Soc504: Causal Inference Topics

Brandon Stewart¹

Princeton

April 10 - April 19, 2017

Stewart (Princeton)

¹This lecture draws from slides by Matt Blackwell, Jens Hainmueller, Erin Hartman and Gary King

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 - Optional: Acharya, Blackwell and Sen. "Explaining Causal Findings Without Bias: Detecting and Assessing Direct Effects." American Political Science Review. (2016).

Assessing Counterfactuals

- 2 A (Brief) Review of Selection on Observables
- 3 Matching as Non-parametric Preprocessing
- Fundamentals of Matching
- 5 Three Approaches to Matching
 - The Propensity Score
- 7 Mechanisms: Estimands and Identification
- Mechanisms: Estimation
- 9 Controlled Direct Effects
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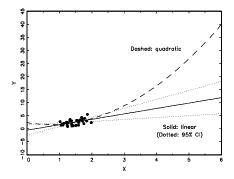
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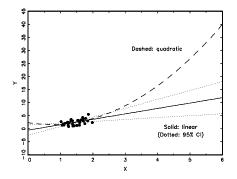
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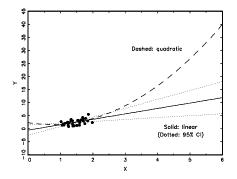
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- Summary of Today: don't ask your model unreasonable questions. (remember the Momentous Sprint?)

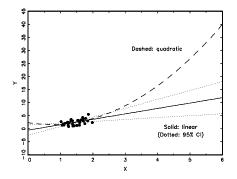




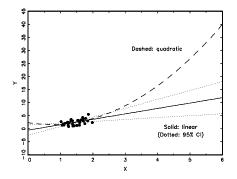
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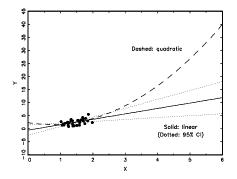
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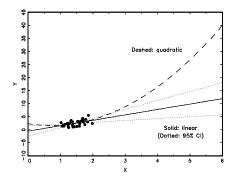
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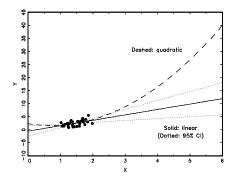
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- How do you choose a model? R²? Some "test"? "Theory"?
- The bottom line: answers to some questions don't exist in the data.
- Our estimate of certain quantities of interest is highly model dependent

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Result

The maximum degree of model dependence: solely a function of the distance from the counterfactual to the data

A (Hypothethical) Research Design

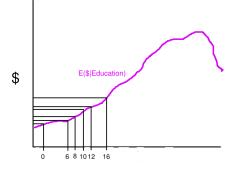
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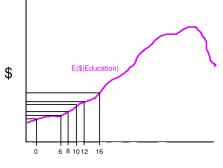
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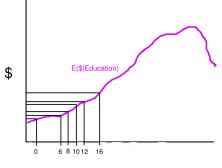
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- We find a coefficient of $\hat{\beta} = \$1,000$, big t-statistics, narrow confidence intervals, and pass every test for auto-correlation, fit, normality, linearity, homoskedasticity, etc.



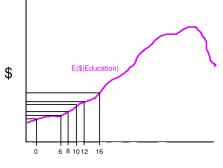


Years of Education

• A Factual Question: How much salary would someone receive with 12 years of education (a high school degree)?

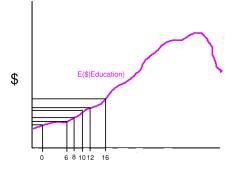


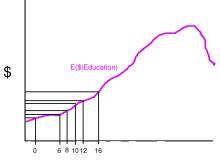
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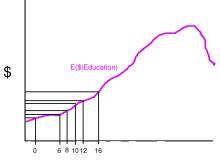
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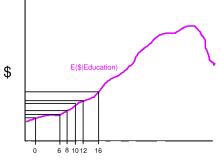


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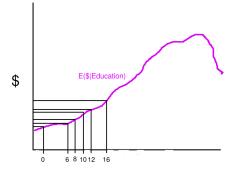
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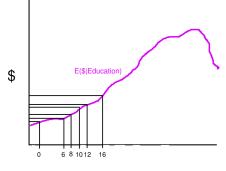


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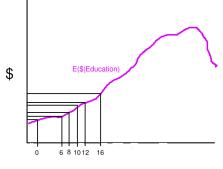
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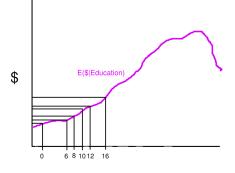
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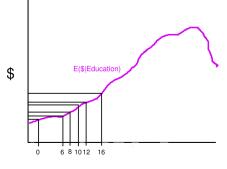
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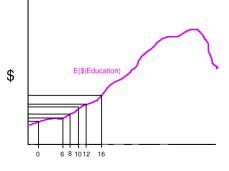
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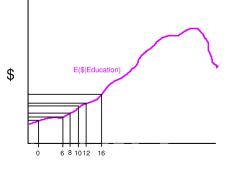
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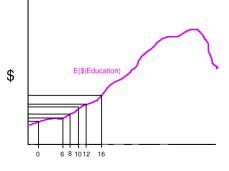


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- Recall: the regression passed every test and met every assumption; identical calculations worked for the other questions.
- What's changed? How would we recognize it when the example is less extreme or multidimensional?

Stewart (Princeton)

Causal Inference

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- (If X were continuous, we would be reducing ∞ to 2, also by assumption)

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- The difference: an enormous assumption based on convenience, not evidence or theory.

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- The curse of dimensionality introduces huge assumptions, often unrecognized.

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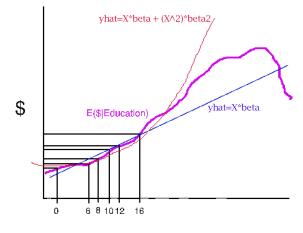
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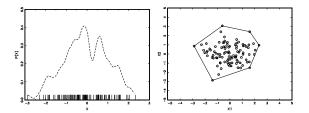
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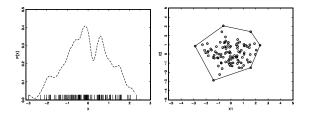
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 - Assume E(Y|X) is (minimally) smooth in X
 - No need to specify models (or a class of models), estimators, or dependent variables.
 - Results of one run apply to the class of all models, all estimators, and all dependent variables.

Interpolation vs Extrapolation in one Dimension

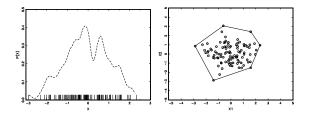


Years of Education

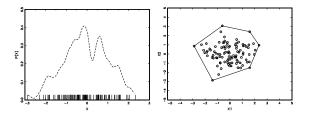




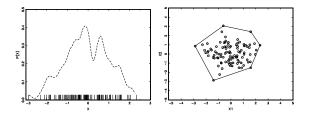
• Interpolation: Inside the convex hull



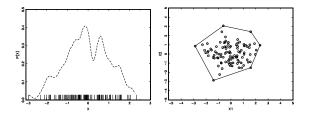
- Interpolation: Inside the convex hull
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- Interpolation: Inside the convex hull
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- Calculating the convex hull would take forever in high-dimensions
- WhatIf package uses linear programming to check if a candidate point is inside the hull
- The key idea is making sure your counterfactual is near the data!

