

Week 9: Regression in the Social Sciences

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Princeton

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¹These 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?

- 1 Making Arguments
- 2 Potential Outcomes
- 3 Average Treatment effects
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- Knowing how methods work also makes you a better reader of work.

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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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- **Law of Decreasing Credibility (Manski)**: The credibility of inference decreases with the strength of the assumptions maintained

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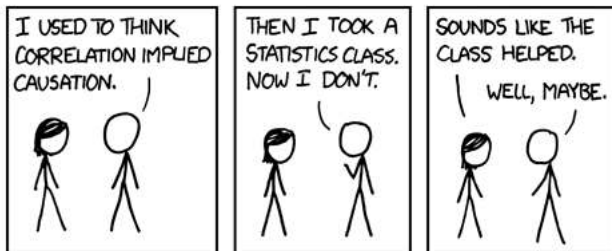
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- Pearl (1995) develops graphical models for causal inference

Causation



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

$$T_i = \begin{cases} 1 & \text{Unit is assigned to treatment} \\ 0 & \text{Unit is not assigned to treatment} \end{cases}$$

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τ_i : The treatment effect

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

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$$T_i = \begin{cases} 1 & \text{Receive Aspirin} \\ 0 & \text{Receive Placebo} \end{cases}$$

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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

Causal Inference is a Missing Data Problem

Definition: Observed Outcome

$$Y_i = T_i * Y_i(1) + (1 - T_i) * Y_i(0)$$

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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: Aspirin's Impact on Headaches

Patient i		Pill T_i	Headache Status $Y_i(\text{🔴})$ $Y_i(\text{🟡})$ 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

Causal Inference is a Missing Data Problem

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Patient i		Pill T_i	Headache Status $Y_i(\text{red pill})$ $Y_i(\text{yellow pill})$ Y_i	Age X_{1i}	Academic X_{2i}
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2			 	55	N
3			 	62	Y
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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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1		1	0	0	0	25	Y
2			0	1		55	N
3			1	1		62	Y
4		0	1	1	1	80	N
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- No interference
 - ▶ We are implicitly stating that the potential outcomes for that unit are unaffected by the treatment status of other units
 - ▶ 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} :

$$\begin{array}{ll} Y_{100} & Y_{000} \\ Y_{110} & Y_{010} \\ Y_{101} & Y_{001} \\ Y_{111} & Y_{011} \end{array}$$

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- Same version of the treatment

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- Potential violations:
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 - ▶ spill-over effects, carry-over effects
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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.

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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.
- 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

The Selection Problem

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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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“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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- 1 Making Arguments
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- 3 Average Treatment effects
- 4 Graphical Models
- 5 Fun With A Bundle of Sticks
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No Causation Without Manipulation

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- It is **fixed** and **unchanging**

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Average Treatment Effect

Imagine a study population with 4 units:

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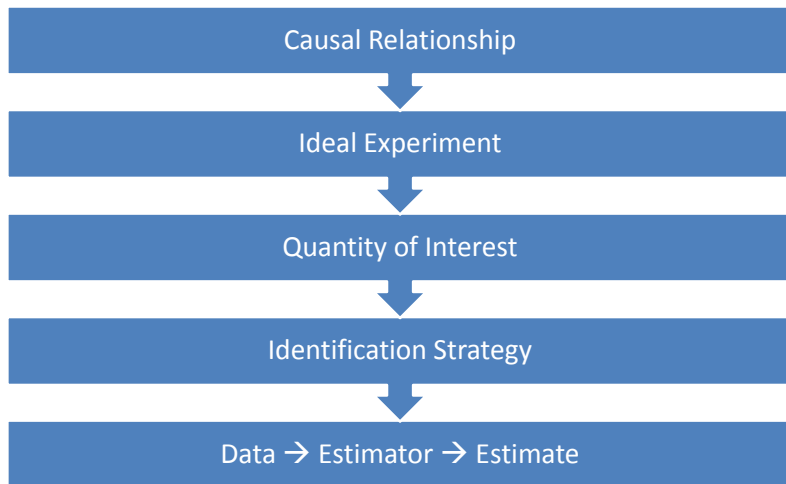
Most statistical models of causal inference attain identification of treatment effects by restricting the assignment mechanism in some way.

No causation without manipulation?

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

Causal Inference Workflow



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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.

