GOV 2001/ 1002/ E-200 Section 9
Causal Inference and Estimation[1]

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[1]These section notes are heavily indebted to past Gov 2001 TFs for slides and R code.

LOGISTICS

Reading Assignment- Ho et. al. (2007), King et. al. (2015), and Maertens and Swinnen (2015) **Problem Set 7-** Last Problem Set!

Due by 6pm Wednesday, March 13th on Canvas.

Overview

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  - learn how to define a causal effect and what assumptions are needed to estimate it.

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  - learn how to estimate a causal effect using data.

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  - learn how to define a causal effect and what assumptions are needed to estimate it.
  - learn how to estimate a causal effect using data.
  - learn the benefits and drawbacks of different methods of estimating causal effects.

Outline

Causal Inference

Identifying Causal Effects

Causal Effects in Observational Data

Matching

What is a Causal Effect?

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  - $Y_i(1)$ is the outcome if the person were to receive treatment $T = 1$.
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- The causal effect τ_i of treatment for that individual is

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

The Fundamental Problem of Causal Inference

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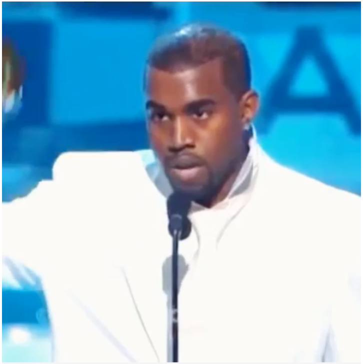

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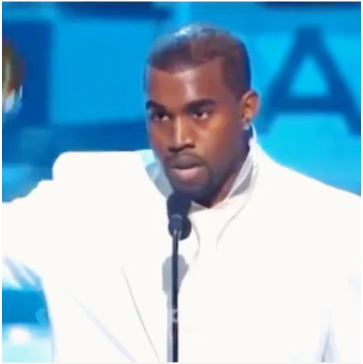

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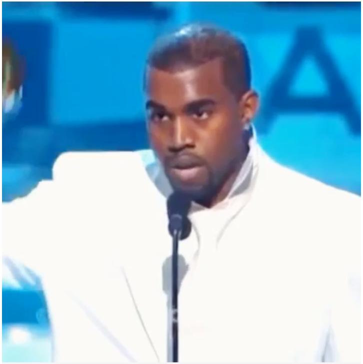

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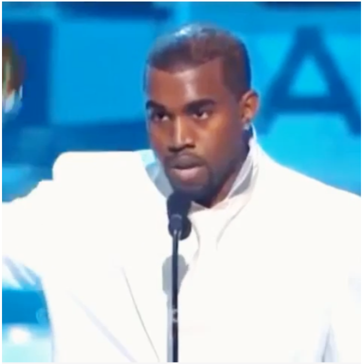

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Kanye West, 2005

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Causal vs. Associational Claims

- What's the difference between

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- and

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  ?
- First is a causal quantity (counterfactual statement). The second is an association (not counterfactual!)

CAUSAL VS. ASSOCIATIONAL CLAIMS

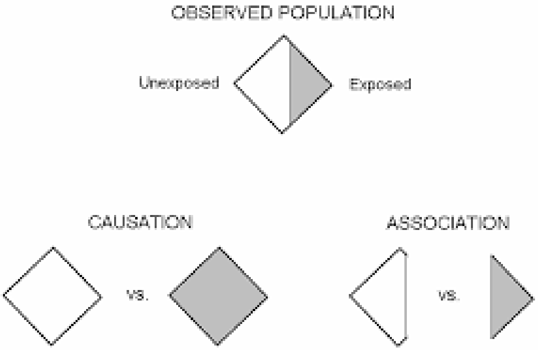

Figure: Difference betweeen causal and association quantities (Hernan and Robins)

Linking Associations to Causal Quantities

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- Your difference-in-means is not a causal effect. Your regression coefficient is not a causal effect. Your post-matching linear model is not a causal effect. Your inverse-probability of treatment weighted marginal structural model estimator is not a causal effect.
- **...without additional assumptions.**
- There is *no such thing* as a causal effect derived purely from data alone.

Linking Associations to Causal Quantities

- We need *assumptions* to connect observed associational quantities (like differences in means) to causal effects.
- Basically: When does correlation actually imply causation?
-
- This is often known as **identification** of a causal effect.

OUTLINE

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IDENTIFYING CAUSAL EFFECTS

- How do we go from something we can estimate

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to something we don't observe

$$E[Y_i(1)] - E[Y_i(0)]$$

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  - Also implies no interference between units - the potential outcome for an individual does not depend on treatments assigned to *other* individuals.
- *Implication:* We can reduce the number of potential outcomes for each unit to two that depend only on the treatment it receives: $Y_i(1)$ and $Y_i(0)$.

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- Randomization is valued because it *gives us* ignorability.

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- What to do?

WHAT IF I DON'T HAVE AN EXPERIMENT!?

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- What to do? Make a weaker assumption - conditional ignorability.

IDENTIFICATION IN OBSERVATIONAL DATA

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- When X is low-dimensional, can *stratify* by X, take differences in means within strata, and then average those estimates to estimate ATE. No additional modeling assumptions necessary!

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- First approach: Make more modeling assumptions.

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- When X is high-dimensional or continuous, what happens to the stratification estimator? Too high variance!
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- First approach: Make more modeling assumptions. Regression!
- Assume not only conditional ignorability, but also a model for $E[Y|T, X]$.

$$E[Y_i|T_i, X_i] = \beta_0 + \beta_1 T_i + \beta_2 X_{1i} + \beta_3 X_{2i}, \dots \dots$$

- Now we can estimate using OLS...

