### CISER Causal Inference Workshop

### Session 2: Advanced Confounder Control

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### Confounder

A **confounder** is a variable that is associated with both the treatment and the outcome.

If we do not control for the confounder, the estimated treatment effect may be biased, because the confounder is unbalanced between the treatment and control groups.

## Simpson's Paradox

The batting averages of two baseball players in 1995 and 1996:

Year Batter	1995		1996		Combined	
Derek Jeter	12/48	.250	183/582	.314	195/630	.310
David Justice	104/411	.253	45/140	.321	149/551	.270

\* batting average = hits / at-bats.

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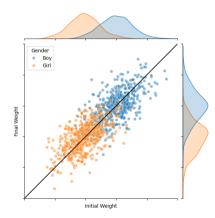
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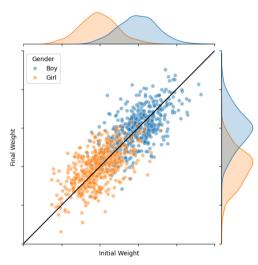
- Each year, player Jeter had a higher batting average than player Justice.
- ► The overall average of Justice is higher than Jeter.
- Time is a confounder (for batting average and for number of at-bats).

## Lord's Paradox

- A large university is interested in investigating the effects on the students of the diet provided in the university dining halls and any sex differences in these effects.
- Two groups: boys vs girls.
- Outcome: weight gain.



## Lord's Paradox

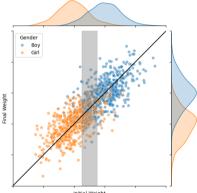


Statistician 1:

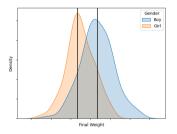
- Average weight gain for boys  $\sim 0$ .
- Average weight gain for girls  $\sim 0$ .
- Conclusion: no significant difference in weight gain between boys and girls.

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## Lord's Paradox



Initial Weight



Statistician 2:

- Choose students with similar initial weights.
- The average weight gains are significantly different for boys and girls.
- Conclusion:

boys have higher weight gains than girls.

### **Explanations**

#### Holland and Rubin (1983):

- Statistician 1 looks at the average descriptive statistics of the two groups.
- The two groups in nature have different distributions of initial weights (a confounder).
- Statistician 2 conditions on the confounder and compares the weight gains.
- Statistician 2's solution is aligned with the potential outcome framework.

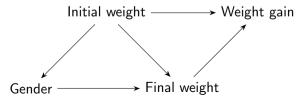
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### Pearl (2014):

Should consider the graphical causal model and do calculus.



## Super-Population Perspective and Unconfoundedness

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**Unconfoundedness** (under super-population perspective) is an assumption on the joint distribution of  $(Y_i(1), Y_i(0), W_i, X_i)$ :

$$(Y_i(0) \mid X_i, W_i = 1) \stackrel{D}{=} (Y_i(0) \mid X_i, W_i = 0)$$
 for all *i*

and

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Or, we can write it as

 $W_i \perp (Y_i(0), Y_i(1)) \mid X_i.$ 

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### Unconfoundedness

Unconfoundedness implies the connection between the observed potential outcomes and the missing potential outcomes:

$$\left(Y_i^{mis} \mid X_i, W_i = w\right) \stackrel{D}{=} \left(Y_i^{obs} \mid X_i, W_i = 1 - w\right)$$
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- ▶ The unconfoundedness assumption is **not** testable.
- The common practice is to include all the pre-treatment covariates in  $X_i$ .

It is usually impossible to find many pair of units from each of the treated and control groups that have the same values of  $X_i$ .

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Choices of  $b(X_i)$  include:

- $\blacktriangleright$   $X_i$  itself.
- ▶ Propensity score:  $e(X_i)$ .
- Expert-selected subsets/transformations of  $X_i$ .

#### Unconfoundedness Given a Balancing Score:

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#### **Coarseness of Balancing Scores:**

The propensity score is the coarsest balancing score. That is the propensity score is a function of any other balancing score.

## Causal Methods using Balancing Scores

- Model-based Imputation: Impute the missing potential outcomes using the covariates.
- Matching: Match treated and control units with similar values of the balancing score.
- **Stratification**: Stratify the sample based on the values of the balancing score.

