Directed acyclic graphs

What's coming this spring?

- Lots of great “Mixtape Tracks” – shorter workshops taught by top professors at Brown, MIT, BYU and elsewhere
- Think of Mixtape Sessions as a bridge to more content and more people

<https://www.mixtapesessions.io/sessions/>

Causal Inference Part 2

Causal Inference II

Prof. Scott Cunningham • Starting March 18th

Causal inference Part II is a 4-day workshop in design based causal inference series. It will cover two contemporary research designs in causal inference -- difference-in-differences (including with covariates) and synthetic control. Each day is 8 hours with 15 minute breaks on the hour plus an hour for lunch. We will review the theory behind each design, go into detail on the intuition of the estimation strategies and identification itself, as well as explore code in R and Stata and applications using these methods. The goal as always is that participants leave the workshop with competency and confidence. This class will be a sequel to the 4-day workshop on Causal Inference Part I.

Causal Inference Part 2

Our workshop will bring you to the *cutting edge*

March 18th

Day 1 • 8am-5pm CST



Introduction to DID



Parallel trends and the DID equation



OLS, TWFE, Triple difference

March 19th

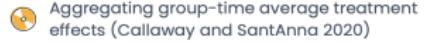
Day 2 • 8am-5pm CST



DID with Covariates (Sant'Anna and Zhao 2020; Abadie 2005)



Differential timing and Bacon decomposition (Goodman-Bacon 2021)



Aggregating group-time average treatment effects (Callaway and SantAnna 2020)

March 25th

Day 3 • 8am-5pm CST



Analyzing event studies (Sun and Abraham 2020)



Imputation Estimators (Gardner 2021 and Borusyak et al 2021)

March 26th

Day 4 • 8am-5pm CST

Continuous Treatment (Callaway, Goodman-Bacon and Sant'Anna 2020)

Synthetic control (Abadie, Diamond and Hainmueller 2010)

Augmented synthetic control (Ben-Michael, Feller, Rothstein 2022)

Machine Learnings and Causal Inference

Machine Learning and Causal Inference

Prof. Brigham Frandsen • Starting February 23rd

Machine Learning's wheelhouse is out-of-sample prediction, but these powerful methods can be deployed in service of causal inference. This two-session workshop will introduce the basics of machine learning prediction methods, including lasso and random forests and how they feature in causal inference methods like double machine learning (DML) and post-double selection lasso (PDS lasso). The course covers the conceptual and theoretical basis for the methods and also gets into the nuts and bolts of implementation in python and Stata using real-world data.

Machine Learnings and Causal Inference

Our workshop will bring you to the *cutting edge*

February 23rd

Day 1 • 6pm-9pm EST

-  Question your questions (are you asking a predictive or a causal question?)
-  What is causality?
-  What is prediction and how does it differ from causality?
-  Review standard tools of causal inference (concepts + python/Stata)
-  RCT gold standard
-  Multiple regression
-  Introduce ML prediction tools (concepts + python/Stata)
-  Prediction objective
-  Bias-variance tradeoff
-  Lasso
-  Random forest

February 24th

Day 2 • 6pm-9pm EST

-  Put ML to work in service of causality (concepts + python/Stata)
-  Post-double selection lasso (PDS)
-  Double/de-biased machine learning (DML)

Advanced DID

Advanced DID

Prof. Jonathan Roth • Starting April 21st

This one-day workshop will cover advanced topics from the recent difference-in-differences literature. One question of particular focus will be, *"what should I do if I'm not 100% sure about the validity of the parallel trends assumption?"* We will cover a variety of relaxations to the parallel trends assumption, and new tools for power calculations and sensitivity analysis. The workshop will focus not just on the theory, but also on practical implementation in statistical software such as R and Stata.