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- If we pick the wrong model, we introduce bias in *estimation*. How do we avoid *model dependence*?

Outline

Causal Inference

Identifying Causal Effects

Causal Effects in Observational Data

Matching

Matching Estimators

- How do we estimate $E[Y_i(1)]$ and $E[Y_i(0)]$ when we have lots of covariates but don't want to assume a model for the outcome?

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- For each observation, we observe either $Y_i(1)$ or $Y_i(0)$.
- If we observed everything, then our estimated ATE $\hat{\tau}$ would be

$$\hat{\tau} = \frac{1}{N}\sum_{i=1}^{N} Y_i(1) - \frac{1}{N}\sum_{i=1}^{N} Y_i(0)$$

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- We could do the same for $Y_i\hat{(0)}$ and use a matching estimator for the ATE

$$\hat{\tau} = \frac{1}{N}\sum_{i=1}^{N} Y_i\hat{(1)} - \frac{1}{N}\sum_{i=1}^{N} Y_i\hat{(0)}$$

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- However, it *does* ameliorate bias from arbitrary choices of models for $E[Y|T, X]$.
- And gives us a way of assessing how "well" we've adjusted for the observed confounders – imbalance.

HOW DO WE PICK MATCHES?

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  - **Coarsened Exact Matching**:

$$D(X_i, X_j) = \begin{cases} 0 & \text{if } X_i \approx X_j \\ \infty & \text{if } X_i \not\approx X_j \end{cases}$$

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TABLE 1. Relationship between Treatment Group Assignment and Covariates (Household-Level Data)

	Control	Civic Duty	Hawthorne	Self	Neighbors
	Mean	Mean	Mean	Mean	Mean
Household size	1.91	1.91	1.91	1.91	1.91
Nov 2002	.83	.84	.84	.84	.84
Nov 2000	.87	.87	.87	.86	.87
Aug 2004	.42	.42	.42	.42	.42
Aug 2002	.41	.41	.41	.41	.41
Aug 2000	.26	.27	.26	.26	.26
Female	.50	.50	.50	.50	.50
Age (in years)	51.98	51.85	51.87	51.91	52.01
N =	99,999	20,001	20,002	20,000	20,000

Note: Only registered voters who voted in November 2004 were selected for our sample. Although not included in the table, there were no significant differences between treatment group assignment and covariates measuring race and ethnicity.

Figure: Balance table from actual experiment by Gerber, Green and Larimer (2008)

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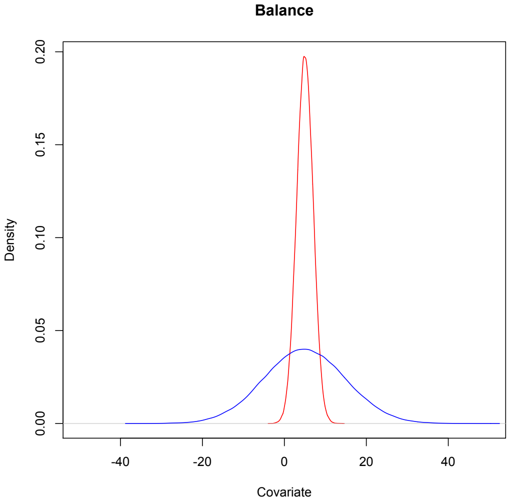
- However, means can be misleading:



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- However, it still relies on untestable modelling assumptions about the propensity score.
- And only approximates complete randomization rather than blocking (and so suffers in efficiency/bias reduction).
- Open research question to find best methods of variable selection in matching without relying on models like propensity score.

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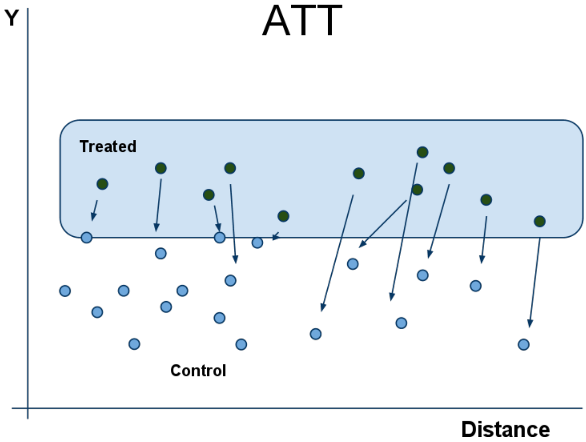

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- Careful - this improves internal validity (reduces bias) but might limit external validity (by changing the population of interest).
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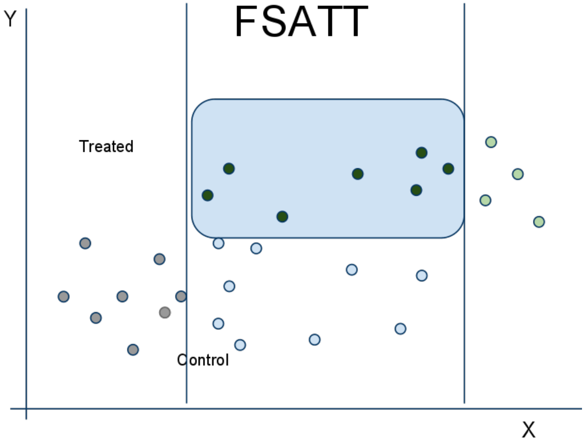

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- But if you have strong unobserved confounding, no amount of matching will save you. Change your research design!

Questions

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