Week 4: Observational Designs

PLSC 30600 - Causal Inference

Last two weeks

- Identification under **ignorability**
  - Treatment assignment is independent of the potential outcomes
- Randomized experiments guarantee ignorability
  - Randomization ensures that treatment is independent of **observed** and **unobserved** confounders.
- How to use covariates in experiments
  - Improve precision for estimating the ATE
  - Subgroup effects

This week

- What happens when **ignorability** does not hold?
  - Treatment is not randomly assigned - we have an *observational* design.
  - Treatment assignment may be driven by other factors that also predict the outcome
- Selection-on-observables assumptions
  - Treatment is ignorable **conditional** on observed covariates
- Estimation under selection-on-observables
  - Stratification
  - Inverse Propensity of Treatment Weighting
  - Regression adjustment
  - Matching

Selection-on-observables

Why experiments worked

- A good causal observational study should try to mimic the features of an experiment
- So what were the nice properties of a **randomized experiment**?
  - **Positivity**: Assignment not deterministic $0 < P(D_i = 1) < 1$
  - **Ignorability/Unconfoundedness**: $P(D_i = 1|Y_i(1), Y_i(0) = P(D_i = 1)$
- We liked experiments because we could ensure treatment was independent of the potential outcomes.
  - $\{Y_i(1), Y_i(0)\} \perp\!\!\!\perp D_i$
- Even in a **conditionally** randomized experiment, we knew $P(D_i = 1|\mathbf{X}_i)$

Observational designs

- Complete **unconfoundedness** is only one kind of design assumption, but there are many settings where it won't hold.
- Suppose we didn't randomize an intervention but simply observe its occurrence
  - $P(D_i = 1)$ is not known.
  - Treatment and control groups might not be comparable. Why? -- confounders!
- Alternative design: **selection-on-observables**
  - Treatment assignment is ignorable **conditional** on a set of **observed** covariates $\mathbf{X}_i$
- Assumptions:
  - **Positivity/Overlap**: $0 < P(D_i = 1|\mathbf{X}_i) < 1$
  - **Conditional ignorability**: $\{Y_i(1), Y_i(0)\} \perp\!\!\!\perp D_i|\mathbf{X}_i$
    - Other names: "No unmeasured confounders", "selection on observables", "no omitted variables", "conditional exogeneity", "condtiional exchangeability", etc...

Approximating experiments

- A well-designed observational study will try to approximate some hypothetical "target" experiment (Rubin, 2008; Hernán and Robins, 2016).
  - Well-defined intervention
  - Clear distinction between treatment and pre-treatment covariates
- You should try to answer the following questions:
  - What's the intervention of interest?
  - What is the assignment process for the intervention?
  - How well does our adjustment model this assignment process?
- What kind of experiment are we mimicking with a **selection-on-observables** identification strategy
  - *Conditional* randomization (given $\mathbf{X}_i$).
  - Treatment probability is not constant across levels of $\mathbf{X}_i$.
- **Problem**: In an experiment we're guaranteed balance on the unobservables (by randomization). In a selection-on-observables design we are assuming these unobservables away!

Identification of the ATE

- Recall that under conditional ignorability, $\{Y_i(1), Y_i(0)\} \not\perp\!\!\!\perp D_i$
- Therefore:

$$E[Y_i(1)|D_i = 1] \neq E[Y_i(1)]$$

- So the difference in means alone will not identify the ATE -- we need to **condition** on the covariates $\mathbf{X}_i$

Identification of the ATE

- Iterated expectations

$$E_X\Big[E[Y_i|D_i=1,\mathbf{X}_i=x]\Big]-E_X\Big[E[Y_i|D_i=0,\mathbf{X}_i=x]\Big]$$

- Consistency:

$$E_X\Big[E[Y_i(1)|D_i=1,\mathbf{X}_i=x]\Big]-E_X\Big[E[Y_i(0)|D_i=0,\mathbf{X}_i=x]\Big]$$

