Week 9: Regression in the Social Sciences

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Princeton

November 14 and 19, 2018

[1] These slides are heavily influenced by Matt Blackwell, Justin Grimmer, Jens Hainmueller, Erin Hartman and Kosuke Imai.

Where We've Been and Where We're Going...

- Last Week
  - diagnostics
- This 'Week'
  - Wednesday:
    - making an argument in social sciences
    - causal inference
  - Monday:
    - more causal inference
    - visualization
- Next Week
  - selection on observables
- Long Run
  - probability $\rightarrow$ inference $\rightarrow$ regression $\rightarrow$ causal inference

Questions?

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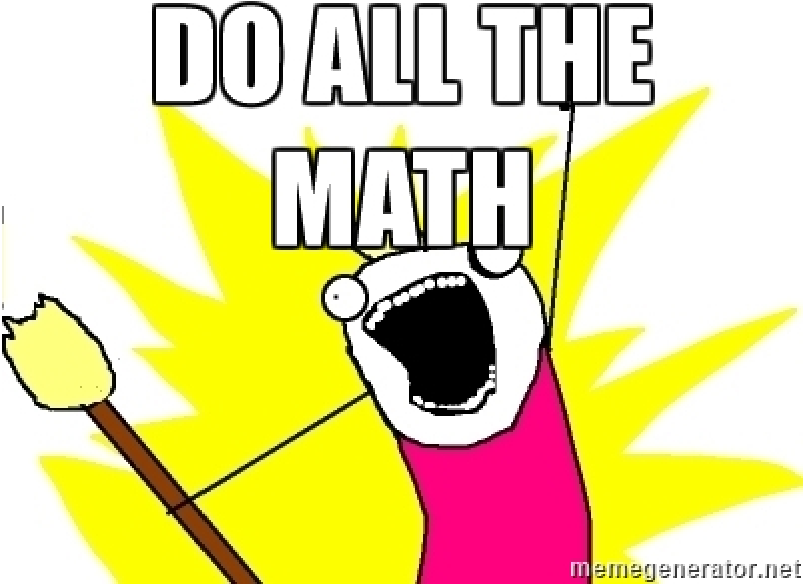
- Knowing how methods work also makes you a better reader of work.


DO ALL THE
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We will mostly talk about statistical methods here (it is a statistics class!) but the best work is a combination of substantive and statistical theory.

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- Like it or not, social science theories are almost always expressed as causal claims: e.g. "an increase in X causes an increase in Y" (or something more opaque meaning the same thing)
- The study of causal inference helps us understand the assumptions we need to make this kind of claim.

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- As we will see this is not a conversation about estimation: in other words, if someone answers "regression" they have made a category error

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- Even when identification is possible, estimation may impose additional assumptions (i.e. that the linear approximation to the CEF is good enough)
- Law of Decreasing Credibility (Manski): The credibility of inference decreases with the strength of the assumptions maintained

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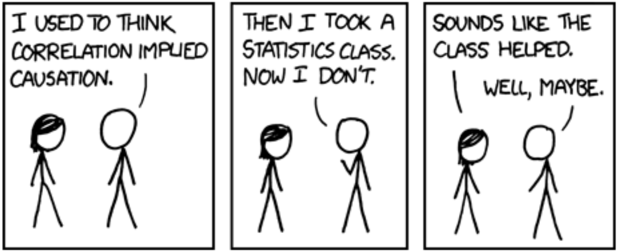
- Pearl (1995) develops graphical models for causal inference

Causation


I USED TO THINK CORRELATION IMPLIED CAUSATION.
THEN I TOOK A STATISTICS CLASS. NOW I DON'T.
SOUNDS LIKE THE CLASS HELPED.
WELL, MAYBE.

Potential Outcomes

Definitions:

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T_i: Dichotomous Treatment assignment for unit i (multi-valued treatments–just more potential outcomes for each unit)

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τ_i: The treatment effect

$$\tau_i = Y_i(1) - Y_i(0)$$

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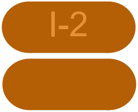

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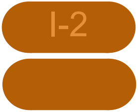

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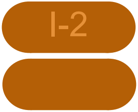

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Illustrated potential outcomes here and later courtesy of Erin Hartman

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Note that potential outcomes are thought of as fixed, and that they, and the difference between them, can vary by arbitrary amounts for each unit i. There is some true distribution of potential outcomes across the population.

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Treatment assignment is the source of randomness

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No methodology allows us to simultaneously observe both potential outcomes, $Y_i(1)$ and $Y_i(0)$, making τ_i unobservable–and unidentifiable without additional assumptions (Fundamental Problem of Causal Inference Holland (1986))

Causal Inference is a Missing Data Problem

Example: Asprin's Impact on Headaches

Patient i		Pill T_i	Headache Status Y_i(PLACEBO)	Y_i(I-2)	Y_i	Age X_{1i}	Academic X_{2i}
1						25	Y
2						55	N
3						62	Y
4						80	N
5						32	Y
6						45	B
⋮	⋮	⋮	⋮	⋮	⋮	⋮	⋮
n						71	N

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Step 1: (Randomly) sample units

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Step 2: Randomly assign treatment

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Step 3: Measure revealed potential outcome

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3			1	1		62	Y
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  - If this is not true, the number of potential outcomes for unit i grows
  - Ex: in an experiment with 3 units, if the potential outcomes for unit i depend on the treatment assignment of units j and k, the potential outcomes for unit i are defined by Y_{ijk}:

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- Same version of the treatment

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We also need to assume Positivity $0 < p(T_i) < 1 \forall i$

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This is highly implausible in most social science

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- Another way of thinking of it: The distributions of the potential outcomes $(Y_i(1), Y_i(0))$ are the same for the treatment and control group.