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Replication: Doyle and Sambanis, APSR 2000

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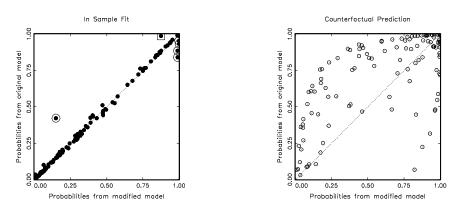
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- \bullet Counterfactuals: UN intervention switched (0/1 to 1/0) for each observation
- Percent of counterfactuals in the convex hull: 0%
- Thus, without estimating any models, we know inferences will be model dependent; for illustration, here is an example....

Doyle and Sambanis, Logit Model

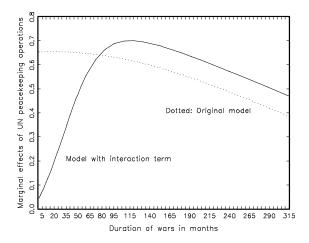
Ori	iginal Mode	el	Modified Model			
Coeff	SE	P-val	Coeff	SE	P-val	
-1.742	.609	.004	-1.666	.606	.006	
445	.126	.000	437	.125	.000	
.006	.006	.258	.006	.006	.342	
-1.259	.703	.073	-1.045	.899	.245	
.062	.065	.346	.032	.104	.756	
.004	.002	.010	.004	.002	.017	
.001	.000	.065	.001	.000	.068	
-6.016	3.071	.050	-6.215	3.065	.043	
299	.169	.077	-0.284	.169	.093	
2.124	.821	.010	2.126	.802	.008	
3.135	1.091	.004	.262	1.392	.851	
—	—	_	.037	.011	.001	
8.609	2.157	0.000	7.978	2.350	.000	
	122			122		
-45.649			-44.902			
	.423			.433		
	Coeff -1.742 445 .006 -1.259 .062 .004 .001 -6.016 299 2.124 3.135 	Coeff SE -1.742 .609 445 .126 .006 .006 -1.259 .703 .062 .065 .004 .002 .001 .000 -6.016 3.071 299 .169 2.124 .821 3.135 1.091 - 8.609 2.157 122 -45.649	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	$\begin{array}{c c c c c c c c c c c c c c c c c c c $	$\begin{array}{c c c c c c c c c c c c c c c c c c c $	

Mardle and Mardal

Doyle and Sambanis: Model Dependence



UN Peacekeeping Operations



Another Example

Remember our negative binomial model?

Coefficients:

	Estimate	Std. Error	z value	Pr(> z)	
(Intercept)	0.5943	0.1718	3.459	0.000541	***
cathunemp	7.9323	0.9150	8.669	< 2e-16	***
protunemp	-19.1683	2.3713	-8.084	6.29e-16	***

Proposed Counterfactuals

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Let's consider two first differences we might plausibly estimate.

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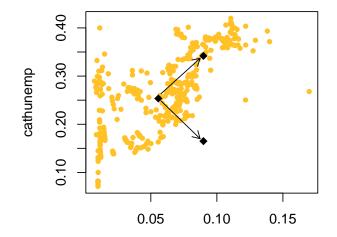
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- 1. Counterfactual 1: Catholic unemployment increases by one standard deviation and Protestant unemployment increases by one standard deviation.
- 2. Counterfactual 2: Catholic unemployment decreases by one standard deviation and Protestant unemployment increases by one standard deviation.

Proposed Counterfactuals Plotted



Causal Inference

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Checking the Convex Hull

```
cf.res1 <- whatif(data = mod, cfact = cf1)
> cf.res1$in.hull
[1] TRUE
```

```
cf.res2 <- whatif(data = mod, cfact = cf2)
cf.res2$in.hull
[1] FALSE</pre>
```

A Measure of Distance

Stewart (Princeton)

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The whatif function also tells us the percentage of data points within 1 geometric variance of the counterfactual.

- > cf.res1\$sum.stat
 1
- 0.2608696

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> cf.res2\$sum.stat

1

0.04603581

The geometric variance is a generalization of the usual variance which is more suitable to discrete and continuous variables- essentially it is the average pairwise Gower distance in the data. The number of GV's away can be altered with the nearby argument.

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- Thus we may get wildly different counterfactuals from different models when we are far from the data, we call this model dependence
- The convex hull provides a way to check for extrapolation
- This is a great way of assessing the reasonableness of our simulated quantities of interest

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- When we have the right set of observed confounders, matching is a strategy that helps reduce model dependence in this conditioning
- Matching itself is not an identification strategy, nor is it fundamentally different than alternatives like weighting or regression adjustment

Repeat After Me

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So what is?

Identification Assumption

•
$$(Y_1, Y_0) \perp D \mid X$$
 (selection on observables)

2 $0 < \Pr(D = 1|X) < 1$ with probability one (common support)

Identification Result

Given selection on observables we have

$$\begin{split} \mathrm{E}[Y_1 - Y_0 | X] &= \mathrm{E}[Y_1 - Y_0 | X, D = 1] \\ &= \mathrm{E}[Y | X, D = 1] - \mathrm{E}[Y | X, D = 0] \end{split}$$

Therefore, under the common support condition:

$$\tau_{ATE} = \mathbf{E}[Y_1 - Y_0] = \int \mathbf{E}[Y_1 - Y_0 | X] \, dP(X)$$
$$= \int \left(\mathbf{E}[Y | X, D = 1] - \mathbf{E}[Y | X, D = 0] \right) \, dP(X)$$

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Identification Result

Similarly,

$$\tau_{ATT} = \mathbf{E}[Y_1 - Y_0 | D = 1]$$

= $\int (\mathbf{E}[Y|X, D = 1] - \mathbf{E}[Y|X, D = 0]) dP(X|D = 1)$

To identify τ_{ATT} the selection on observables and common support conditions can be relaxed to:

- $Y_0 \perp D \mid X$ (SOO for Controls)
- $\Pr(D = 1|X) < 1$ (Weak Overlap)

unit	Potential Outcome	Potential Outcome		
1	Y_{1i}	Y_{0i}	D_i	X_i
1	$\mathbf{E}[Y_1 X=0,D=1]$	$E[Y_0 X = 0, D = 1]$	1	0
2	$E[I_1 X = 0, D = 1]$	$\mathbb{E}[I_0 X=0,D=1]$	1	0
3	$\mathbf{E}[Y_1 X=0,D=0]$	$\mathbf{E}[Y_0 X=0,D=0]$	0	0
4			0	0
5	$\mathbf{E}[Y_1 X=1,D=1]$	$\mathbf{E}[Y_0 X=1,D=1]$	1	1
6			1	1
7	$\mathbf{E}[Y_1 X=1, D=0]$	$E[Y_0 X = 1, D = 0]$	0	1
8	$\mathbb{E}[I_1 X = 1, D = 0]$	$\mathbb{E}[I_0 X = 1, D = 0]$	0	1

unit	Potential Outcome under Treatment	Potential Outcome under Control		
i	Y_{1i}	Y_{0i}	Di	X_i
	, 1,	01	D_1	λ_{l}
1	$\mathbf{E}[Y_1 X=0,D=1]$	$E[Y_0 X=0, D=1]=$	1	0
2		$\mathbf{E}[Y_0 X=0,D=0]$	1	0
3	$\mathbf{E}[Y_1 X=0,D=0]$	$\mathbf{E}[Y_0 X=0,D=0]$	0	0
4			0	0
5	$E[Y_1 X = 1, D = 1]$	$E[Y_0 X = 1, D = 1] =$	1	1
6	$\mathbf{E}[T_1 X=1,D=1]$	$\mathbf{E}[Y_0 X=1,D=0]$	1	1
7	$\mathbf{E}[Y_1 X=1, D=0]$	$\mathbf{E}[Y_0 X=1, D=0]$	0	1
8	$\mathbb{E}[I_1 X = 1, D = 0]$	$\mathbb{E}[r_0 X=1,D=0]$	0	1