Advanced DID

Our workshop will bring you to the cutting edge

April 21st

Day 1 • 9am-5pm EST



Canonical Difference-in-Differences



Staggered Treatment Timing



Violations of Pre-Trends

Doing Applied Research

Doing Applied Research

Prof. Daniel Rees and Prof. D. Mark Anderson • Starting May 4th

This course is intended to be a practical guide for graduate students and early career economists doing applied research. The nuts and bolts of writing, publishing, and service to the profession are covered in two half-day sessions, each lasting roughly four hours (including short breaks). We begin by providing tips on how to start a research project, when to switch topics, and how to effectively manage multiple projects at once. Next, we provide practical advice on how to write an applied economics paper, from structuring the introduction to crafting the conclusion. The second half of the course takes participants through the publication process. In addition, we discuss networking, refereeing for economics journals, getting the most out of conferences, and how to successfully navigate the academic job market.

REGISTER TODAY

Sign-up today to ensure access to this workshop.

[REGISTER TODAY](#)

\$125 USD + Fees

Doing Applied Research

Our workshop will bring you to the cutting edge

May 4th

Day 1 • 12pm-4:30pm EST

-  Starting Your Research Project

-  Practical Tips for Writing Your Applied Paper

-  Grad Student Job Market Q&A

May 5th

Day 2 • 12pm-4pm EST

-  The Publication Process

-  Refereeing

-  Conferences and Networking

-  Ask the editor!

-  Grad Student Job Market Q&A

Synthetic control and Clustering

Synthetic Control and Clustering

Prof. Alberto Abadie • Starting April 27th

In this course, we will cover the fundamentals of synthetic control estimation and inference, with special emphasis on actionable guidance for applied research. We will discuss seven crucial guiding principles for empirical studies using synthetic controls and how these principles are applied in practice. Towards the end of the course, we will change topics to address "the" FAQ of econometrics office hours: When and how should we cluster standard errors?

Synthetic control and Clustering

Our workshop will bring you to the *cutting edge*

April 27th

Day 1 • 6pm–9pm EST



Synthetic Control

April 28th

Day 2 • 6pm–9pm EST



Clustering

Who will be hosting *this session*?



Alberto Abadie is an econometrician and empirical microeconomist with broad disciplinary interests. Professor Abadie received his Ph.D. in Economics from MIT in 1999. Upon graduating, he joined the faculty at the Harvard Kennedy School, where he was promoted to full professor in 2005. He returned to MIT in 2016, where he is Professor of Economics and Associate Director of the Institute for Data, Systems, and Society (IDSS).

His research areas are econometrics, statistics, causal inference, and program evaluation. Professor Abadie's methodological research focuses on econometric methods to estimate causal effects and, in particular, the effects of public policies, such as labor market, education, and health policy interventions. He is Associate Editor of AER: Insights, and has previously served as Editor of the Review of Economics and Statistics and Associate Editor of Econometrica and the Journal of Business and Economic Statistics. He is a Fellow of the Econometric Society and a Member of the American Academy of Arts and Sciences.

Heterogenous treatment effects and machine learning

Machine Learning and Heterogeneous Effects

Prof. Brigham Frandsen • Starting May 15th

The holy grail of causal inference is the individual-level treatment effect: how would a particular patient respond to a drug? Which users will respond most to a targeted ad? Would a given student be helped or harmed by a classroom intervention? This session introduces machine learning tools for estimating heterogeneous treatment effects like random causal forests. The course goes over the theory and concepts as well as the nitty-gritty of coding the methods up in python, R, and Stata using real-world examples. This course can be taken as a follow-up to the Machine Learning and Causal Inference mixtape session, or as a stand-alone course.

REGISTER TODAY

Sign-up today to ensure access to this workshop.

WHAT'S INCLUDED

295 USD + Fees

Heterogenous treatment effects and machine learning

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www.mltapessions.com/presentations/het_effects_may15/

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Machine Learning and Heterogeneous Effects

Our workshop will bring you to the *cutting edge*

May 15th
Day 1 • 6pm–8pm EST

Machine Learning and Heterogeneous Effects

Who will be hosting *this session*?