Summary: Observational Studies and 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>

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- 2 Potential Outcomes
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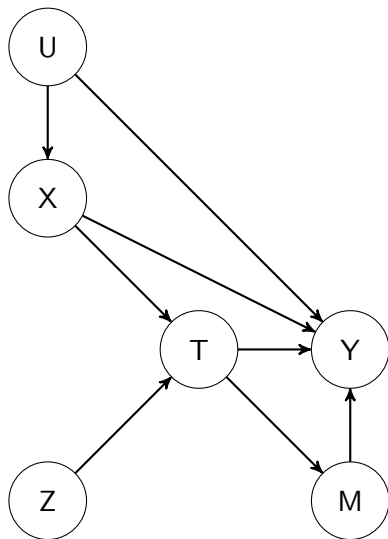
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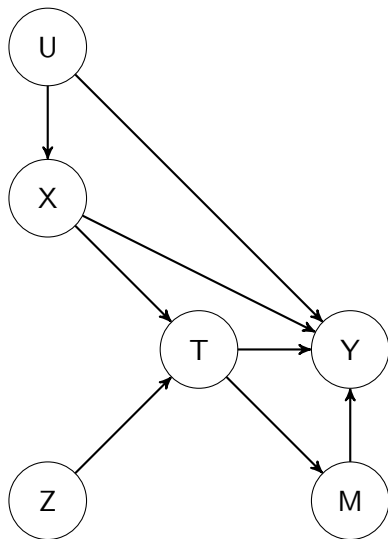
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- Nice software that takes the graph and returns an identification strategy: **DAGitty** at <http://dagitty.net>

Components of a DAG



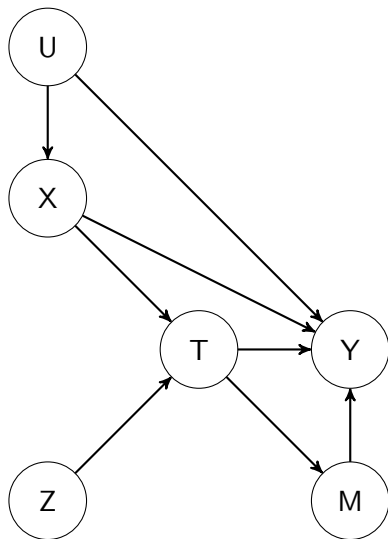
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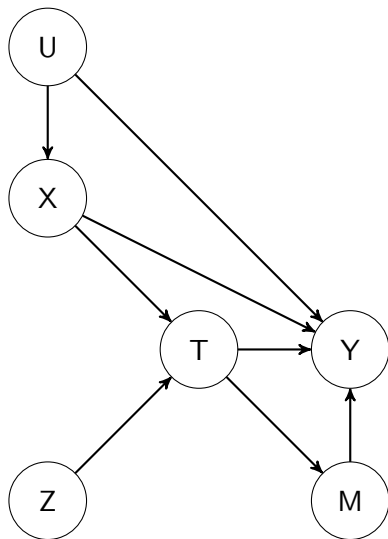
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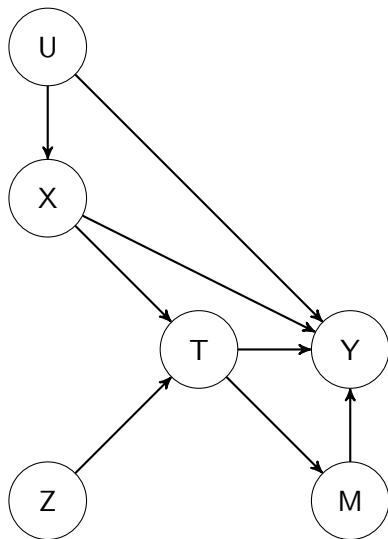
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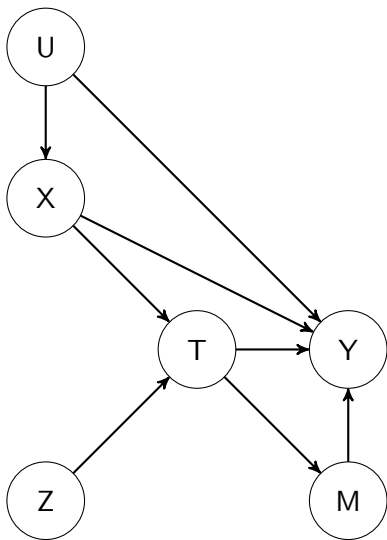
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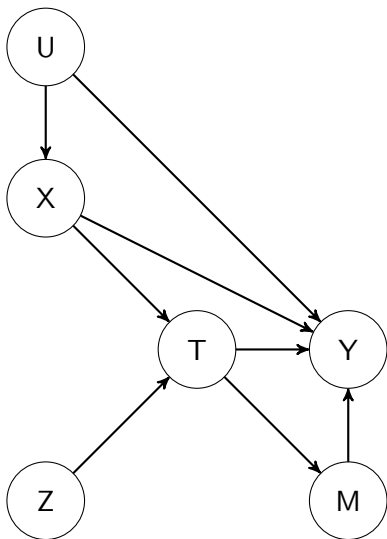
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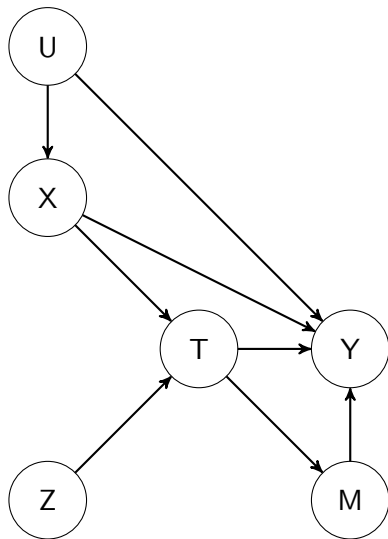
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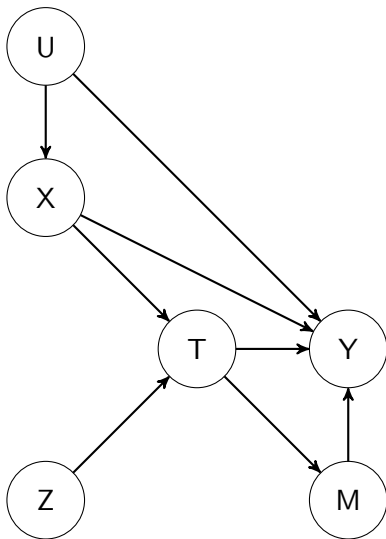
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- all relationships are **non-parametric**