 $(Y_1, Y_0) \perp D \mid X$ implies that we conditioned on all confounders. The treatment is randomly assigned within each stratum of X:

$$\begin{split} \mathbf{E}[Y_0|X=0,D=1] &= \mathbf{E}[Y_0|X=0,D=0] \text{ and} \\ \mathbf{E}[Y_0|X=1,D=1] &= \mathbf{E}[Y_0|X=1,D=0] \end{split}$$

	Potential Outcome	Potential Outcome		
unit	under Treatment	under Control		
i	Y_{1i}	Y _{0i}	Di	Xi
1	$E[Y_1 X = 0, D = 1]$	$E[Y_0 X = 0, D = 1] =$	1	0
2	$\mathbf{E}[r_1 \mathbf{\lambda}=0, D=1]$	$\mathbf{E}[Y_0 X=0,D=0]$	1	0
3	$E[Y_1 X = 0, D = 0] =$	$\mathbf{E}[Y_0 X=0,D=0]$	0	0
4	$\mathbf{E}[Y_1 X=0, D=1]$	$\mathbf{E}[I_0 X=0,D=0]$	0	0
5	$\mathbf{E}[\mathbf{V} \mid \mathbf{V} = 1 \mathbf{D} = 1]$	$E[Y_0 X = 1, D = 1] =$	1	1
6	$\mathbf{E}[Y_1 X=1,D=1]$	$\mathbf{E}[Y_0 X=1,D=0]$	1	1
7	$E[Y_1 X = 1, D = 0] =$	$E[Y_0 X = 1, D = 0]$	0	1
8	$\mathbf{E}[Y_1 X=1,D=1]$	$\mathbb{E}[I_0 X = 1, D = 0]$	0	1

 $(Y_1, Y_0) \perp D \mid X$ also implies

$$\begin{split} \mathbf{E}[Y_1|X=0,D=1] &= & \mathbf{E}[Y_1|X=0,D=0] \text{ and} \\ \mathbf{E}[Y_1|X=1,D=1] &= & \mathbf{E}[Y_1|X=1,D=0] \end{split}$$

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 - **2** Reduces dependence of estimates on parametric models.

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 - If treated and control groups are better balanced than when you started, due to pruning, model dependence is reduced

Matching as Preprocessing

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• Y_i dep var, T_i (1=treated, 0=control), X_i confounders

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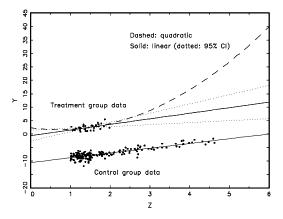
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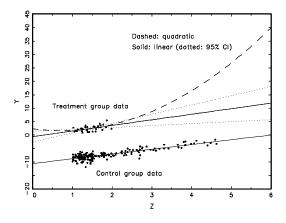
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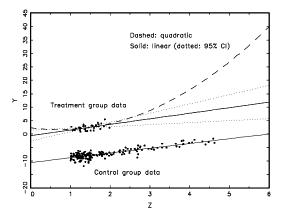


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What to do?

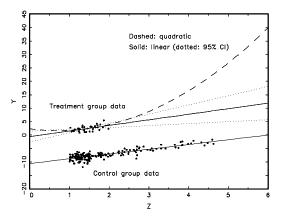
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What to do?

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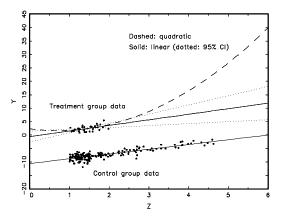
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What to do?

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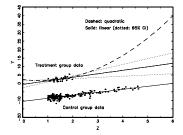


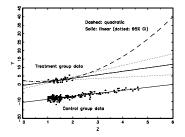
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- Preprocess I: Eliminate extrapolation region
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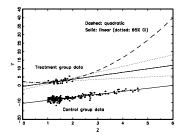
Stewart (Princeton)

Causal Inference

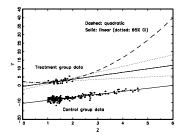




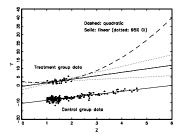
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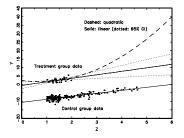
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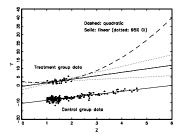
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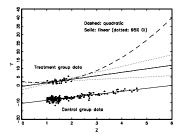
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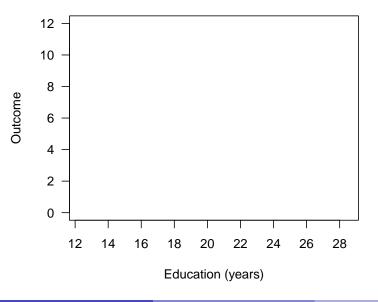


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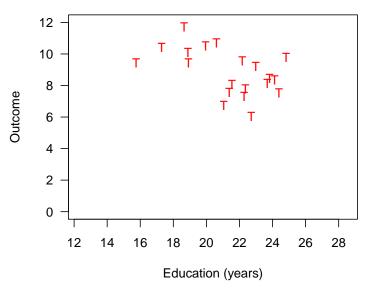
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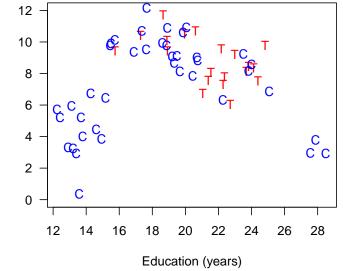
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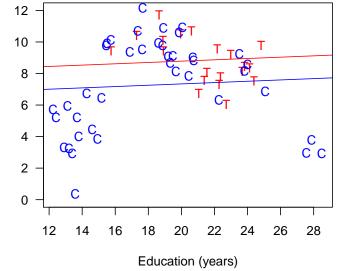


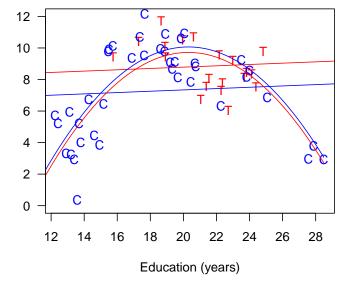
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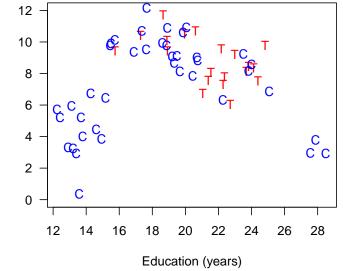


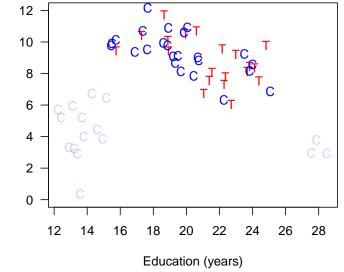
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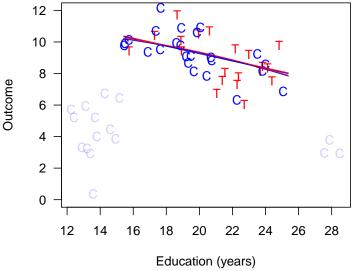












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- 18 control variables (clinical factors, firm characteristics, media variables, etc.)