How do we proceed?

Identification by randomization:

- If treatment is randomized, then treatment is unrelated to any and all underlying characteristics, observed and unobserved
- Randomization therefore means treatment assignment is independent of the potential outcomes $Y_i(1)$ and $Y_i(0)$, i.e.

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- This is sometimes called unconfoundedness or ignorability
- Another way of thinking of it: The distributions of the potential outcomes $(Y_i(1),\ Y_i(0))$ are the same for the treatment and control group.
- Yet another way of thinking of it: The treatment and control group are exchangeable, or balanced (on observables and unobservables) on average

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- Conditioning set should yield $Y_i(0), Y_i(1)$ and T_i conditionally independent

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- Treatment effect heterogeneity: Zero ATE doesn't mean zero effect for everyone

Three Big Assumptions

1. SUTVA
2. Positivity
3. (Conditional) Ignorability

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- Selection bias: how different the treated and control groups are in terms of their potential outcome under control.

Selection Makes Us Care About Assignment Mechanisms

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Assignment Mechanism

"The process that determines which units receive which treatments, hence which potential outcomes are realized and thus can be observed, and, conversely, which potential outcomes are missing."
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No Causation Without Manipulation

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- Population parameter
- It is fixed and unchanging

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Average Treatment Effect

Imagine a study population with 4 units:

i	D_i	Y_{1i}	Y_{0i}	τ_i
1	1	10	4	6
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Note: Average effect is positive, but τ_i are negative for some units!

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- this means we have violated the assumption of unconfoundness $(Y(1), Y(0)) \perp D$

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Example: Gender Quotas and Redistribution Towards Women

- Countries with gender quotas are likely countries where women are politically mobilized.
- Given this difference, policies targeted towards women would be more common in quota countries even if these countries had not adopted quotas.

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Most statistical models of causal inference attain identification of treatment effects by restricting the assignment mechanism in some way.

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Always ask:
what is the experiment I would run if I had infinite resources and power?

Causal Inference Workflow

Causal Relationship

↓

Ideal Experiment

↓

Quantity of Interest

↓

Identification Strategy

↓

Data → Estimator → Estimate

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- Causal inference is a missing data problem: we typically make assumptions about the assignment mechanism to go from descriptive inference to causal inference.

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- Many, many, potential strategies for limiting bias

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- estimation of causal effects does not require identical treatment and control groups
- you need a clear counterfactual to have a well-defined causal effect (hence no causation without manipulation). For example of ‘the recession was caused by Wall Street’ may make intuitive sense but is it well-defined?

http://egap.org/methods-guides/10-things-you-need-know-about-causal-inference

1. Making Arguments
2. Potential Outcomes
3. Average Treatment effects
4. Graphical Models
5. Fun With A Bundle of Sticks
6. Visualization

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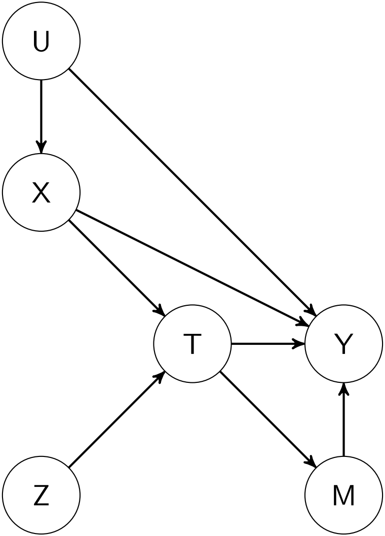
- Nice software that takes the graph and returns an identification strategy: DAGitty at `http://dagitty.net`

Components of a DAG



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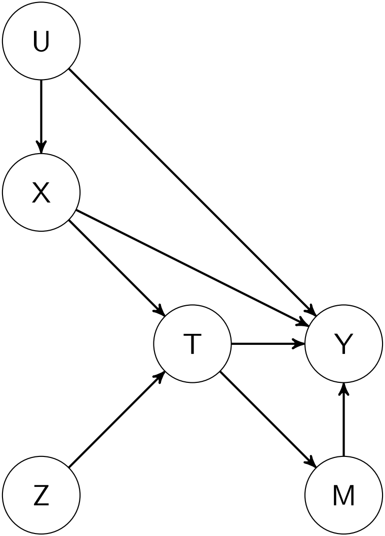