• Weighting: Weight the treated and control units.

Suppose we consider a linear regression model for the potential outcomes:

$$\begin{pmatrix} Y_i(0) \\ Y_i(1) \end{pmatrix} = \begin{pmatrix} X_i \beta_0 \\ X_i \beta_1 \end{pmatrix} + \begin{pmatrix} \epsilon_{i0} \\ \epsilon_{i1} \end{pmatrix}, \quad \text{with } \begin{pmatrix} \epsilon_{i0} \\ \epsilon_{i1} \end{pmatrix} \sim \mathcal{N} \left( \begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{bmatrix} \sigma_0^2 & \sigma_0 \sigma_1 \\ \sigma_0 \sigma_1 & \sigma_1^2 \end{bmatrix} \right)$$

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The coefficients can be estimated by OLS:

$$\hat{\beta}_0 = \operatorname*{arg\,min}_{\beta} \sum_{i:W_i=0} (Y_i^{obs} - X_i\beta)^2, \quad \hat{\beta}_1 = \operatorname*{arg\,min}_{\beta} \sum_{i:W_i=1} (Y_i^{obs} - X_i\beta)^2$$

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The average treatment effect can be estimated by

$$\hat{\tau}^{ols} = \frac{1}{N} \sum_{i=1}^{N} \left[ W_i (Y_i^{obs} - X_i \hat{\beta}_0) + (1 - W_i) (X_i \hat{\beta}_1 - Y_i^{obs}) \right]$$

 $\hat{\beta}_0$  is fitted using  $\{X_i: W_i = 0\}$ , but  $\hat{\beta}_0$  is used in prediction for  $\{X_i: W_i = 1\}$ .

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- Solution: the observations should be weighted in OLS such that the covariates are balanced in distribution between the two groups.

The inverse propensity score weighting (IPW) is a popular method to balance the covariates.

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Two properties of the propensity score in weighting:

### Unbiasedness:

$$\mathbb{E}\left(\frac{W_i Y_i^{obs}}{e(X_i)}\right) = \mathbb{E}(Y_i(1)), \quad \mathbb{E}\left(\frac{(1-W_i)Y_i^{obs}}{1-e(X_i)}\right) = \mathbb{E}(Y_i(0)),$$

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#### Balanced Covariates:

$$\frac{p(X_i \mid W_i = 1)}{p(X_i \mid W_i = 0)} \propto \frac{e(X_i)}{1 - e(X_i)} \implies \frac{p(X_i \mid W_i = 1)}{e(X_i)} \propto \frac{p(X_i \mid W_i = 0)}{1 - e(X_i)}$$

The density of IPW-ed  $X_i$  is the same for the two groups.

The IPW estimator of the average treatment effect is

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

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- Advantage: always unbiased under unconfoundedness.
- Disadvantage: the variance of the estimator can be large due to extreme propensity scores.
- It is also called the Horvitz-Thompson estimator in survey sampling.
- **Caution**:  $e(X_i)$  still remains to be estimated.

A few ways to solve extreme propensity scores.

- Trimming: exclude the units with extreme propensity scores. Drawback: lose information.
- Truncation: saturate the propensity scores at a certain level. Drawback: added bias.
- ▶ Hájek Estimator: reduces variance by normalizing weights.

$$\hat{\tau}^{hajek} = \frac{\sum_{i} W_i Y_i^{obs} / e(X_i)}{\sum_{i} W_i / e(X_i)} - \frac{\sum_{i} (1 - W_i) Y_i^{obs} / (1 - e(X_i))}{\sum_{i} (1 - W_i) / (1 - e(X_i))}$$

Drawback: added bias for finite sample.

## Subclassification on Propensity Scores

We divide the domain of the propensity score into J strata:

$$b_0 = 0 < b_1 < b_2 < \dots < b_{J-1} < b_J = 1.$$

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For each stratum j, the difference-in-means is

$$\hat{\tau}^{dif}(j) = \frac{1}{N_t(j)} \sum_{i:b_{j-1} < e(X_i) < b_j} W_i Y_i^{obs} - \frac{1}{N_c(j)} \sum_{i:b_{j-1} < e(X_i) < b_j} (1 - W_i) Y_i^{obs}$$

The overall estimator is

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Advantage: the variance is reduced by stratification.