• Focus on the causal effect of a Democratic majority in the Senate (identified by Carpenter as not robust).

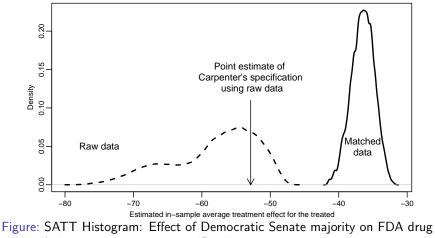
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- (Normal applications would only use one or a few specifications.)

Reducing Model Dependence



approval time, across 262, 143 specifications.

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 $\mathsf{Imbalance} \rightsquigarrow \mathsf{Model} \ \mathsf{Dependence}$

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- It does not help you if there are unobservable common causes of treatment and outcomes
- We can (and should!) verify that we have improved balance on the observed covariates, but this does not imply we have improved balanced on unobserved covariates.

Assessing Counterfactuals

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- 3 Matching as Non-parametric Preprocessing
- Fundamentals of Matching
- 5 Three Approaches to Matching
 - The Propensity Score
- 7 Mechanisms: Estimands and Identification
- Mechanisms: Estimation
- 9 Controlled Direct Effects
- O Appendix: The Case Against Propensity Score Matching

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- The distribution of X_i will be exactly the same for treated and matched control:

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• Balance is checkable \rightsquigarrow are D_i and X_i related in the matched data?

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• Under no unmeasured confounding, $\widehat{Y}_i(0)$ is a good predictor of the true potential outcome under control, Y_i .

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- If *M* varies by treated unit, need to weight observations to ensure balance.

With or without replacement

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 - Choice of distance metric will lead to different matches.

Exact distance metric

• Exact: only match units to other units that have the same exact values of X_i.

$$D_{ij} = \begin{cases} 0 & \text{if } X_i = X_j \\ \infty & \text{if } X_i \neq X_j \end{cases}$$

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• Here, $\hat{\sigma}_k^2$ is the variance of the *k*th variable:

$$\widehat{\sigma}_k^2 = rac{1}{N-1}\sum_{i=1}^N (X_{ik} - \bar{X}_k)$$

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• $\hat{\Sigma}$ is the estimated variance-covariance matrix of the observations:

$$\widehat{\Sigma} = rac{1}{N}\sum_{i=1}^N (X_i - ar{X})(X_i - ar{X})^{\mathcal{T}}$$

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Estimated:

$$\widehat{\tau} = \widehat{\tau}_{ATT} \left(\frac{N_t}{N} \right) + \widehat{\tau}_{ATC} \left(\frac{N_c}{N} \right)$$

Moving the goalposts

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- Note: in nearest neighbor without replacement the order matters!

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• All matching methods seek to maximize balance:

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- 3 Matching as Non-parametric Preprocessing
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- 5 Three Approaches to Matching
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- Which is the best method? The one that produces the best balance!

(Approximates Fully Blocked Experiment)

Preprocess (Matching)

- Checking Measure imbalance, tweak, repeat, ...
- Sestimation Difference in means or a model

(Approximates Fully Blocked Experiment)

Preprocess (Matching)

• Distance
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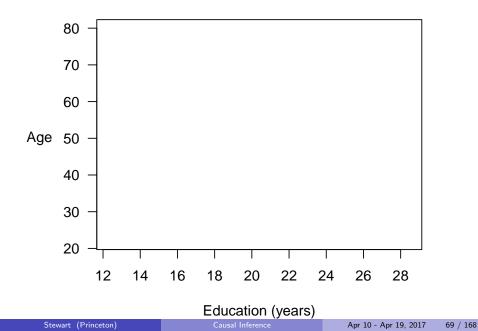
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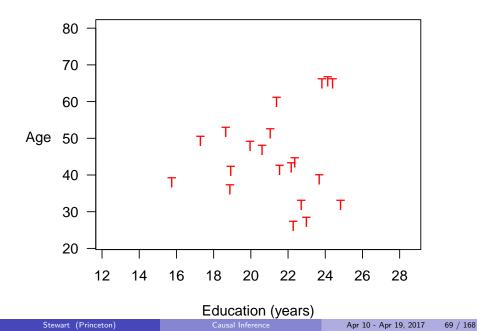
Method 1: Mahalanobis Distance Matching

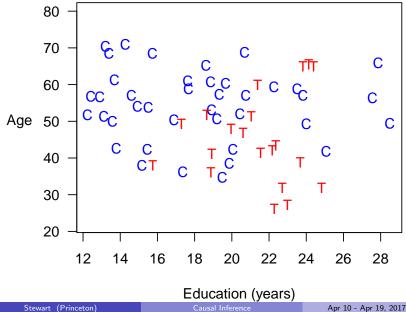
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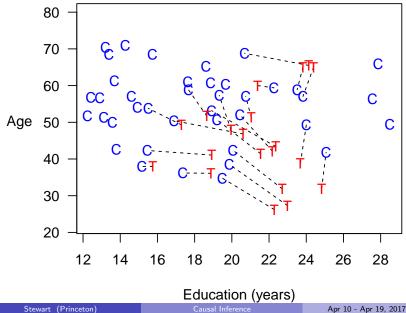
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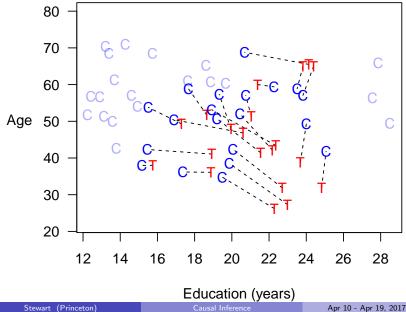