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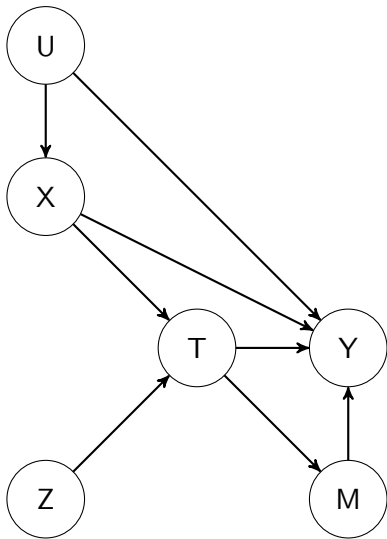
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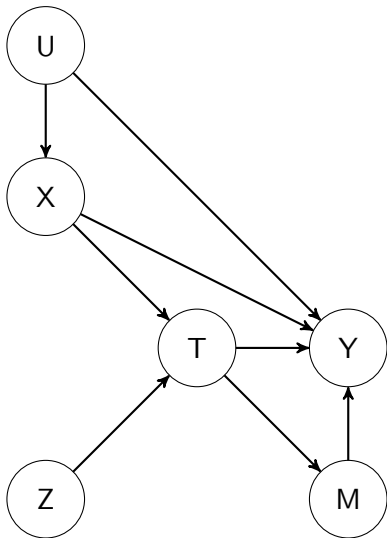
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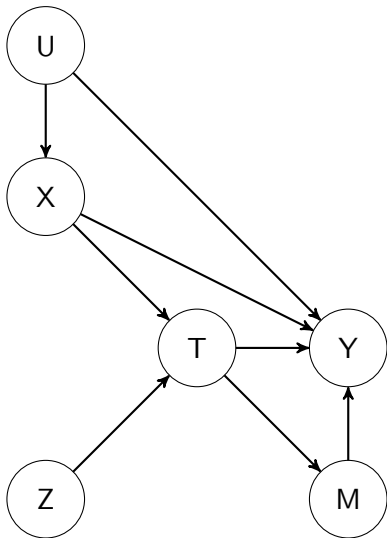
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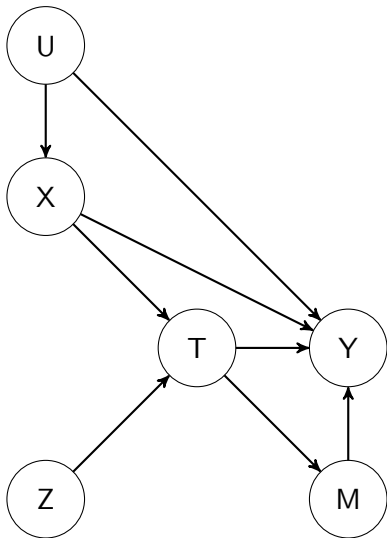
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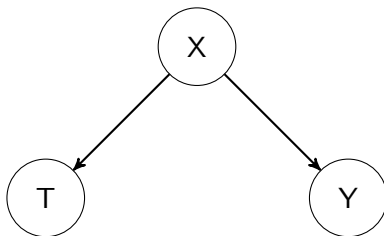
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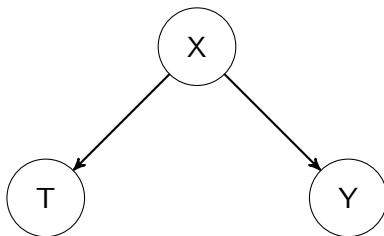
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- We will talk in depth about two types of relationships: **confounders** and **colliders**