Disadvantage: potential bias due to the discretization of the propensity score.

#### Subclassification on Propensity Scores

For each stratum j, the bias comes from

- We use  $\bar{Y}_t^{obs}(j)$  to estimate  $\mathbb{E}(Y_i(1) \mid W_i = 0)$ .
- We use  $\bar{Y}_c^{obs}(j)$  to estimate  $\mathbb{E}(Y_i(0) \mid W_i = 1)$ .

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#### Bias Correction:

▶ We fit a parametric model for the potential outcomes:

 $Y_i(W_i) = f(X_i, W_i; \theta) + \epsilon_i.$ 

The model can be as simple as linear.

- ▶ We estimate the difference between  $\mathbb{E}(Y_i(1) | W_i = 0)$  and  $\mathbb{E}(Y_i(1) | W_i = 0)$  using the fitted model and the discrepancy in the covariates.
- The bias is substrated from the stratified estimator.

Consider an experiment with more treated than control units. The **matching** method is to find for each treated unit a **similar** control unit.

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#### closeness:

- **Exact Matching**: the treated and control units have the same values of the covariates. Often for categorical variables.
- Metric Matching: the treated and control units are close in the covariate space. Often for continuous variables. E.g. using Mahalanobis distance.
- Propensity Score Matching: the treated and control units have similar propensity scores.

$$d(i,j) = \left[\log \frac{e(X_i)}{1 - e(X_i)} - \log \frac{e(X_j)}{1 - e(X_j)}\right]^2$$

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#### Replacement:

- **With Replacement**: the control unit can be matched to multiple treated units.
- **Without Replacement**: the control unit can be matched to only one treated unit.

Algorithms:

- Greedy Matching 1: for each treated unit, find the available closest control unit. Repeat until all treated units are matched.
- **Greedy Matching 2**: within all avaiable pairs, find the closest pair. Repeat until all treated units are matched.

 Optimal Matching: find the best matching pairs that minimize the total distance. (NP-hard, optimal transportations)

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#### **Common Practices:**

- Poor matches are often excluded.
- Could also use one-to-many matching using a caliper.
- Balance of the covariates should be checked after matching.
- Cautious: distribution shift after matching!!!

The estimator is

$$\hat{\tau}^{match} = \frac{1}{N_t} \sum_{i:W_i=1} (Y_i^{obs} - Y_{m(i)}^{obs}),$$

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where m(i) is the matched control unit for treated unit i.

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We are estimating the **average treatment effect for the treated units**, not for the population.

Finite Sample Version:

$$\tau_{fs,t} = \frac{1}{N_t} \sum_{i:W_i=1} (Y_i(1) - Y_i(0))$$

Super-Population Version:

$$\tau_{sp,t} = \mathbb{E}(Y_i(1) - Y_i(0) \mid W_i = 1)$$

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If the matching is **exact**, the estimator is unbiased.

$$E(Y_i^{obs} - Y_{m(i)}^{obs} \mid X_i) = E(Y_i(1) - Y_i(0) \mid X_i).$$

The variance of the estimator  $\hat{\tau}_t^{match}$  can be estimated from the sample variance of the differences.

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- The bias is due to the difference in the covariates/propensity scores between the treated and control units.
- ▶ The bias can be corrected by the regression adjustment.

One-to-many matching can be used to reduce bias and variance of the estimator.

- Bias is reduced because multiple control units are matched to a treated unit smaller discrepancy in the averaged covariates.
- Variance is reduced because the sample size is increased. But the improvement in variance is limited.

To estimate the average treatment effect for the control  $\tau_{sp,c}$ , we can match all controlled units instead.