Brigham Frandsen is an associate professor at Brigham Young University after completing his Ph.D. in Economics at MIT, where his dissertation focused on econometric methodology and labor economics. After his Ph.D., Dr. Frandsen was selected as a Robert Wood Johnson Scholar in Health Policy Research at Harvard University where he spent two years in residence furthering his research in econometrics and labor economics, as well as adding health policy to his research agenda. Dr. Frandsen's methodological research focuses on causal inference on distributional effects. He applies these methodologies to questions about the impact of labor market institutions and interventions on education and earnings outcomes. His health policy research deals with the consequences of fragmentation in the U.S. health care system. In addition to research, Dr. Frandsen enjoys hiking and mountain biking with his wife, Christine, and their four children.

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Josh Angrist on the negative results at the time (10 sec)

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Machine Learning and Heterogeneous Effects

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- Future Tracks

Potential outcomes

- Naive causal inference

- Potential outcomes notation

- Selection bias

- Independence

- Industry example of RCT: eBay advertising

- Policy example of RCT: HIV status

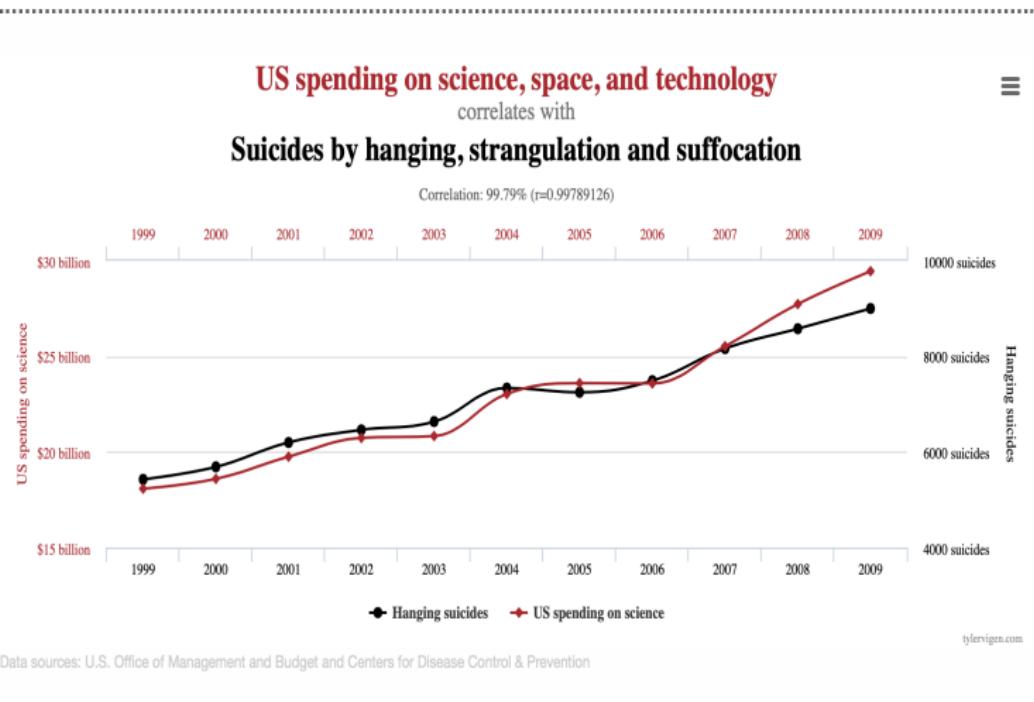
Coming to causal inference

- 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 i th variety on the k th 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.

Prediction		Causal Inference		Statistics/Econometrics	Machine Learning
		Observational			
ANOVA		Difference-in-Differences		Experimental	
Linear Regression		Instrumental Variables		A/B Testing	
Logistic Regression		Propensity Score Matching		Business Experimentation	
Time Series Forecasting		Regression Discontinuity		Randomized Controlled Trials	
Boosting		Additive Noise Models		Causal Reinforcement Learning	
Decision Trees & Random Forests		Causal Forests		Multiarmed Bandits	
Lasso, Ridge & Elastic Net		Causal Structure Learning		Reinforcement Learning	
Neural Networks		Directed Acyclic Graphs			
Support Vector Machines		Double/Debiased Machine Learning			