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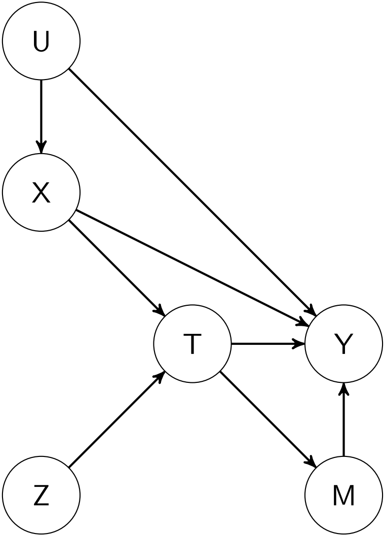

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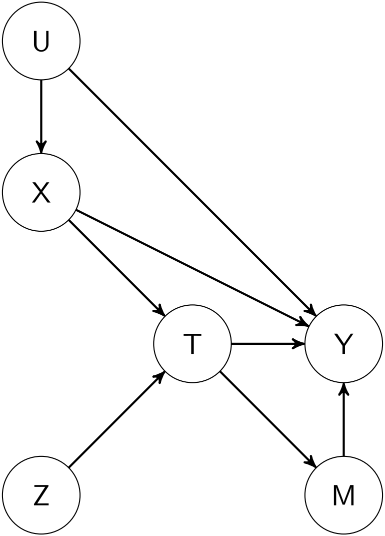

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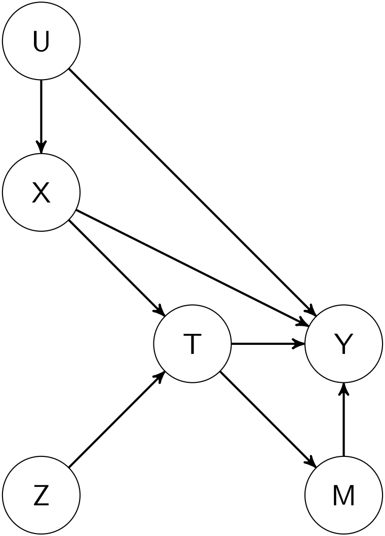

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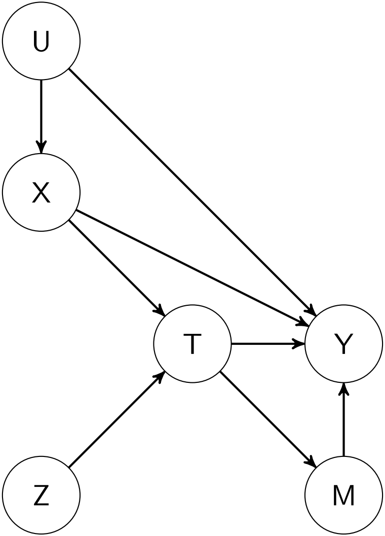

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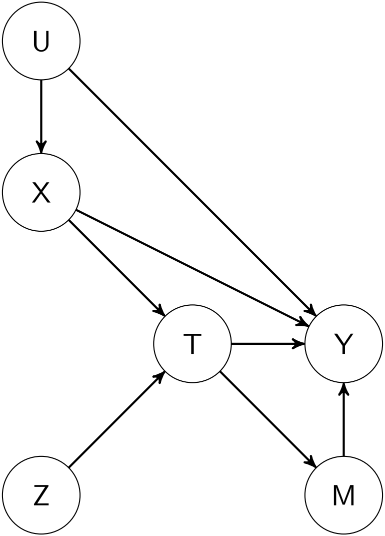

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- all relationships are non-parametric

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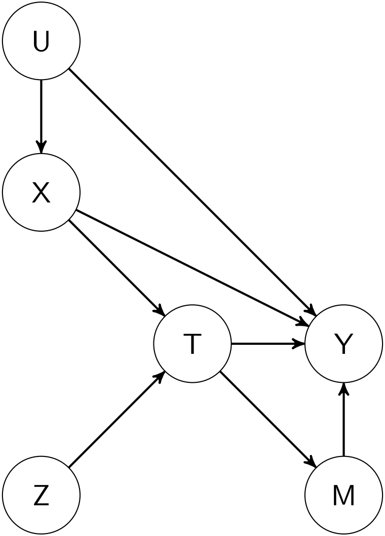

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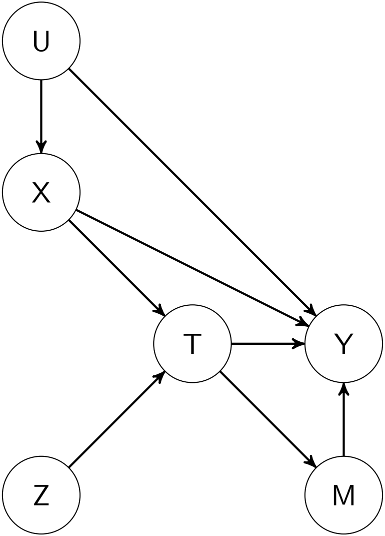

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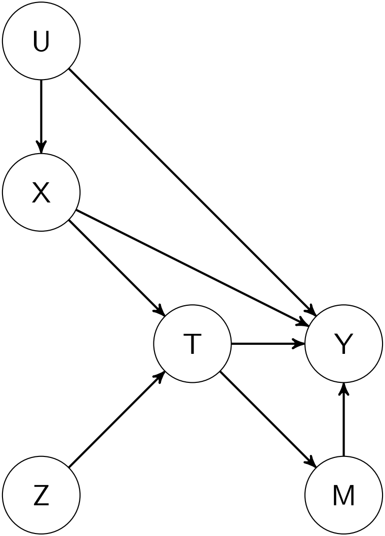