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To estimate the average treatment effect for the population  $\tau_{sp}$ , we need to estimate both  $\tau_{sp,t}$  and  $\tau_{sp,c}$  and combine them.

$$\tau_{sp} = \frac{N_t}{N} \tau_{sp,t} + \frac{N_c}{N} \tau_{sp,c}$$

#### Other Technical Issues

All the propensity scores need to be estimated in the first place. The most common model is a logistic regression model:

$$\log \frac{P(W_i = 1 \mid X_i)}{1 - P(W_i = 1 \mid X_i)} = X_i \beta.$$

- The model specification is crucial. The model should be flexible enough to capture the relationship between the covariates and the treatment assignment.
- Besides the caual effect estimators, the standard errors should also be estimated. We skip the details here.
- The hypothesis testing for the average treatment effect usually follows a Z-test (when the sample size is large).

We take one example from Imbens and Rubins' book to demonstrate the techniques in balancing the covariates.

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- Treatment: prenatal exposure to barbiturates
- Covariates: quite a few regarding the born child and the mother.
- Outcome: cognitive development measured many years later
- ▶ 745 treated and 7198 control.
- It is an observational study.

Label	Variable Description		trols =7198)		ated =745)	t-Stat
		Mean	(S.D.)	Mean	(S.D.)	Difference
sex	Sex of child (female is 0)	0.51	(0.50)	0.50	(0.50)	-0.3
antih	Exposure to antihistamine	0.10	(0.30)	0.17	(0.37)	4.5
hormone	Exposure to hormone treatment	0.01	(0.10)	0.03	(0.16)	2.5
chemo	Exposure to chemotherapy agents	0.08	(0.27)	0.11	(0.32)	2.5
cage	Calendar time of birth	-0.00	(1.01)	0.03	(0.97)	0.7
cigar	Mother smoked cigarettes	0.54	(0.50)	0.48	(0.50)	-3.0
lgest	Length of gestation (10 ordered categories)	5.24	(1.16)	5.23	(0.98)	-0.3
lmotage	Log of mother's age	-0.04	(0.99)	0.48	(0.99)	13.8
lpbc415	First pregnancy complication index	0.00	(0.99)	0.05	(1.04)	1.2
lpbc420	Second pregnancy complication index	-0.12	(0.96)	1.17	(0.56)	55.2
motht	Mother's height	3.77	(0.78)	3.79	(0.80)	0.7
motwt	Mother's weight	3.91	(1.20)	4.01	(1.22)	2.0
mbirth	Multiple births	0.03	(0.17)	0.02	(0.14)	-1.9
psydrug	Exposure to psychotherapy drugs	0.07	(0.25)	0.21	(0.41)	9.1
respir	Respiratory illness	0.03	(0.18)	0.04	(0.19)	0.7
ses	Socioeconomic status (10 ordered categories)	-0.03	(0.99)	0.25	(1.05)	7.0
sib	If sibling equal to 1, otherwise 0	0.55	(0.50)	0.52	(0.50)	-1.6

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Step 1: Estimate the propensity score.

- ► The propensity score is estimated by a logistic regression model.
- Due to the large number of parameters and possible interactions, the logistic regression model selects covariates by a sequential manner.

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Variable	EST	( <u>s.e.</u> )	t-Stat
Intercept	-5.67	(0.23)	-24.4
Linear terms			
sex	0.12	(0.09)	1.3
lmotage	0.52	(0.11)	4.7
ses	0.06	(0.09)	0.6
lpbc420	2.37	(0.36)	6.6
mbirth	-2.11	(0.36)	-5.9
chemo	-3.51	(0.67)	-5.2
psydrug	-3.37	(0.55)	-6.1
sib	-0.24	(0.22)	-1.1
cage	-0.56	(0.26)	-2.2
lgest	0.57	(0.23)	2.5
motwt	0.49	(0.17)	2.9
cigar	-0.15	(0.10)	-1.5
antih	0.17	(0.13)	1.3