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:

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

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 $\hat{\delta}$ 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

$$E[Y^1|D = 1] = a$$

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

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

$$E[Y^0|D = 0] = d$$

$$\text{ATE} = e$$

Rewrite ATE definition

Rewrite ATE

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

Simple manipulation of ATE definition

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

$$e = \pi a + b - \pi b - \pi c - d + \pi d$$

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

$$0 = e - \pi a - b + \pi b + \pi c + d - \pi d - \mathbf{a} + \mathbf{a} - \mathbf{c} + \mathbf{c} - \mathbf{d} + \mathbf{d}$$

$$\mathbf{a} - \mathbf{d} = e - \pi a - b + \pi b + \pi c + d - \pi d + \mathbf{a} - \mathbf{c} + \mathbf{c} - \mathbf{d}$$

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

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

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

Carry forward from previous slide

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

Replace letters with original terms

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

Decomposition of the SDO

Decomposition of the SDO

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

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_Ewea7zQoLTFYHbnvqaTVDhn2GDzq30a6EQ/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.

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

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

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:

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

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

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

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

- If treatment is independent of potential outcomes, then:

$$E_N[y_i|d_i = 1] - E_N[y_i|d_i = 0] = E[Y^1] - E[Y^0]$$

$$SDO = ATE$$

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

Identification with Randomization

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

SDO is unbiased estimate of ATE with randomized treatment assignment because it sets selection bias to zero and $ATT = ATU$.

Different paths may have implications

Strange things when we discuss randomization with potential outcomes – for example, selection bias is zero because randomization distributed potential outcomes equally across treatment and control, but one of them doesn't exist!

$$\underbrace{E[Y^0|D=1]}_{\text{Doesn't exist}} = \underbrace{E[Y^0|D=0]}_{\text{Exists}}$$

Contrast that with people who come to randomization via experimental design tradition – randomization distributes *realized variables* equally across treatment and control – but note we never even discussed variables

I wonder if this is why some stop at RCT but some branch into quasi-experimental?

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**: *stable*
 2. **U**: across all *units*, or the population
 3. **TV**: *treatment-value* (“treatment effect”, “causal effect”)
 4. **A**: *assumption*
- 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)

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

BY THOMAS BLAKE, CHRIS NOSKO, AND STEVEN TADELIS¹

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

PAID SEARCH EFFECTIVENESS

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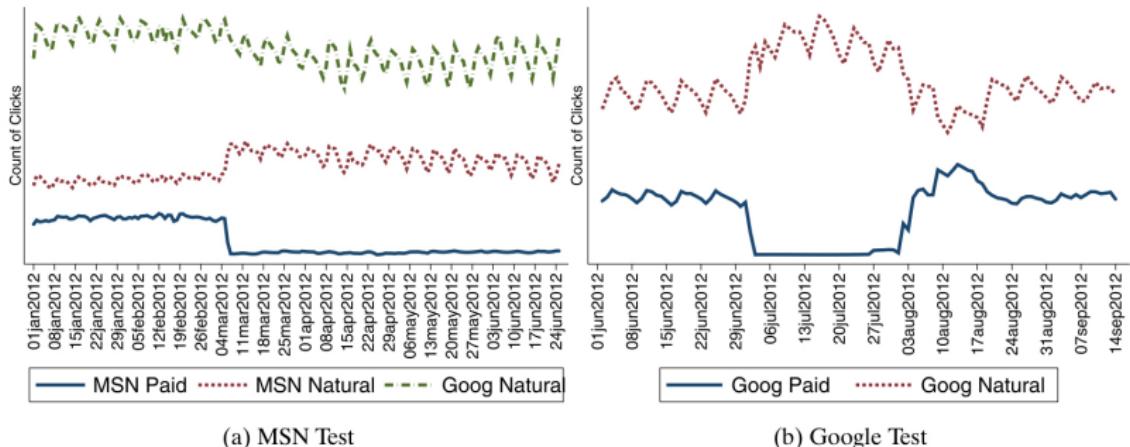


FIGURE 2.—Brand keyword click substitution. MSN and Google click-traffic counts to eBay on searches for ‘ebay’ terms are shown for two experiments where paid search was suspended (panel (a)) and suspended and resumed (panel (b)).