Confounders



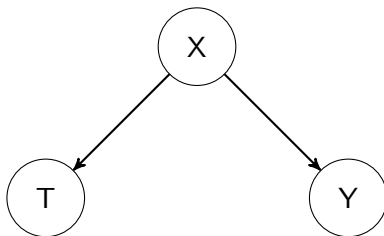
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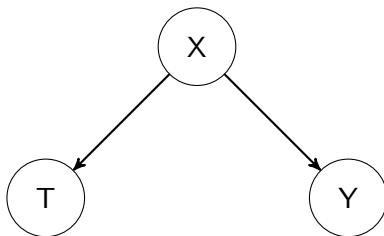
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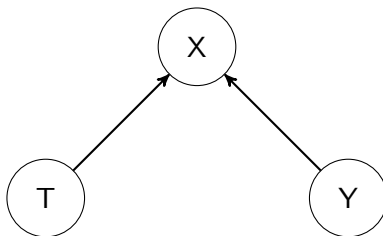
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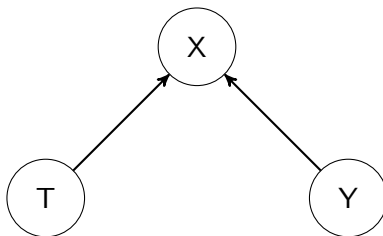
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- We can think of conditioning on a confounder as blocking the flow of association.

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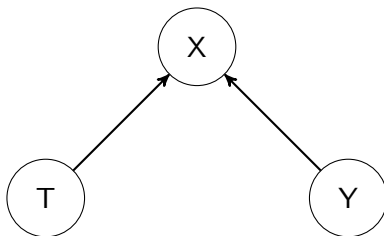
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- In this scenario T and Y are **not marginally associated**
- If we control for X they become associated and create a connection between T and Y

Colliders are scary because you can induce dependence



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

Felix Elwert¹ and Christopher Winship²

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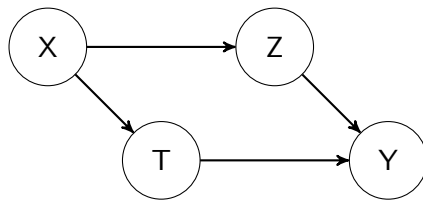
Keywords

causality, directed acyclic graphs, identification, confounding,
selection

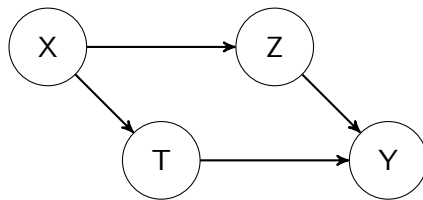
Abstract

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 (Hernan 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.