#### Second-order terms

$lpbc420 \times sib$	0.60	(0.19)	3.1
motwt× motwt	-0.10	(0.02)	-4.5
lpbc420 × psydrug	1.88	(0.39)	4.8
ses× sib	-0.22	(0.10)	-2.2
cagex antih	-0.39	(0.14)	-2.8
$lpbc420 \times chemo$	1.97	(0.49)	4.0
$lpbc420 \times lpbc420$	-0.46	(0.14)	-3.3
cage x lgest	0.15	(0.05)	3.0
lmotage × lpbc420	-0.24	(0.10)	-2.5
mbirth× cage	-0.88	(0.39)	-2.3
lgest × lgest	-0.04	(0.02)	-2.0
sesx cigar	0.20	(0.09)	2.2
lpbc420×motwt	0.15	(0.07)	2.0
chemo × psydrug	-0.93	(0.46)	-2.0
lmotage× ses	0.10	(0.05)	1.9
cage x cage	-0.10	(0.05)	-1.8
mbirth × chemo	$-\infty$	(0.00)	$-\infty$

Step 2: Stratification.

- ► The stratification is based on the propensity score.
- An adaptive procedure is conducted:
  - Starting from one stratum, the balance of the covariates is checked.
  - If the balance is not achieved, the stratum is split into two new stratum of equal size.

Repeat until all strata are balanced.

Step	Block	Lower Bound	Upper Bound	Width	# Controls	# Treated	t-Stat
1	1	0.00	0.94	0.94	4462	742	36.3
2	1	0.00	0.06	0.06	2540	61	3.2
2	2	0.06	0.94	0.88	1922	681	23.7
3	1	0.00	0.02	0.01	1280	20	2.2
	2	0.02	0.06	0.05	1260	41	0.5
	3	0.06	0.20	0.14	1163	138	3.9
	4	0.20	0.94	0.74	759	543	10.9
4	1	0.00	0.01	0.00	644	6	-0.0
	2	0.01	0.02	0.01	636	14	1.7
	3	0.02	0.06	0.05	1260	41	0.5
	4	0.06	0.11	0.05	604	46	-0.3
	5	0.11	0.20	0.09	559	92	1.0
	6	0.20	0.37	0.17	458	192	1.2
	7	0.37	0.94	0.57	301	351	5.6

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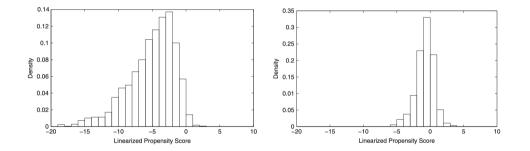
5	1	0.00	0.01	0.00	644	6	-0.0
	2	0.01	0.02	0.01	636	14	1.7
	3	0.02	0.06	0.05	1260	41	0.5
	4	0.06	0.11	0.05	604	46	-0.3
	5	0.11	0.20	0.09	559	92	1.0
	6	0.20	0.37	0.17	458	192	1.2
	7	0.37	0.50	0.13	181	144	2.5
	8	0.50	0.94	0.44	120	207	2.3
6	1	0.00	0.01	0.00	644	6	-0.0
	2	0.01	0.02	0.01	636	14	1.7
	3	0.02	0.06	0.05	1260	41	0.5
	4	0.06	0.11	0.05	604	46	-0.3
	5	0.11	0.20	0.09	559	92	1.0
	6	0.20	0.37	0.17	458	192	1.2
	7	0.37	0.42	0.05	101	61	0.3
	8	0.42	0.50	0.08	80	83	0.7
	9	0.50	0.61	0.11	73	90	0.8
	10	0.61	0.94	0.34	47	117	-0.3