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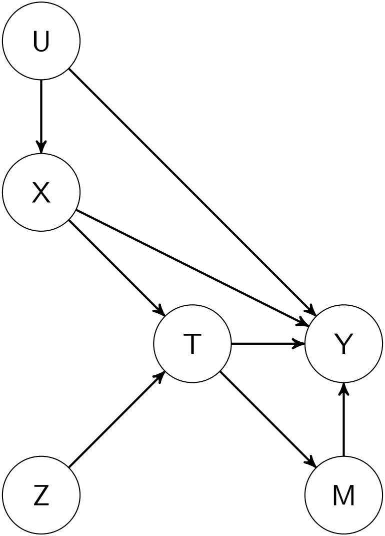

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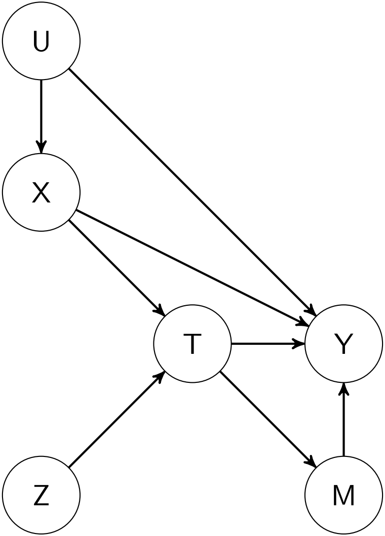

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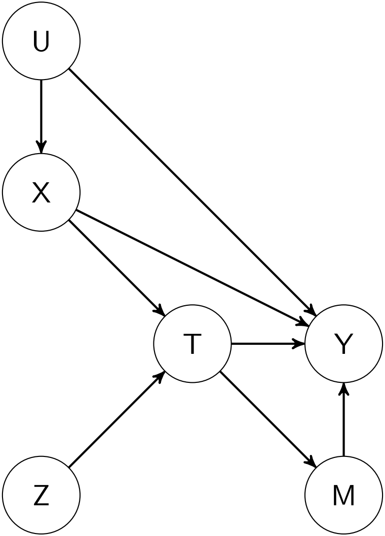

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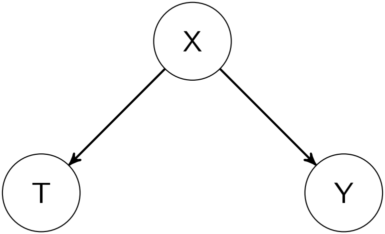
- We will talk in depth about two types of relationships: confounders and colliders

Confounders



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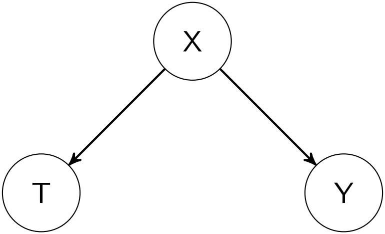

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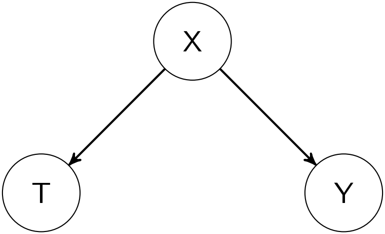

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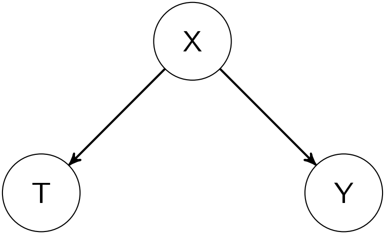

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- We can think of conditioning on a confounder as blocking the flow of association.

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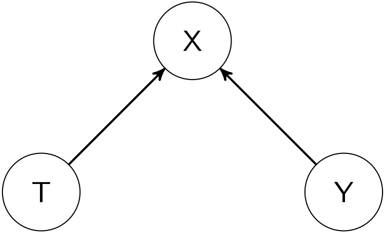

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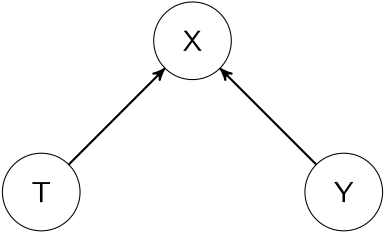

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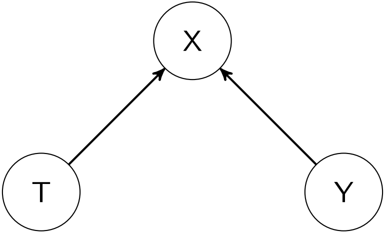

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- If we control for X they become associated and create a connection between T and Y

Colliders are scary because you can induce dependence

Endogenous Selection Bias: The Problem of Conditioning on a Collider Variable

Felix Elwert[1] and Christopher Winship[2]

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Annu. Rev. Sociol. 2014. 40:31–53