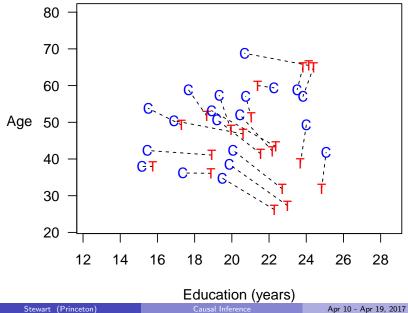


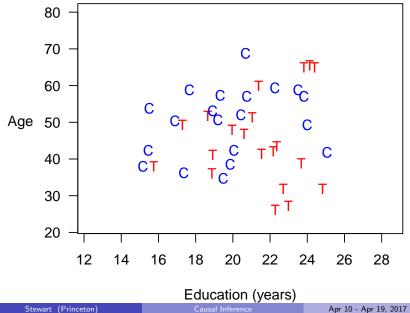












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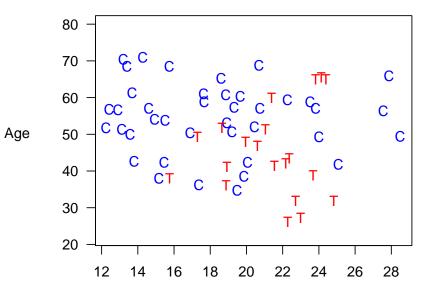
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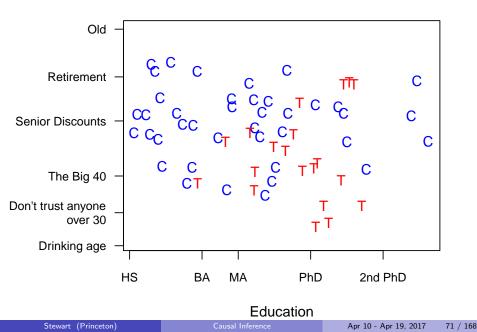
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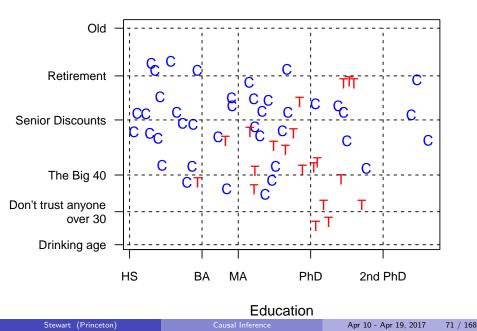
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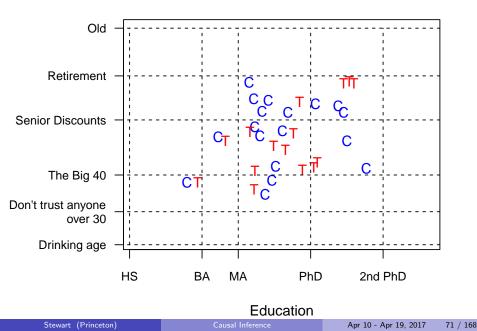
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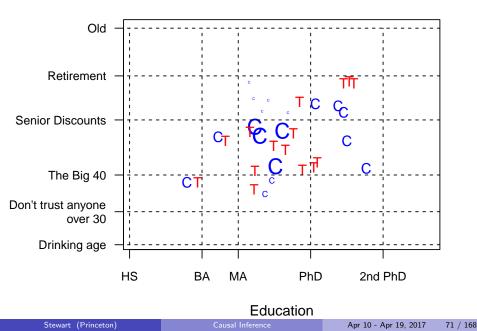


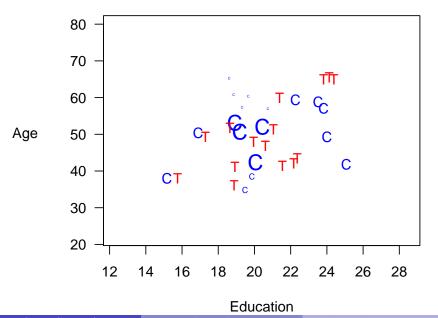
Education











(Approximates Completely Randomized Experiment)

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- Preprocess (Matching)
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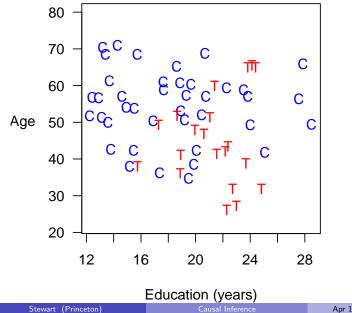
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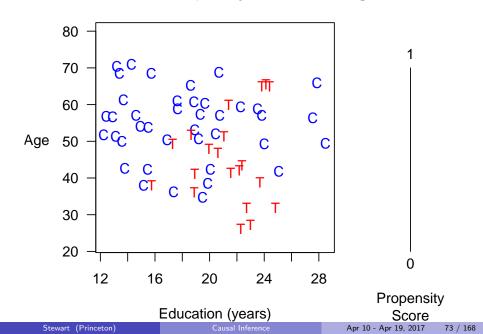
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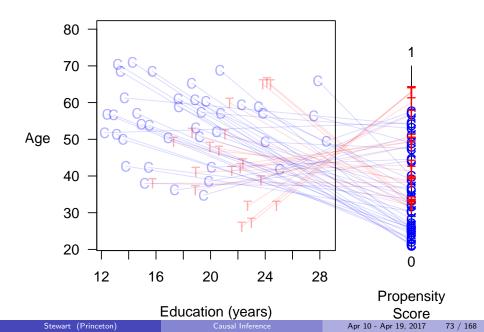
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 - Reduce k elements of X to scalar $\pi_i \equiv \Pr(T_i = 1|X) = \frac{1}{1+e^{-X_i\beta}}$
 - Distance $(X_i, X_j) = |\pi_i \pi_j|$
 - Match each treated unit to the nearest control unit
 - Control units: not reused; pruned if unused
 - Prune matches if Distance>caliper
- Obecking Measure imbalance, tweak, repeat, ...
- Setimation Difference in means or a model

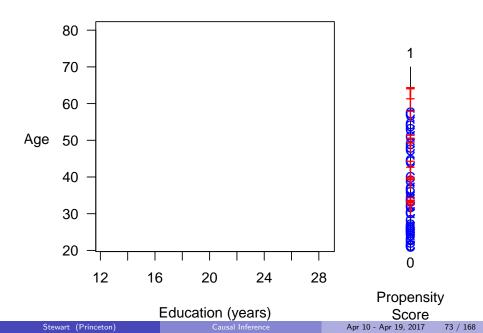


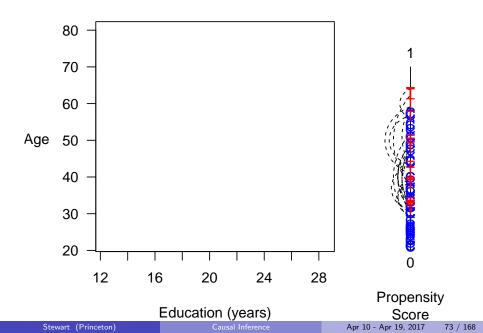
Propensity Score Matching

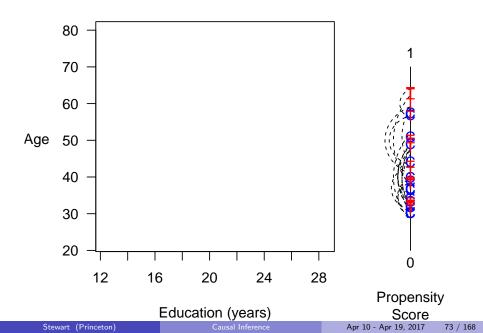


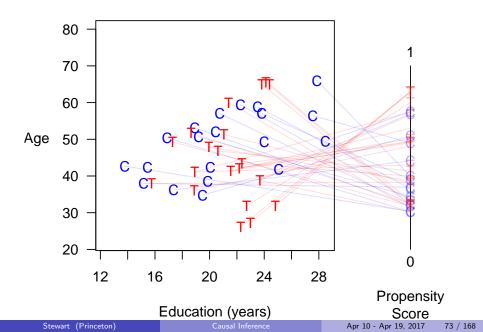


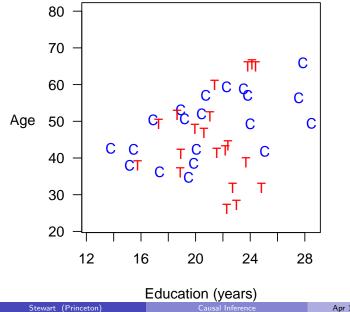












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- Matching discrepancy will grow with the dimension of X_i

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unit-level bias

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• If X_i has a big effect on the mean of $Y_i(0)$ then this bias could be big!