	Within Blocks										Overall		1-Block
	1	2	3	4	5	6	7	8	9	10	t-Test	F-Test (z-Value)	t-Test
Covariate													
sex	-0.05	-2.27	1.97	0.81	0.89	-1.28	0.04	-0.39	-1.42	1.14	0.13	1.22	-0.73
antih	-0.67	-0.47	0.67	0.03	0.37	-0.25	0.38	-0.53	-0.11	0.27	-0.17	-2.88	3.21
hormone	-0.14	-0.42	-0.65	-1.00	0.25	0.71	-0.22	-1.05	-1.10	0.21	-0.99	-0.66	1.66
chemo	0.55	-0.39	-0.78	-0.75	-1.17	1.47	-0.94	0.61	0.66	0.29	-0.27	-0.61	1.76
cage	-1.41	-0.29	-1.04	-0.46	2.11	0.28	0.20	0.46	-1.48	-0.74	-1.38	0.34	1.15
cigar	-0.37	0.55	0.58	1.50	0.31	-0.93	0.21	-0.99	0.25	-0.39	0.52	-1.17	-3.13
lgest	0.90	0.58	-0.07	-0.82	0.79	-0.36	0.05	-0.33	-1.14	1.21	0.71	-1.48	0.12
lmotage	-2.20	-1.37	0.56	1.64	0.95	0.60	-0.96	-1.73	-1.47	0.36	-1.26	1.45	8.56
lpbc415	-0.48	-1.84	-1.00	-0.34	0.59	0.44	-0.20	-0.16	1.07	-0.10	-1.49	-0.82	0.75
lpbc420	1.04	0.84	-0.67	-0.86	-1.61	1.80	-0.39	1.62	1.14	-1.80	0.51	0.59	32.04
motht	-0.84	0.45	-0.67	0.75	0.64	0.09	0.30	-1.37	-0.60	-0.13	-0.50	-1.37	0.90
motwt	1.23	1.14	0.12	-1.23	-0.05	-0.45	-0.32	1.94	-0.01	-0.47	1.08	-0.18	1.44
mbirth	-0.44	-0.80	-1.54	-0.37	1.80	0.20	0.00	2.25	-1.58	-1.60	-1.28	1.00	-2.93
psydrug	-0.66	-1.01	1.05	-0.15	-0.78	0.06	-0.18	0.08	0.09	0.89	-0.29	-1.40	6.32
respir	-0.49	0.53	-0.21	0.98	1.38	0.24	-0.78	-1.51	0.22	-0.28	0.24	-0.49	0.19
ses	-0.60	-0.31	-0.74	1.16	0.82	-0.08	-0.03	-0.82	-0.91	0.36	-0.56	-1.37	5.19
sib	1.42	2.37	-1.09	-1.58	-1.53	0.11	0.63	1.63	1.19	0.23	0.98	1.64	1.48

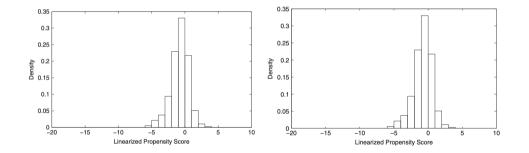
Step 3: Matching Method.

The matching is based on the Mahalanobis distance or the propensity scores.

▶ The performance is check by the overlapping scores of the covariates.



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		Full	Sample		Matched Samples							
						Mahalanobis		Propensity Score				
	Nor	Log Rat	$\pi^{0}$	05	Nor	Log Rat	$\pi^{0}$	.05	Nor	Log Rat	π <sup>0</sup>	05
	Dif	of STD	Controls	Treated	Dif	of STD	Controls	Treated	Dif	of STD	Controls	Treated
sex	-0.01	0.00	1.00	1.00	0.00	-0.00	1.00	1.00	-0.03	0.00	1.00	1.00
antih	0.19	0.20	1.00	1.00	0.02	0.01	1.00	1.00	-0.03	-0.02	1.00	1.00
normone	0.11	0.43	1.00	0.97	0.00	0.00	1.00	1.00	0.01	0.03	1.00	0.97
chemo	0.10	0.14	1.00	1.00	0.00	0.00	1.00	1.00	0.08	0.10	1.00	1.00
cage	0.03	-0.04	0.93	0.97	-0.03	0.03	0.96	0.95	-0.01	-0.00	0.95	0.95
cigar	-0.12	0.00	1.00	1.00	-0.01	-0.00	1.00	1.00	-0.01	-0.00	1.00	1.00
lgest	-0.01	-0.17	0.95	0.98	-0.02	0.13	0.98	0.97	0.00	0.01	0.98	0.97
lmotage	0.53	0.00	0.93	0.93	0.13	0.02	0.97	0.95	0.02	-0.01	0.95	0.97
lpbc415	0.05	0.06	0.99	0.97	0.03	0.06	0.98	0.99	0.07	-0.06	0.99	0.97
lpbc420	1.63	-0.55	0.52	0.72	0.59	-0.01	0.90	0.86	0.10	0.09	0.96	0.94
notht	0.03	0.03	1.00	1.00	-0.03	0.15	1.00	1.00	-0.03	0.03	1.00	1.00
notwt	0.08	0.02	1.00	1.00	0.02	0.09	1.00	1.00	0.05	-0.02	1.00	1.00
nbirth	-0.07	-0.21	0.97	1.00	0.00	0.00	0.98	0.98	0.03	0.12	0.99	0.98
psydrug	0.41	0.47	1.00	1.00	0.00	0.00	1.00	1.00	0.13	0.09	1.00	1.00
respir	0.03	0.07	1.00	1.00	0.00	0.00	1.00	1.00	0.03	0.07	1.00	1.00
ses	0.28	0.06	1.00	1.00	0.03	0.08	0.99	0.96	-0.04	0.02	0.99	0.96
sib	-0.06	0.00	1.00	1.00	0.03	-0.00	1.00	1.00	0.04	-0.00	1.00	1.00
Multivariate measure	0.43				0.24				0.05			
	1 (7	0.70	~	0.70	1.00	0.00	0.00	0.00	0.00	0.11	0.07	0.00