- Conditional ignorability:

$$E_X\Big[E[Y_i(1)|\mathbf{X}_i=x]\Big]-E_X\Big[E[Y_i(0)|\mathbf{X}_i=x]\Big]$$

- Law of iterated expectations

$$E[Y_i(1)]-E[Y_i(0)]=\tau$$

Identification vs Estimation

- With infinite data, it would be possible to simply plug in sample analogues for $E[Y_i|D_i = 1, X_i = x]$ for each unique value of x (as long as positivity holds).
- But as the dimensionality of $\mathbf{X}_i$ grows large, within our sample this might be impossible (few to no observations for any given $\mathbf{X}_i$)
  - We'll make **additional** assumptions to address this problem as part of *estimating* these conditional expectations and consider different estimation strategies with different assumptions
  - But it's important not to confuse these assumptions (e.g. linearity in a regression model) with the *identification* assumptions needed to even get a causal quantity from the observed data.
- **Identification** assumptions
  - What do we need to assume is true about the world in order to get **any** causal quantity from the observed data?
  - In selection-on-observables designs: Treatment is independent of the potential outcomes conditional on observed covariates.
- **Estimation** assumptions
  - What do we need to assume in order to get a decent estimator of the treatment effect?
  - If these assumptions are wrong, might introduce additional bias **even if** ignorability holds
  - This is where fancy stats can help us!

Adjustment via stratification

- If our $\mathbf{X}_i$ are sufficiently low-dimensional, we don't really need any strong modeling assumptions to estimate the ATE. We can use our usual stratification/sub-classification estimator:

$$\hat{\tau} = \sum_{x \in \mathcal{X}} \hat{\tau(x)} \widehat{P}(\mathbf{X}_i = x)$$

where

$$\hat{\tau(x)} = \widehat{E}[Y_i | D_i = 1, X_i = x] - \widehat{E}[Y_i | D_i = 0, X_i = x]$$

- What happens if $\mathbf{X}_i$ is high-dimensional? **Coarsen** into bins:
  - Fewer bins = more (potential) bias.

Example: Washington (2008)

- Washington (2008; AER) examines whether having daughters affects a legislator's voting behavior on feminist/pro-women issues (measured by AAUW voting scores)
- Let's use the data to estimate the effect of having any daughters vs. having 0 daughters
- What's the unadjusted estimate?

```
washington <- read_dta("assets/washington.dta")
lm_robust(aauw ~ anygirls, data=washington)
```

```
##             Estimate Std. Error t value Pr(>|t|) CI Lower CI Upper  DF
## (Intercept)    49.47       3.94  12.554 4.93e-31     41.7    57.22 432
## anygirls       -2.83       4.59  -0.617 5.38e-01    -11.8     6.19 432
```

Example: Washington (2008)

- What's the confounder?
- Number of children!

```
# Number of girls by total number of children
table(washington$ngirls, washington$totchi)
```

```
##
##      0  1  2  3  4  5  6  7  8  9 10
##   0 60 15 28 12  3  0  1  0  0  0  0
##   1  0 25 79 33 14  6  0  0  0  0  0
##   2  0  0 31 37 24  7  1  1  0  0  0
##   3  0  0  0 12 13 12  1  1  1  0  0
##   4  0  0  0  0  3  5  2  1  1  0  1
##   5  0  0  0  0  0  1  0  1  0  1  0
##   7  0  0  0  0  0  0  0  0  1  0  0
```

```
# Association between total number of children and AAUW score
lm_robust(aauw ~ totchi, data=washington)
```

```
##             Estimate Std. Error t value Pr(>|t|) CI Lower CI Upper  DF
## (Intercept)    60.15       3.51   17.13 1.39e-50    53.25    67.05 432
## totchi         -5.11       1.08   -4.73 3.09e-06    -7.23    -2.98 432
```

Example: Washington (2008)

- Identification strategy: **selection-on-observables**
  - Conditional on the **total number of children**, the number of girls is assigned *as-if-random*
- For which strata can we not identify a treatment effect?
  - Legislators with 0 children! Positivity/overlap violation. By definition a legislator w/ 0 children can't have more than 0 girls
- Other strata are just very sparse (4+ children).
  - Let's estimate the "any child" effect for legislators with 1 to 3 children, stratifying on the total amount.
  - Note that we've changed the target population here.