First published online as a Review in Advance on June 2, 2014

The *Annual Review of Sociology* is online at soc.annualreviews.org

This article's doi: 10.1146/annurev-soc-071913-043455

Keywords

causality, directed acyclic graphs, identification, confounding, selection

Abstract

Endogenous selection bias is a central problem for causal inference. Recognizing the problem, however, can be difficult in practice. This article introduces a purely graphical way of characterizing endogenous selection bias and of understanding its consequences (Hernán et al. 2004). We use causal graphs (direct acyclic graphs, or DAGs) to highlight that endogenous selection bias stems from conditioning (e.g., controlling, stratifying, or selecting) on a so-called collider variable, i.e., a variable that is itself caused by two other variables, one that is (or is associated with) the treatment and another that is (or is associated with) the outcome. Endogenous selection bias can result from direct conditioning on the outcome variable, a post-outcome variable, a post-treatment variable, and even a pre-treatment variable. We highlight the difference between endogenous selection bias, common-cause confounding, and overcontrol bias and discuss numerous examples from social stratification, cultural sociology, social network analysis, political sociology, social demography, and the sociology of education.

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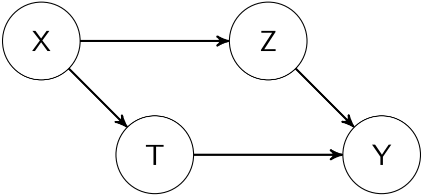

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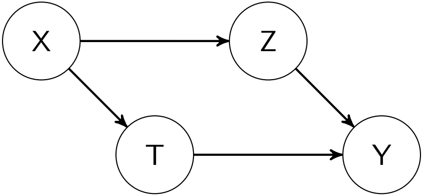

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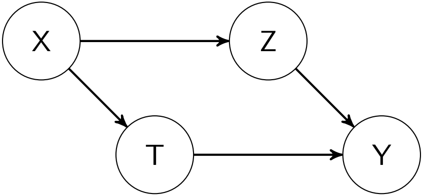

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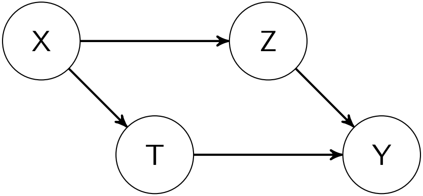

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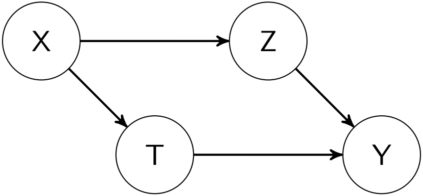

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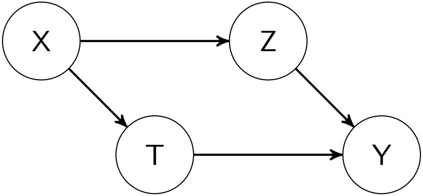

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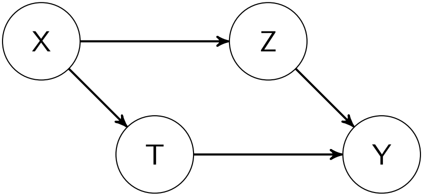

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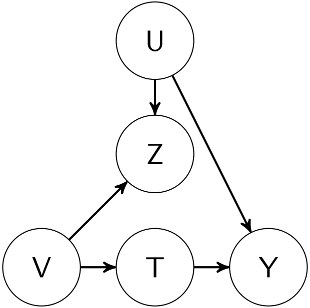
- We want to block the back-door path to leave only the causal effect

Colliders and Back-Door Paths



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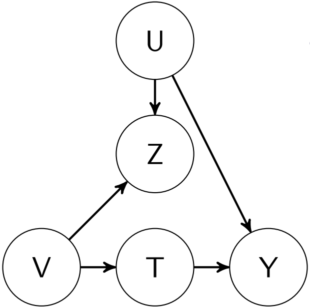

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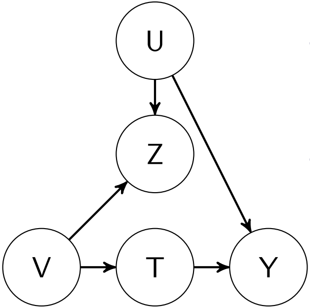

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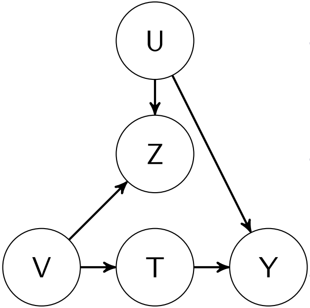
- Here we are fine unless we condition on Z which opens a path $T \leftarrow V \leftrightarrow U \rightarrow Y$ (this particular case is called M-bias)

Colliders and Back-Door Paths



- Z is a collider and it lies along a back-door path from T to Y
- Conditioning on a collider on a back-door path does not help and in fact causes new associations
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- So how do we know which back-door paths to block?

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- If A is not D-separated from B by C we say that A is *D*-connected to B by C

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- We will see some other approaches late in the semester.

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Fun with a Bundle of Sticks

Sen and Wasow (2016) “Race as a Bundle of Sticks: Designs that Estimate Effects of Seemingly Immutable Characteristics” *Annual Review of Political Science*.