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 Specify a parametric model for μ_c(x) = α_c + x'β_c and estimate β_c from the control data:

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- Create bias-corrected/adjusted imputations for $Y_i(0)$:

$$\widehat{Y}_i(0) = Y_{j(i)} + (X_i - X_{j(i)})'\widehat{\beta}_c$$

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• Simply take the variance of the within-match differences:

$$\widehat{\operatorname{Var}}[\widehat{\tau}_m] = \frac{1}{N_t} \sum_{i=1}^{N_t} \left(Y_i - \widehat{Y}_i(0) - \widehat{\tau}_{m,bc} \right)^2$$

• What if we simply run our original analysis model on the pooled, matching data:

$$\tilde{Y}_i = \alpha_p + \tau_p \cdot \tilde{D}_i + \tilde{X}'_i \beta_p + \nu_i$$

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- Still corrects for some of the residual bias left over from the matching.
- SEs from these models might make additional assumptions (homoskedasticity, etc).

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- Even if you aren't running an experiment, thinking through the ideal experiment will help you.
- and remember: Matching is not an identification strategy

Assessing Counterfactuals

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- 3 Matching as Non-parametric Preprocessing
- Fundamentals of Matching
- 5 Three Approaches to Matching
 - The Propensity Score
- 7 Mechanisms: Estimands and Identification
- Mechanisms: Estimation
- 9 Controlled Direct Effects
- O Appendix: The Case Against Propensity Score Matching

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- A useful way to think about this problem is what causes selection into treatment.
- Propensity score methods emphasize this interpretation by focusing on estimating the probability that a unit will take the treatment.

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$$D_i \bot\!\!\!\!\perp (Y_i(0), Y_i(1)) \mid X_i \implies D_i \bot\!\!\!\perp (Y_i(0), Y_i(1)) \mid e(X_i)$$

► \sim stratifying on e_i is the same in expectation as stratifying on the full X_i .

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- Conditional on the propensity score, treatment is independent of the covariates.
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 - $f(X_i|D_i = 1, e(X_i)) = f(X_i|D_i = 0, e(X_i))$
- However, we have to know the true PS to have all these results work!

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 - Covariate Balancing Propensity Scores (Imai and Ratkovic)

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- It also shows up in a number of more advanced methods for heterogeneous treatment effects, causal inference in longitudinal data etc.
- We will focus on settings where the propensity score is a tool to achieve balance.
- However, note that the propensity score only achieves balance in expectation

Identification with Propensity Scores

Definition

Propensity score is defined as the selection probability conditional on the confounding variables: $\pi(X) = \Pr(D = 1|X)$

Identification Assumption

•
$$(Y_1, Y_0) \perp D \mid X$$
 (selection on observables)

2 $0 < \Pr(D = 1|X) < 1$ with probability one (common support)

Identification Result

Under selection on observables we have $(Y_1, Y_0) \perp D \mid \pi(X)$, ie. conditioning on the propensity score is enough to have independence between the treatment indicator and potential outcomes. Implies substantial dimension reduction.

Identification with Propensity Scores

Proof.

Show that $Pr(D = 1|Y_0, Y_1, \pi(X)) = Pr(D = 1|\pi(X)) = \pi(X)$, implying independence of (Y_0, Y_1) and D conditional on $\pi(X)$.

 $\Pr(D = 1 | Y_1, Y_0, \pi(X)) = \mathbb{E}[D | Y_1, Y_0, \pi(X)]$

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$$Pr(D = 1|Y_1, Y_0, \pi(X)) = E[D|Y_1, Y_0, \pi(X)]$$

= $E[E[D|Y_1, Y_0, X]|Y_1, Y_0, \pi(X)]$ (LIE)

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$$= \mathbf{E} \left[\mathbf{E}[D|X] | Y_1, Y_0, \pi(X) \right]$$
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(SOO)

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= $E[E[D|Y_1, Y_0, X]|Y_1, Y_0, \pi(X)]$ (LIE)
= $E[E[D|X]|Y_1, Y_0, \pi(X)]$ (SOO)
= $E[\pi(X)|Y_1, Y_0, \pi(X)]$
= $\pi(X)$

Using a similar argument

$$\Pr(D = 1|\pi(X)) = \operatorname{E}[D|\pi(X)] = \operatorname{E}[\operatorname{E}[D|X]|\pi(x)]$$

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Using a similar argument

$$Pr(D = 1|\pi(X)) = E[D|\pi(X)] = E[E[D|X]|\pi(x)]$$
$$= E[\pi(X)|\pi(X)] = \pi(X)$$

therefore $\Pr(D = 1 | Y_1, Y_0, \pi(X)) = \Pr(D = 1 | \pi(X))$

 Given selection on observables we have (Y₁, Y₀) ⊥LD|π(X) which implies the balancing property of the propensity score:

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- Estimate \mapsto Check Balance \mapsto Re-estimate \mapsto Check Balance

Weighting with the Propensity Score

Intuition

• Treated and control samples are unrepresentative of the overall population.

Weighting with the Propensity Score

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- Treated and control samples are unrepresentative of the overall population.
- Leads to imbalance in the covariates.

Weighting with the Propensity Score

Intuition

- Treated and control samples are unrepresentative of the overall population.
- Leads to imbalance in the covariates.
- Reweight them to be more representative.

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$$\sum_{i=1}^{n} \pi_i = n$$

$$\mathbf{E}\left[\frac{1}{n}\sum_{i=1}^{N}Z_{i}Y_{i}\right]=\frac{1}{n}\sum_{i=1}\pi_{i}Y_{i}$$

• Sample mean is biased:

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• Reweights the sample to be representative of the population.

• With a completely randomized experiment, we can just use the simple differences in means:

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• With subclassification, we binned X_i, calclulated within-bin differences and then averaged across the bins, just like this.

Stewart (Princeton)

Searching for the weights

$$\mathbf{E}[Y_i(d)] = \sum_{x \in \mathcal{X}} \mathbf{E}[Y_i | D_i = d, X_i = x] \mathbb{P}(X_i = x)$$

• Compare this to the the within treatment group average:

$$\mathbf{E}[Y_i|D_i = d] = \sum_{x \in \mathcal{X}} \mathbf{E}[Y_i|D_i = d, X_i = x] \mathbb{P}(X_i = x|D_i = d)$$

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• How should we reweight the data from an observational study?

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- How should we reweight the data from an observational study?
- If we were to reweight the data by W_i = 1/P(D_i = d|X_i), then we would break the relationship between D_i and X_i.

• Single binary covariate. Define the weight function:

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• If $(D_i, X_i) = (0, 0)$:

$$W_i = rac{1}{1 - e(0)} = rac{1}{\mathbb{P}(D_i = 0 | X_i = 0)}$$

$$\begin{array}{c|cccc} X_i = 0 & X_i = 1 \\ \hline D_i = 0 & 4 & 3 \\ D_i = 1 & 4 & 9 \end{array}$$

•
$$\mathbb{P}(D_i = 1 | X_i = 0) = 0.5$$

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$$\begin{array}{c|cccc} & X_i = 0 & X_i = 1 \\ \hline D_i = 0 & 1/0.5 & 1/0.25 \\ D_i = 1 & 1/0.5 & 1/0.75 \end{array}$$

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• Weighted data (the pseudo-population):

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• $\mathbb{P}_{W}(D_{i} = 1 | X_{i} = x) = 0.5$ for all x

$$\mathbb{P}_W[D_i=1|X_i=x]$$

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Important point: P_W(D_i = 1|X_i = 1) = P_W(D_i = 1|X_i = 0) = 1/ω*
→ D_i independent of X_i in the reweighted data.