From Confounders to Back-Door Paths

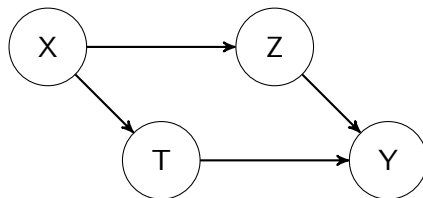


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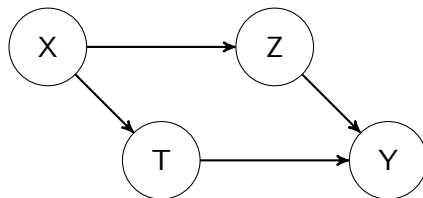
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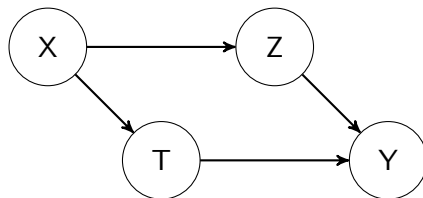
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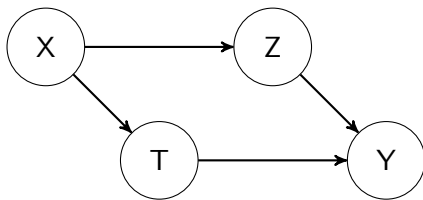
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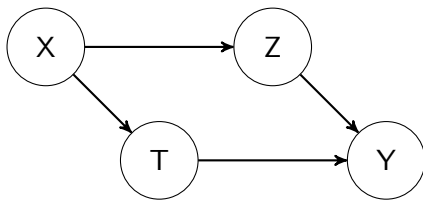
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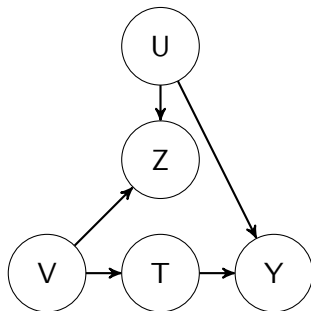
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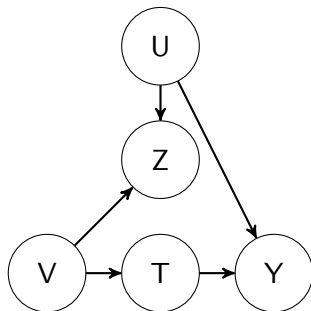
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- We want to **block** the back-door path to leave only the causal effect

Colliders and Back-Door Paths

- Z is a **collider** and it lies along a back-door path from T to Y

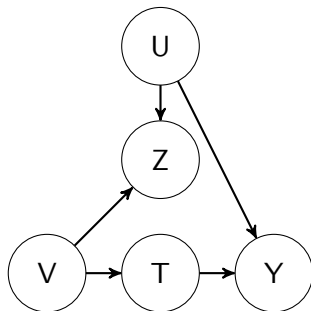


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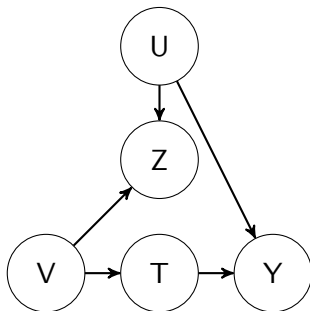
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- So how do we know which back-door paths to block?

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 - 1 p contains a **chain** structure $a \rightarrow c \rightarrow b$ or a **fork** structure $a \leftarrow c \rightarrow b$ where the node c is in the set 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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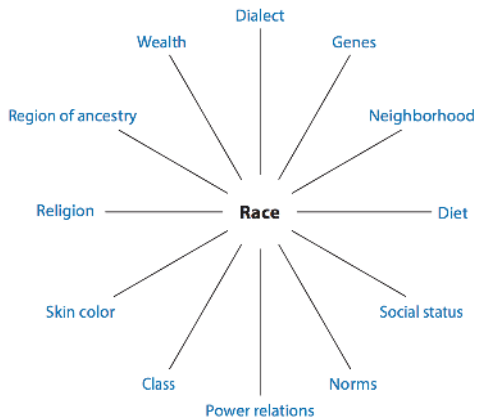
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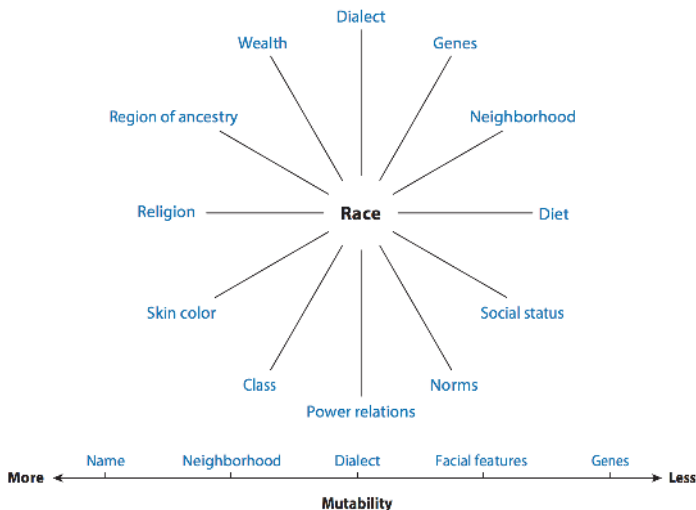
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- ▶ Observational Studies (Greiner and Rubin 2010, Wasow 2012)