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- Sen and Wasow argue that we can improve our empirical work on this by seeing race/ethnicity as a composite variable or 'a bundle of sticks' which can be manipulated separately

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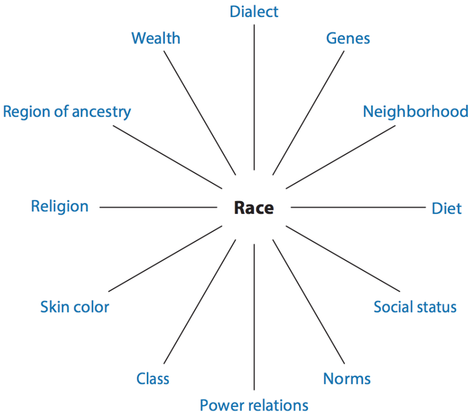
   - there is substantial variance across treatments which is a SUTVA violation

The Bundle of Sticks



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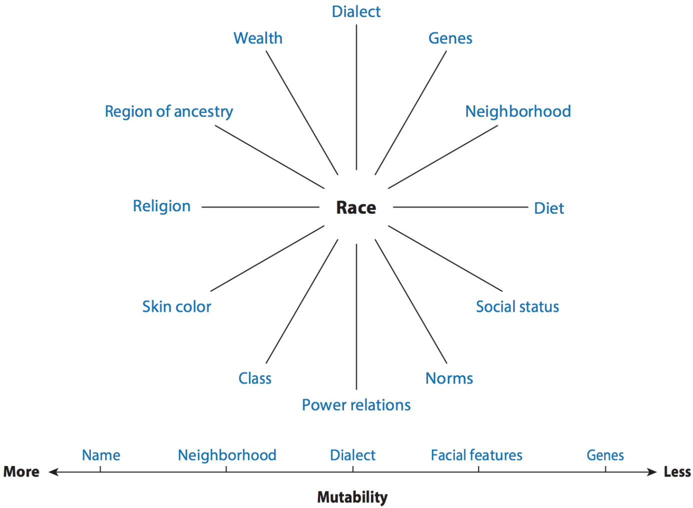

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  - Observational Studies (Greiner and Rubin 2010, Wasow 2012)

Design 2: Within-Group Studies

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- Example: Sharkey (2010) exploiting temporal variation in local homicides in Chicago to identify a significant neighborhood effect of proximity to violence on cognitive performance of African-American children

Concluding Thoughts

We can study race with causal inference, it just takes very careful design.

Table 2 Overview of exposure and within-group research designs

	Exposure	Within-Group
Unit	Individuals or institutions, potentially from any group	Members of a particular group
Typical treatment	Racial cue or signal (e.g., include distinctively ethnic names on a resume)	Constitutive element of the composite of race (e.g., address anxiety about social belonging in college)
Role of element of race	One "stick" is a proxy for the bundle (e.g., in a phone call with a landlord, dialect signals many traits associated with race)	One "stick" explains part of the bundle (e.g., Middle Passage might partly explain high rates of hypertension among African-Americans)
Examples	Correspondence and audit studies Implicit Association Tests	Experimental manipulation of a constitutive psychological dimension of race Within-race matching

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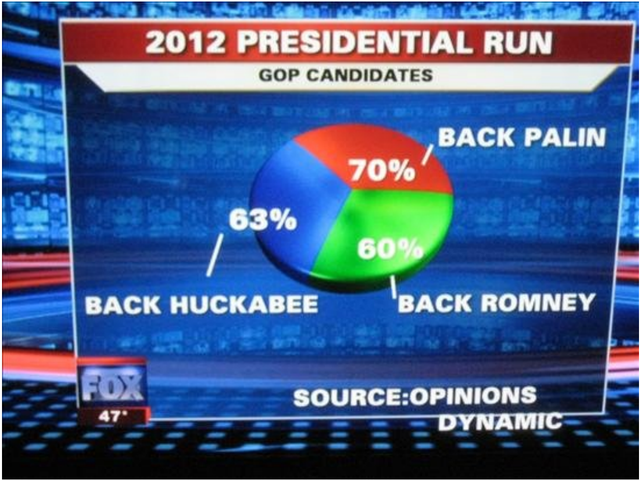

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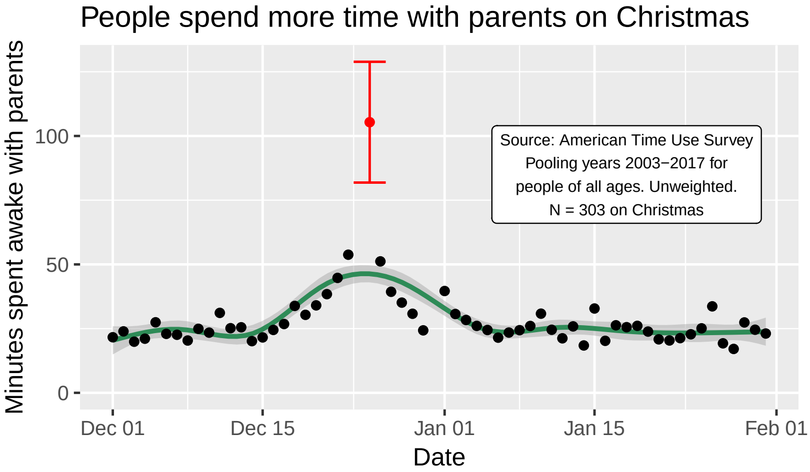
- I strongly recommend Kieran Healy's new visualization book — great summary of the fundamentals plus `R` code.