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- *W_iY_i* is the weighted outcome, *D_i* is there to select out the treated observations.
- We want to see what the conditional weighted mean identifies:

$$\mathbf{E}\left[\frac{1}{N}\sum_{i=1}^{N}W_{i}D_{i}Y_{i}\right] = \frac{1}{N}\sum_{i=1}^{N}\mathbf{E}[W_{i}D_{i}Y_{i}] = \mathbf{E}[W_{i}D_{i}Y_{i}]$$

$$\mathbf{E}[W_i D_i Y_i] = \mathbf{E}\left[\frac{D_i Y_i}{e(X_i)}\right]$$

• Weighted mean of treated units is mean of potential outcome:

$$\mathbf{E}[W_i D_i Y_i] = \mathbf{E}\left[\frac{D_i Y_i}{e(X_i)}\right]$$
$$= E\left[\frac{D_i Y_i(1)}{e(X_i)}\right]$$

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(Propensity Score Definition)

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• These two facts provide an estimator for the average treatment effect:

$$\hat{\tau} = \frac{1}{N} \sum_{i=1}^{N} \left(\frac{D_i Y_i}{e(X_i)} - \frac{(1-D_i) Y_i}{1-e(X_i)} \right)$$

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• These two facts provide an estimator for the average treatment effect:

$$\hat{\tau} = \frac{1}{N} \sum_{i=1}^{N} \left(\frac{D_i Y_i}{e(X_i)} - \frac{(1-D_i) Y_i}{1-e(X_i)} \right)$$

- The above two results give us that this esimator is unbiased.
- This is sometimes called the Horvitz-Thompson estimator due to the close connection to the survey sampling estimator.

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• Obviously we often don't have the true propensity score, but in some circumstances, the estimate propensity score can be better than the true propensity score. Why?

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- The true p-score only adjusts for the expected differences between samples.

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 - \blacktriangleright \rightsquigarrow modeling via logit, etc.

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• Challenge: specifying the propensity score model.

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- The distribution of the estimates, $\hat{\tau}_b$, will give us the bootstrapped standard errors and confidence intervals.

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- We can actually put any other function of the treatment vector in the numerator, which can reduce the variation in the weights.
- We call these stabilized weights:

$$sw(d, x) = rac{\mathbb{P}[D_i = 1]^d (1 - \mathbb{P}[D_i = 1])^{1-d}}{e(x)^d (1 - e(x))^{1-d}}$$

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• These are the means that the weighted.mean() function in R calculates. It normalizes the weights before calculating the mean.

Stewart (Princeton)

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- Also remember, you don't need to include everything that predicts treatment, just variables on open backdoor paths (those that also influence the outcome).
- Propensity scores have also been used to think about treatment effect heterogeneity (see work by Jennie Brand and Yu Xie).

Assessing Counterfactuals

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- Put differently: what is the mechanism that drives a particular causal effect?
 - How do we get from cause to effect?

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 - Ø Moderation (effect modification, subgroup effects)
- We are going to focus on mediation today.

Notation

• Treatment variable D_i

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- Outcome variable Y_i

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- Treatment variable D_i
- Outcome variable Y_i
- An intermediate, post-treatment variable, *M_i*, which we call a mediator.

$$\begin{array}{c}
M_i \\
\uparrow \searrow \\
D_i \rightarrow Y_i
\end{array}$$

• Moderator: pretreatment variable that is correlated with the treatment effect.

 $Cov(\tau_i, X_i) \neq 0$

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• Mediator: a posttreatment variable that changes the effect of treatment.

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- Potential outcomes $Y_i(d, m)$: the value that the outcome takes when the treatment has value d and the mediator takes the value m.
- Consistency assumption to connect the potential outcomes to the observed outcomes:

$$egin{aligned} M_i &= M_i(D_i) \ Y_i &= Y_i(D_i, M_i(D_i)) \end{aligned}$$

Potential outcomes example

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- D_i is exercise, M_i is diet, and Y_i is weight.
- d is "run 10 km/day" and m is "eat 1500 kcals"
- $Y_i(d, m)$ is the weight you would have if we forced you to run 10 km/day and eat 1500 kcals a day.

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- We can define the typical individual causal effect, here called the total causal effect:

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• The total causal effect allows the effect of the treatment "propagate" through all causal pathways.

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Direct and indirect effects

• The indirect effect is the part of the effect of treatment that "flows through" the mediator



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• These are loose definitions, let's be precise.

$$\delta_i(d) = Y_i(d, M_i(1)) - Y_i(d, M_i(0))$$

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- If D_i doesn't affect M_i , so that $M_i(1) = M_i(0)$, then $\delta_i = 0$.
- Fundamental Problem of Causal Inference → focus on the average natural indirect effect (ANIE):

$$\overline{\delta}(d) = \mathbb{E}[\delta_i(d)] = \mathbb{E}[Y_i(d, M_i(1)) - Y_i(d, M_i(0))]$$

 $Y_i(1, M_i(1)) - Y_i(1, M_i(0))$

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 - Crossover experimental designs require strong no carry-over assumptions.
- Leads some to dismiss mediation altogether.
- However still an important topic for policy makers, theory-driven scholars etc.

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• Thus, the natural direct effect is the effect of moving from control to treatment while holding the mediator fixed at the value it would have under treatment status *d*.

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- But what would happen if we created a tar-less cigarette?
 - So that $M_i(1) = M_i(0)$ for all *i*.
- NDE answers this question.

Effect decomposition

• The total causal effect and the natural indirect and direct causal effects are related:

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• The fact that we can decompose the total effect of treatment into the sum of a direct and indirect effect if very important to social science researchers.

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 - ANDE: set M_i to $M_i(0)$ for all units
- ACDE is identified under weaker conditions than the ANDE but it does not create a nice decomposition of effects.

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• This holds in a (well-executed) experiment

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- Is this plausible? It depends on the application.

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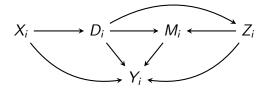
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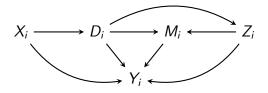


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More on this in a bit.

Identifying (in)direct effects

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- With a binary mediator and a binary treatment:

$$\bar{\delta}(d) = \{ \mathbb{P}[M_i = 1 | D_i = 1, X_i] - \mathbb{P}[M_i = 1 | D_i = 0, X_i] \} \\ \cdot \{ \mathbb{E}[Y_i | M_i = 1, D_i = d, X_i] - \mathbb{E}[Y_i | M_i = 0, D_i = d, X_i] \\ = (\text{effect of } D_i \text{ on } M_i) \times (\text{effect of } M_i \text{ on } Y_i) \}$$

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• Intuitive given the DAG:

$$\begin{array}{c}
M_i \\
\uparrow \\
D_i \rightarrow Y_i
\end{array}$$

(In)direct effects with non-binary mediators

• Let's say that the mediator has J categories:

$$ANIE(d) = \sum_{m=0}^{J-1} \mathbb{E}[Y_i | M_i = m, D_i = d, X_i] \\ \cdot \{\mathbb{P}[M_i = m | D_i = 1, X_i] - \mathbb{P}[M_i = m | D_i = 0, X_i]\}$$

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• The ANDE is the effect of D_i for a fixed m, averaged over the distribution of M_i when $D_i = 0$.

• Robins proposed a different identification strategy, based on a no-interactions assumption:

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- \rightarrow ACDE = ANDE.
- Strong assumption because it has to hold at the individual level (like monotonicity for IV).

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Stewart (Princeton)

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- For the sake of saving time and not promoting confusion, I'm going to skip these alternatives.
- In general though: if someone says this is easy- they are fooling themselves.

Nonparametric estimation

• How far can we get with nonparametrics?