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 website. We overcome this endogeneity challenge with our controlled experiments."

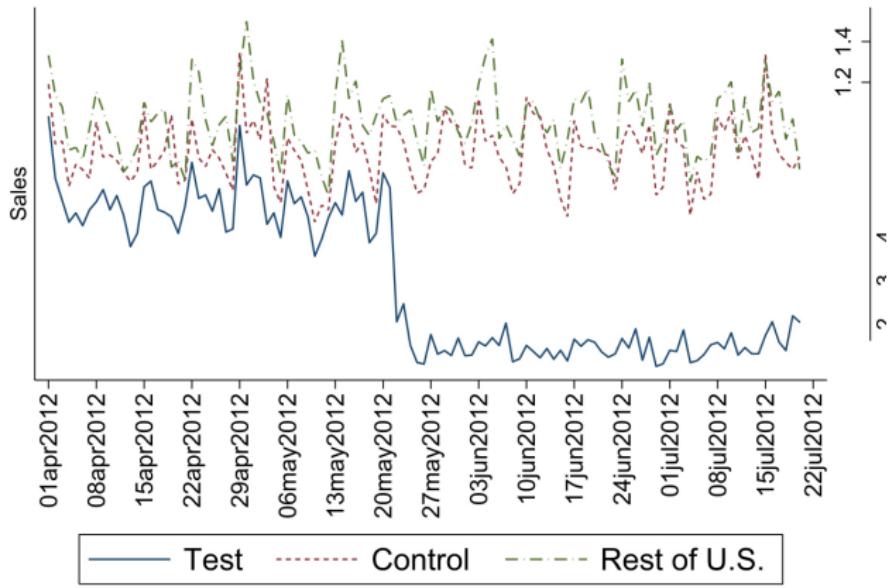
RCT

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

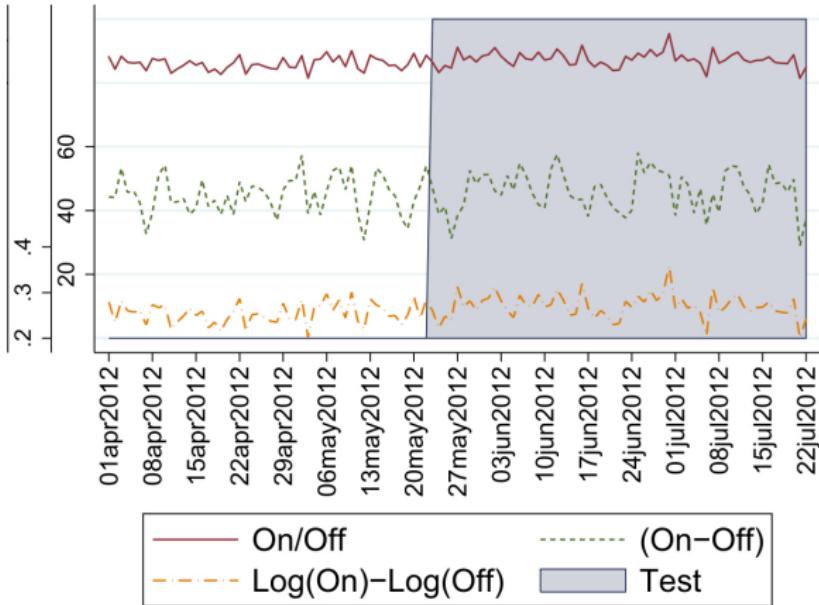
Design of the experiment

- 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
<i>N</i>	10,500	10,500
$\Delta \ln(Spend)$ Adjustment	3.51	3.51
$\Delta \ln(Rev)$ (β)	3.10635	0.44226
<i>Spend</i> (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%