Examples



Source: Ian Lundberg

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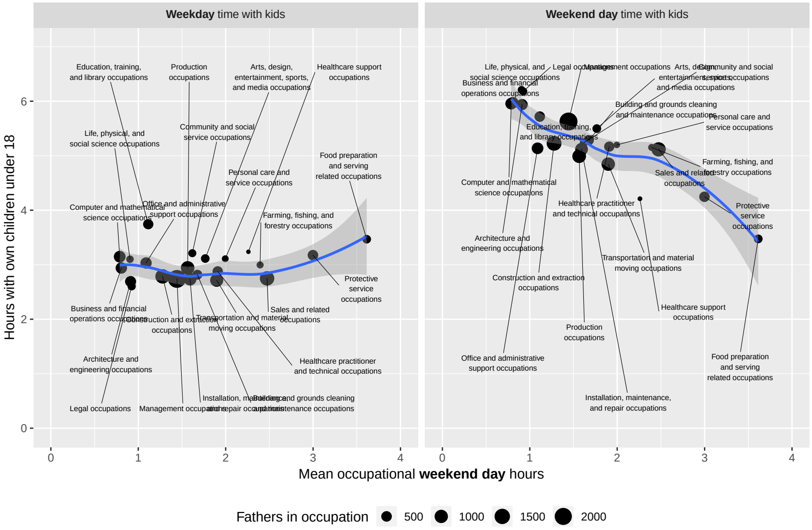

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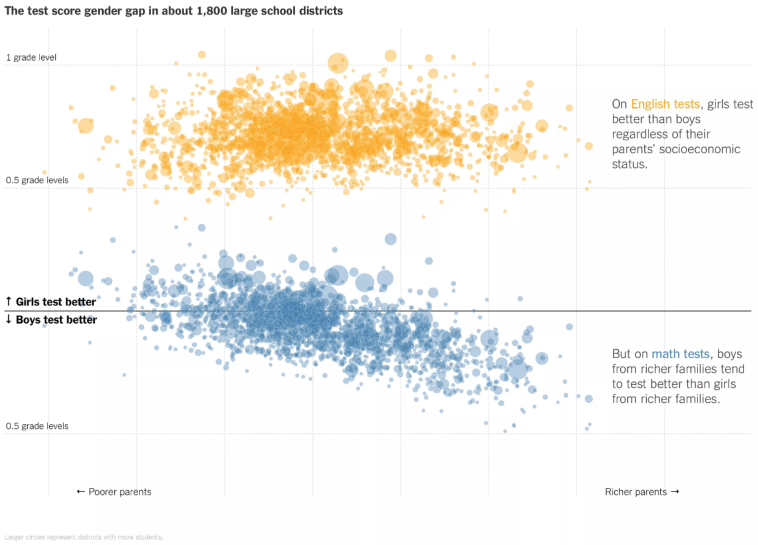

Source: New York Times

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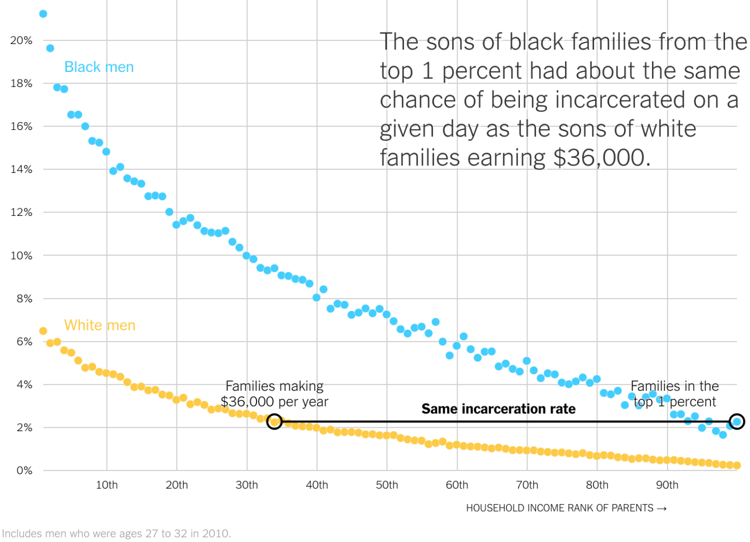