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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

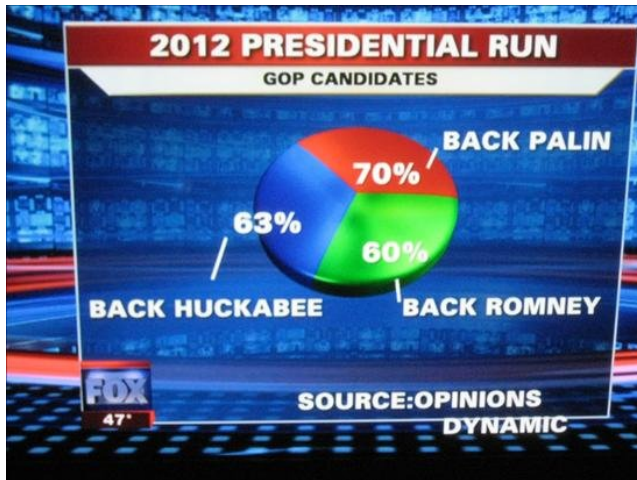
	Exposure	Within-Group
Unit	Individuals or institutions, potentially from any group	Members of a particular group
Typical treatment	Racial cue or signal (e.g., include distinctively ethnic names on a resume)	Constitutive element of the composite of race (e.g., address anxiety about social belonging in 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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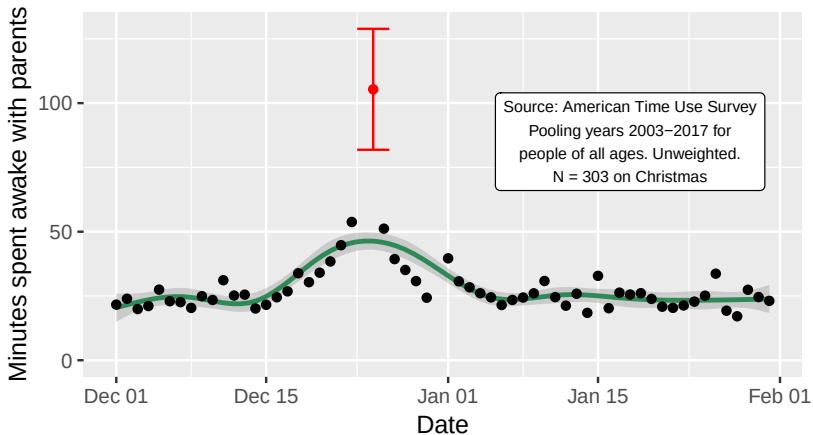
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- I **strongly recommend** Kieran Healy's new visualization book — great summary of the fundamentals plus R code.