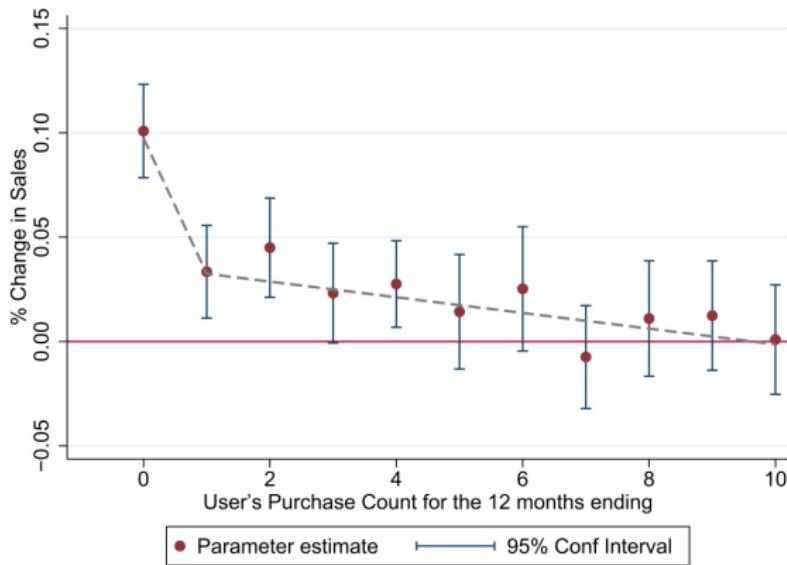
Figure: Spending effect on revenue using OLS but not the randomization.
 Effects are gigantic.

	(5)
Estimated Coefficient	0.00659
(Std Err)	(0.0056)
DMA Fixed Effects	Yes
Date Fixed Effects	Yes
<i>N</i>	23,730
$\Delta \ln(Spend)$ Adjustment	1
$\Delta \ln(Rev)$ (β)	0.00659
<i>Spend</i> (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.

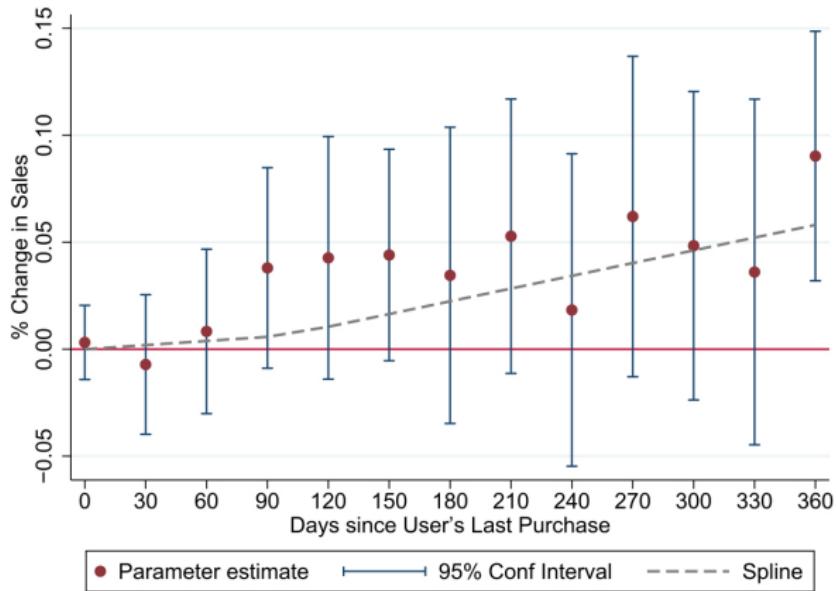
Heterogenous treatment effects

- 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*** (0.023)	0.309*** (0.026)	0.219*** (0.029)	0.220*** (0.029)	0.219 *** (0.029)
Amount of incentive		0.091*** (0.012)	0.274*** (0.036)	0.274*** (0.035)	0.273*** (0.036)
Amount of incentive ²			-0.063*** (0.011)	-0.063*** (0.011)	-0.063*** (0.011)
HIV	-0.055* (0.031)	-0.052 (0.032)	-0.05 (0.032)	-0.058* (0.031)	-0.055* (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