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- If the number of categories in M_i , D_i , and X_i are small, use plug-in estimator for the CEF of Y_i :

$$\widehat{\mathbf{E}}[Y_i|M_i = m, D_i = d, X_i = x] = \frac{\sum_i Y_i \mathbb{V}\{M_i = m, D_i = d, X_i = x\}}{\sum_i \mathbb{W}\{M_i = m, D_i = d, X_i = x\}}$$

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• Same for M_i :

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- Need to be careful with the curse of dimensionality in X_i. Use good nonparametric strategies (cross-validation, etc)

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- Modeling *M_i* probably appropriate here.

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 - Outcome model: $p(Y_i|D_i, M_i, X_i)$
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- Evaluate sensitivity to assumptions

See mediation package in R.

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- These things are hard and relatively new. Adding another variable massively increases the assumptions. Adding a separate analysis messes with the decomposition.

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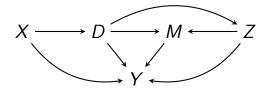
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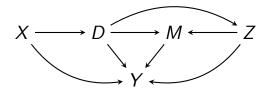
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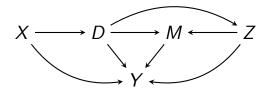


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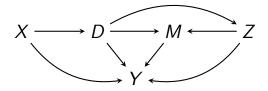


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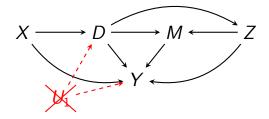


- Can also be thought of as other mediators, about which we aren't directly interested.
- Avin, Shpitser and Pearl (2003) showed that ANDE/ANIE identification not possible when SI incorporates intermediate confounders.



• New version of sequential ignorability with intermediate confounders:

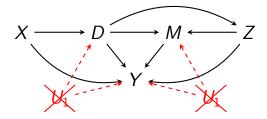
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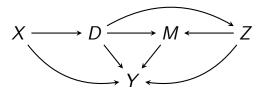
Identifying the ACDE

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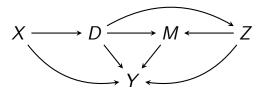
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- Relationship can generalized to any number of treatments, and is called the g-formula by Robins.

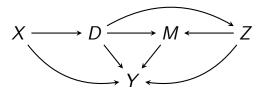
Stewart (Princeton)



• Controlling for Z_i and $M_i \sim$ posttreatment bias

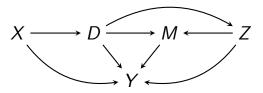


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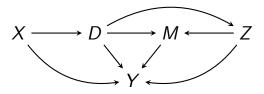
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$$E[Y_i|x, d = 1, z, m] - E[Y_i|x, d = 0, z, m]$$



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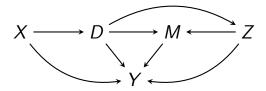
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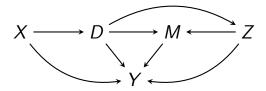
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• And the identification result from the g-formula:

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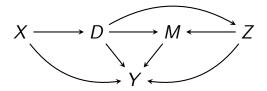


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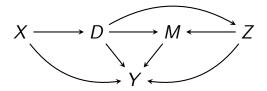
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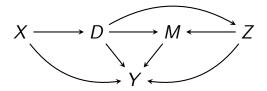


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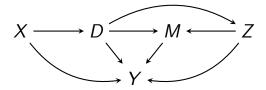


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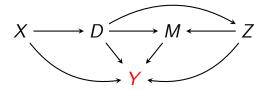
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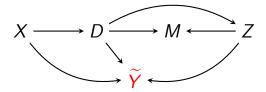
- γ_1 is not the CDE (posttreatment bias)
- γ_2 is the effect of M_i on Y_i



• Create a blipped down (or demediated) outcome: $\widetilde{Y}_i = Y_i - \widehat{\gamma}_2 M_i$

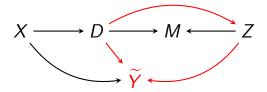


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- Acharya, Blackwell and Sen (2016) is a great paper on this.



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PSM's Statistical Properties (Nielsen and King 2016)

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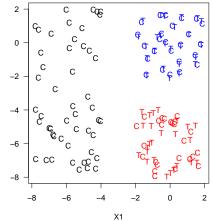
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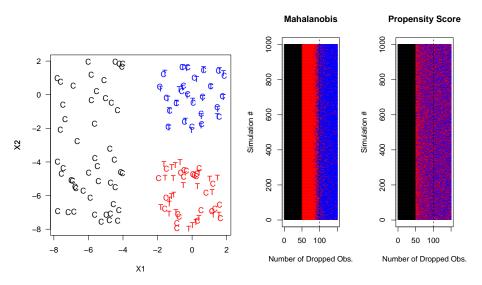
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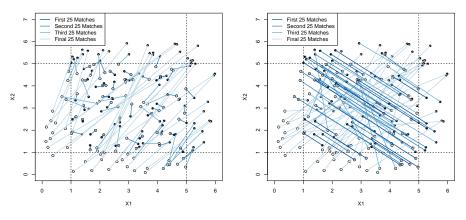
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What Does PSM Match?

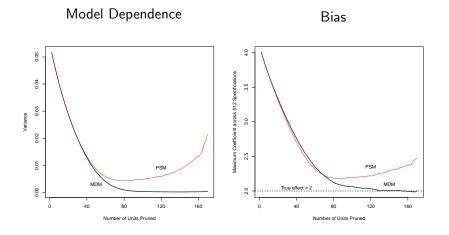
MDM Matches

PSM Matches



Controls: $X_1, X_2 \sim \text{Uniform}(0,5)$ Treateds: $X_1, X_2 \sim \text{Uniform}(1,6)$

PSM Increases Model Dependence & Bias



$$Y_i = 2T_i + X_{1i} + X_{2i} + \epsilon_i$$

$$\epsilon_i \sim N(0, 1)$$

The Propensity Score Paradox

Finkle et al. (2012) Nielsen et al. (2011) 30 10-25-8 20mbalance Imbalance 6 15-Random Raw Random PSM 4-Raw 10-1/4 SD caliper △1/4 SD caliper 2. CEM CEM 5 MDM MDM 0 0 500 500 0 1000 2000 2500 3000 0 1000 1500 2000 2500 1500 Number of units pruned Number of units pruned

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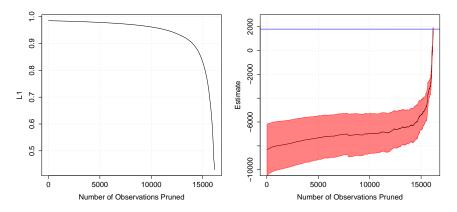
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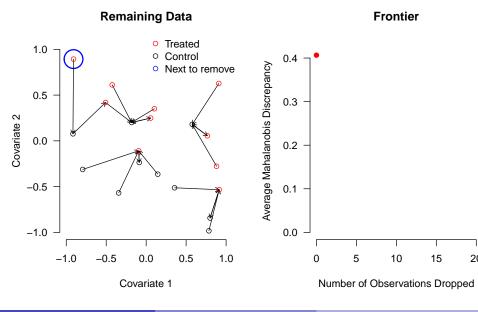
Job Training Data: Frontier and Causal Estimates



- 185 Ts; pruning most 16,252 Cs won't increase variance much
- Huge bias-variance trade-off after pruning most Cs
- Estimates converge to experiment after removing bias
- No mysteries: basis of inference clearly revealed

Stewart (Princeton)

Causal Inference



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