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# Summary

#### Model Imputation Methods:

- Advantages: easy to implement, flexible.
- Disadvantages: model specification is crucial, extrapolation is a big issue.

#### Weighting Methods:

- Advantages: unbiased, balance the covariates.
- Disadvantages: variance can be large, extreme propensity scores.

#### Stratification Methods:

- Advantages: reduce variance, balance the covariates.
- Disadvantages: bias due to discretization.

#### Matching Methods:

- Advantages: balance the covariates, reduce variance.
- Disadvantages: bias due to inexact matching, distribution shift.

The confounder control so far is based on a single treatment (cause). In reality, there could be many (cause, consequence) pairs.

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The Book of Why by Judea Pearl Causal inference in statistics: An overview by Judea Pearl Causal Inference in Statistics: A Primer by Judea Pearl

# Causal Diagram

A **causal diagram** is a directed acyclic graph (DAG) that represents the causal relationships between the variables.

▶  $A \rightarrow B$  means A affacts B directly.

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A **causal diagram** is a directed acyclic graph (DAG) that represents the causal relationships between the variables.

▶  $A \rightarrow B$  means A affacts B directly.



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It is very easy to identify the confounders in the causal diagram.

For the diagram above, Z is a confounder for the effect of X on Y.

#### Do Operator

The operator do(X = x) is used to represent the intervention of setting X to x.



Conventional probability:

$$P(y, z \mid X = x) = \frac{P(x, y, z)}{P(x)} = \frac{P(y \mid x, z)P(x \mid z)P(z)}{\int P(x \mid z)P(z)dz}$$

Do operator: (x is no longer random after intervention)

$$P(y, z \mid do(x)) = P(y \mid x, z)P(z)$$

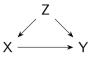
For any DAG causal diagram with independent noise terms, the conditional distribution conditioned on the do operator is

$$P(v_1, \dots, v_k \mid do(x_0)) = \prod_{i: v_i \neq X} P(v_i \mid pa_i)|_{x=x_0},$$

where  $pa_i$  is the parent set of  $v_i$  in the DAG.

#### Do Operator

Any unmeasured confounders should be integrated out.



We already know

$$P(y, z \mid do(x)) = P(y \mid x, z)P(z)$$

Therefore,

$$P(y \mid do(x)) = \sum_{z} P(y \mid x, z) P(z)$$

### Do Operator

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Therefore,

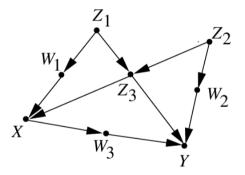
$$P(y \mid do(x)) = \sum_{z} P(y \mid x, z) P(z)$$

- As long as we can estimate  $P(y \mid x, z)$  and P(z) from the data, we can estimate  $P(y \mid do(x))$ .
- ▶ The **causal effect** of *X* on *Y* is

$$\tau = E(Y \mid do(x = 1)) - E(Y \mid do(x = 0))$$

In practice, if we want to estimate the causal effect of X on Y, there could be many confounders. We need to determine all the necessary confounders and adjust for them.