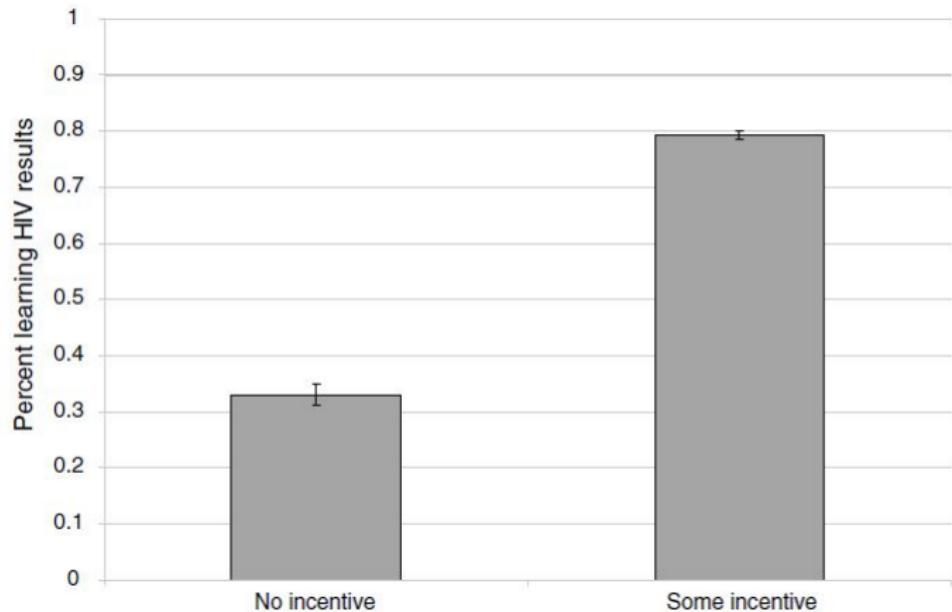


Figure: Visual representation of cash transfers on learning HIV test results.

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)

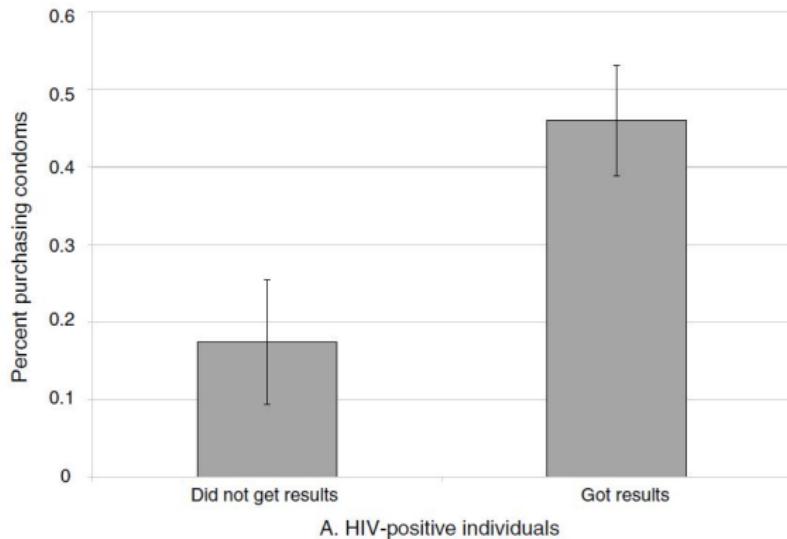


Figure: Visual representation of cash transfers on condom purchases for HIV positive individuals.

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 (0.025)	-0.069 (0.062)	-0.193 (0.148)	-0.303 (0.285)
Got results × HIV	0.418*** (0.143)	0.248 (0.169)	1.778*** (0.564)	1.689** (0.784)
HIV	-0.175** (0.085)	-0.073 (0.123)	-0.873 (0.275)	-0.831 (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