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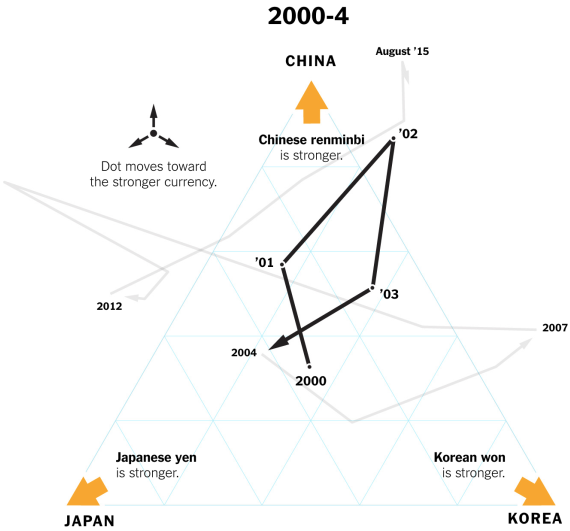

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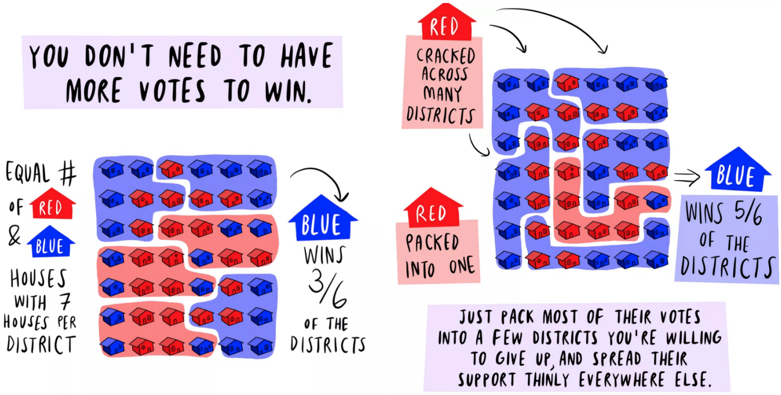

Source: Olivia Walch

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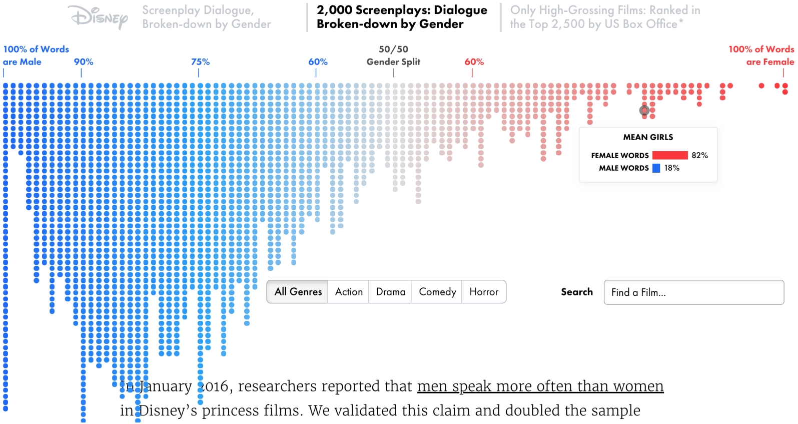


Source: The Pudding

Examples

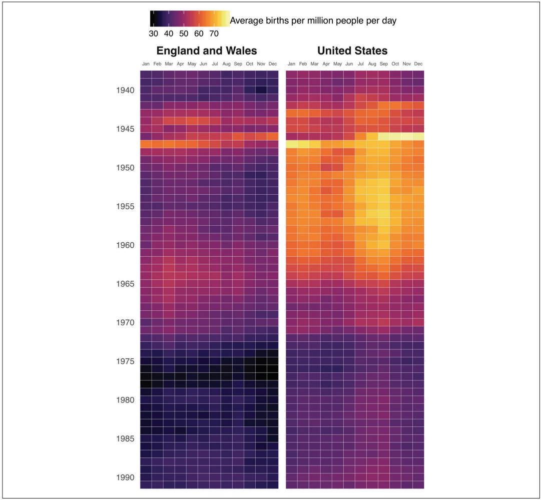
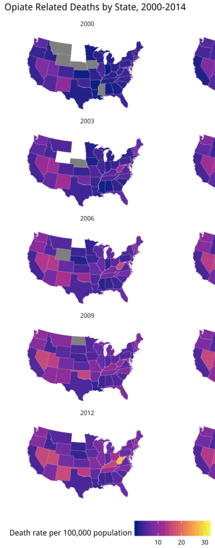


Figure 1. Average births per million people per day, 1938–1991. Each tile represents one month. The underlying count is number of births per month, standardized first by the total population for the period and then by the number of days in that month. Data for the United States are from the U.S. Census Bureau. Data for England and Wales are from the U.K. Office of National Statistics.

Source: Kieran Healy

Examples



Source: Kieran Healy

Reading

- Angrist and Pishke Chapter 2 (The Experimental Ideal) Chapter 3 (Regression and Causality)
- Morgan and Winship Chapters 3-4 (Causal Graphs and Conditioning Estimators)
- Hernan and Robins Chapter 3 Observational Studies
- Optional: Hernan (2018) "The C-word: Scientific euphemisms do not improve causal inference from observational data" *American Journal of Public Health*.
- Optional: Elwert and Winship (2014) "Endogenous selection bias: The problem of conditioning on a collider variable" *Annual Review of Sociology*
- Optional: Morgan and Winship Chapter 11 Repeated Observations and the Estimation of Causal Effects