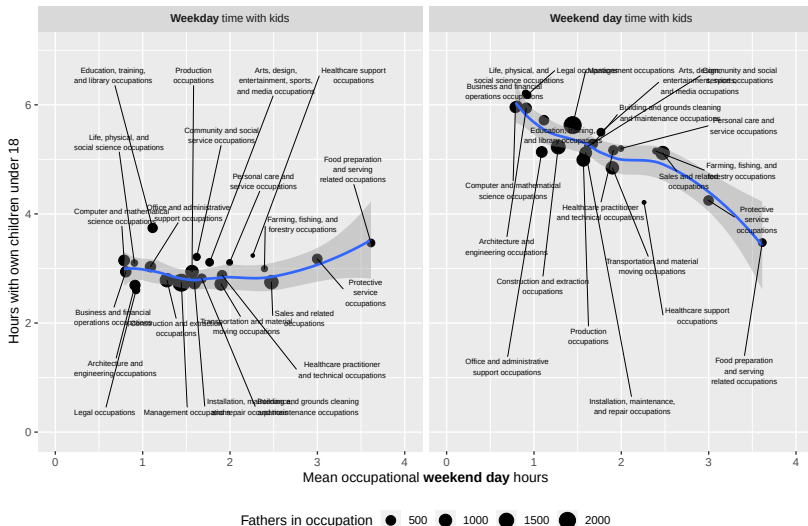
Examples

People spend more time with parents on Christmas



Source: Ian Lundberg

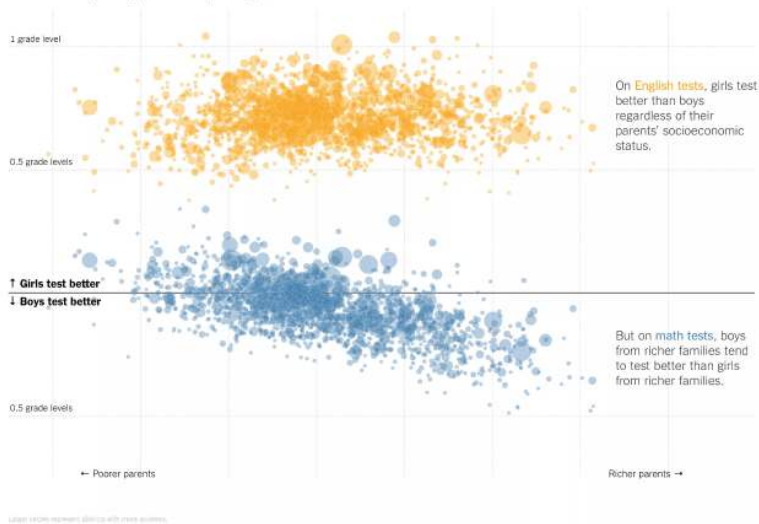
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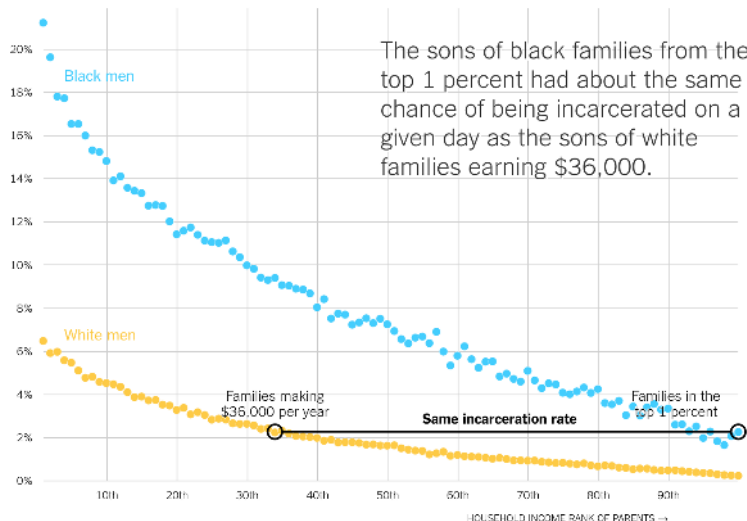
Examples

The test score gender gap in about 1,800 large school districts



Source: New York Times

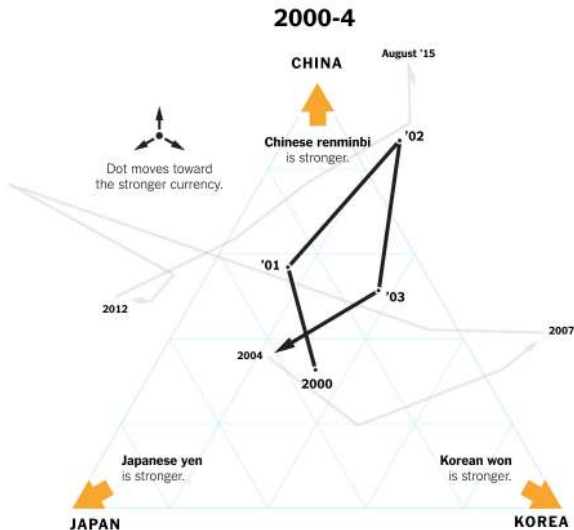
Examples



Includes men who were ages 27 to 32 in 2010.