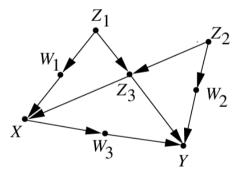
In practice, if we want to estimate the causal effect of X on Y, there could be many confounders. We need to determine all the necessary confounders and adjust for them.



Confounders for the effect of X on Y:  $Z_1, Z_2, Z_3$ .

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In practice, if we want to estimate the causal effect of X on Y, there could be many confounders. We need to determine all the necessary confounders and adjust for them.



Confounders for the effect of X on Y:  $Z_1, Z_2, Z_3$ .

Such a set of confounders is called a **admissible set**, such that as long as the set is adjusted for, the causal effect is identified.

#### Back-door Criterion:

A set S is admissible for adjustment if two conditions hold:

- 1. No element of S is a descendant of X.
- 2. S blocks all back-door paths between X and Y.

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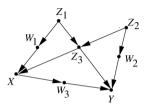
- 1. No element of S is a descendant of X.
- 2. S blocks all back-door paths between X and Y.

#### Back-door Adjustment Formula:

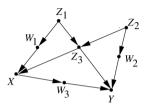
If S is an admissible set, then

$$P(Y = y \mid do(X = x)) = \sum_{s} P(Y = y \mid X = x, S = s)P(S = s)$$

where the summation is over all possible values of S.



- $\{Z_1, Z_3\}$  is an admissible set.
- $\triangleright$  { $Z_3$ } is an admissible set.
- $\{Z_1, Z_2\}$  is not an admissible set.

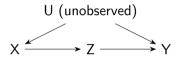


If we adjust for  $Z_3$ , the back-door adjustment formula is

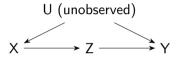
$$P(Y = y \mid do(X = x)) = \sum_{z_3} P(Y = y \mid X = x, Z_3 = z_3) P(Z_3 = z_3)$$

In practice, we only need to estimate the conditional distribution  $P(Y \mid X, Z_3)$  and the marginal distribution  $P(Z_3)$ .

Suppose we have the following causal diagram. But U is unobserved.



Suppose we have the following causal diagram. But U is unobserved.

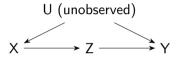


▶ The back-door adjustment does not work because U is unobserved.

• However, the path  $X \to U \to Y$  is blocked by U such that U works as a proxy for X.

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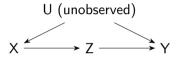


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- ▶ The effect of X on Z can be estimated.
- ▶ The effect of Z on Y can be estimated.

Suppose we have the following causal diagram. But U is unobserved.



- ▶ The back-door adjustment does not work because U is unobserved.
- However, the path  $X \to U \to Y$  is blocked by U such that U works as a proxy for X.
- ▶ The effect of *X* on *Z* can be estimated.
- ▶ The effect of Z on Y can be estimated.
- ▶ The effect of X on Y can be estimated by combining the two effects.

#### Front-door Criterion:

A set S satisfies the front-door criterion if three conditions hold:

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- 1. S intercepts all directed paths from X to Y.
- 2. There is no backdoor path from X to S.
- 3. All backdoor paths from S to Y are blocked by X.

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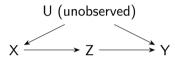
- 1. S intercepts all directed paths from X to Y.
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- 3. All backdoor paths from S to Y are blocked by X.

#### Front-door Adjustment Formula:

If S is an admissible set, then

$$P(Y = y \mid do(X = x)) = \sum_{s} \sum_{x'} P(Y = y \mid X = x', S = s) P(X = x') P(S = s \mid X = x).$$

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The set  $\{Z\}$  satisfies the front-door criterion. The front-door adjustment formula is

$$P(Y = y \mid do(X = x)) = \sum_{z} \sum_{x'} P(Y = y \mid X = x', Z = z) P(X = x') P(Z = z \mid X = x).$$

# Thank you for joining the workshop!

Contact: Me chencheng.cai@wsu.edu CISER ciser.info@wsu.edu