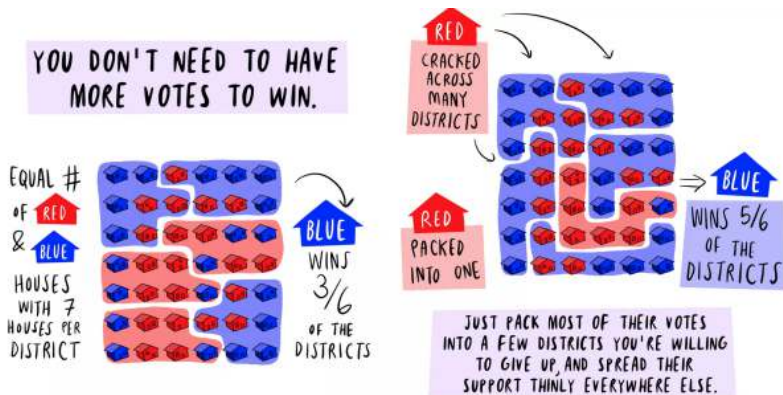
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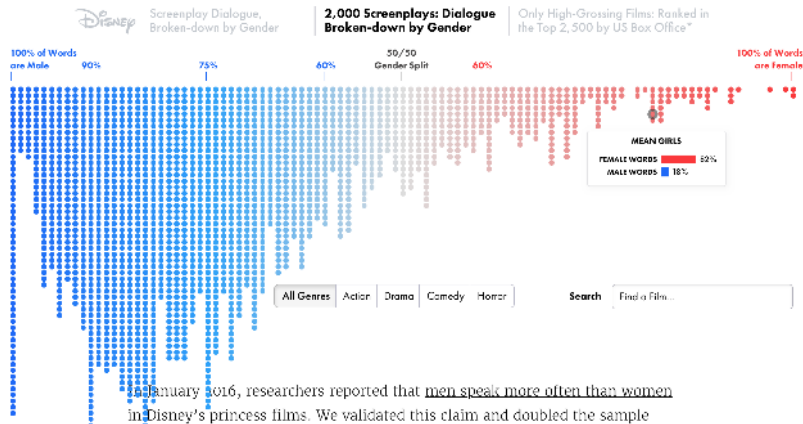
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Examples



Source: Olivia Walch

Examples



Source: The Pudding

Examples

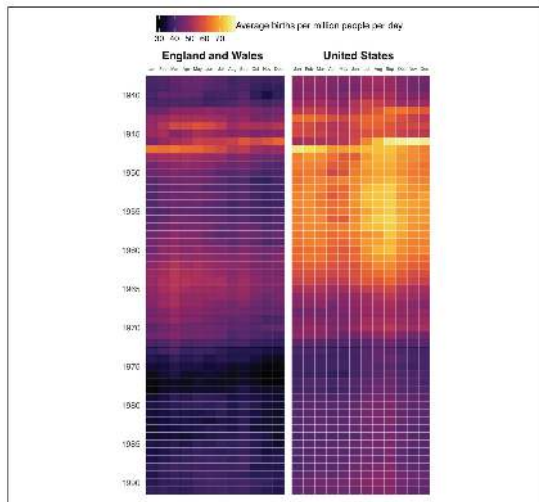
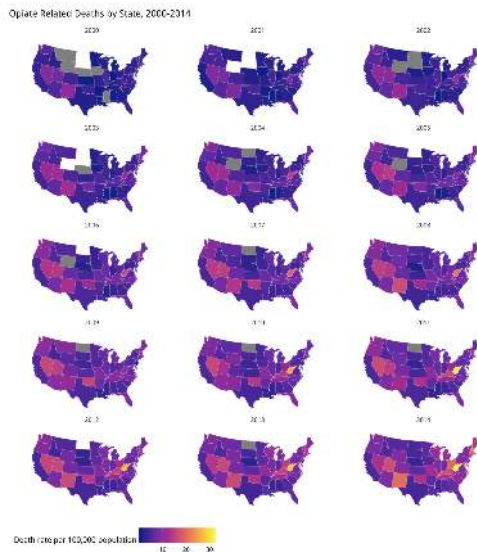


Figure 1. Average births per million people per day, 1930–1991. Each tile represents one month. The underlying count is number of births per month, standardized first by the total population for the period and then by the number of days in that month. Data for the United States are from the U.S. Census Bureau. Data for England and Wales are from the U.K. Office of National Statistics.

Source: Kieran Healy

Examples



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Reading

- Angrist and Pischke 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