Example: Washington (2008)

```
# Subset down to cases with overlap
wash_subset <- washington %>% filter(totchi>0&totchi<4)

# Unadjusted
washington_unadjusted <- lm_robust(aauw ~ anygirls, data=wash_subset)
tidy(washington_unadjusted) %>% filter(term == "anygirls") %>%
  select(term, estimate, std.error, p.value)
```

```
##       term estimate std.error p.value
## 1 anygirls     5.58      6.62     0.4
```

Example: Washington (2008)

```
# Get the difference-in-means in each stratum
stratum_ate <- wash_subset %>% group_by(totchi) %>%
  do(tidy(lm_robust(aauw ~ anygirls, data=.))) %>%
  select(totchi, term, estimate, std.error, df) %>%
  filter(term == "anygirls") %>% mutate(n=df+2) %>%
  ungroup()
stratum_ate
```

```
## # A tibble: 3 × 6
##   totchi term     estimate std.error    df     n
##    <dbl> <chr>       <dbl>     <dbl> <dbl> <dbl>
## 1      1 anygirls    -15.8     13.5     38    40
## 2      2 anygirls     14.8      9.18   136   138
## 3      3 anygirls     19.1     11.8     92    94
```

Example: Washington (2008)

```
# Weighted average to get the point estimate
stratum_ate %>% summarize(ate = sum(estimate*n/sum(n)),
                          std.err = sqrt(sum(std.error^2*(n/sum(n))^2))) %>%
  mutate(p.val = 2*(pnorm(-abs(ate/std.err))))
```

```
## # A tibble: 1 × 3
##     ate std.err  p.val
##   <dbl>   <dbl>  <dbl>
## 1  11.8    6.50 0.0701
```

Example: Washington (2008)

- The **Lin (2013)** estimator with de-meaned dummy variables for each stratum indicator interacted with treatment is also equivalent to the stratification estimator

```
wash_subset <- wash_subset %>% mutate(totchi1 = as.numeric(totchi==1),
                                      totchi2 = as.numeric(totchi==2),
                                      totchi3 = as.numeric(totchi==3))
# Adjusted
washington_strat <- lm_robust(aauw ~ anygirls*I(totchi2 - mean(totchi2)) +
                                anygirls*I(totchi3 - mean(totchi3)), wash_subset)
tidy(washington_strat) %>% filter(term == "anygirls") %>%
  select(term, estimate, std.error, p.value)
```

```
##       term estimate std.error p.value
## 1 anygirls     11.8       6.5  0.0712
```

Example: Washington (2008)

- We might still include other covariates to improve precision even if we don't think that they're part of the confounding story.
  - E.g. We might think that controlling for total number of children is enough to break the relationship between party and number of girls, but party is still really predictive of AAUW voting score.

```
# Adjusted + Party
washington_strat <- lm_lin(aauw ~ anygirls,
                           covariates = ~ as.factor(totchi)*as.factor(repub),
                           data=wash_subset)
tidy(washington_strat) %>% filter(term == "anygirls") %>%
  select(term, estimate, std.error, p.value)
```

```
##       term estimate std.error p.value
## 1 anygirls      7.5      3.77  0.0479
```

Confounding and the direction of the bias

Another Benefit to Going to Museums? You May Live Longer

Researchers in Britain found that people who go to museums, the theater and the opera were less likely to die in the study period than those who didn't.

Omitted Variable Bias

- Suppose there exists an omitted confounder U_i and ignorability holds conditional on it. Suppose we ignore it and just use a simple difference-in-means estimator.
- What's the bias for the ATT? Recall our selection-into-treatment bias formula!

$$\underbrace{E[Y_i|D_i=1] - E[Y_i|D_i=0]}_{\text{Difference-in-means}} = \underbrace{E[Y_i(1) - Y_i(0)|D_i=1]}_{\text{ATT}} + \left(\underbrace{E[Y_i(0)|D_i=1] - E[Y_i(0)|D_i=0]}_{\text{Selection-into-treatment bias}}\right)$$

Omitted Variable Bias

- Let's write the selection-into-treatment bias conditioning on U_i

$$\text{Selection Bias} = \sum_{u \in \mathcal{U}} E[Y_i(0)|D_i = 1, U_i = u] Pr(U_i = u|D_i = 1) - \sum_{u \in \mathcal{U}} E[Y_i(0)|D_i = 0, U_i = u] Pr(U_i = u|D_i = 0)$$

- Ignorability conditional on U_i

$$\text{Selection Bias} = \sum_{u \in \mathcal{U}} E[Y_i(0)|U_i = u] Pr(U_i = u|D_i = 1) - \sum_{u \in \mathcal{U}} E[Y_i(0)|U_i = u] Pr(U_i = u|D_i = 0)$$

- Combining terms

$$\text{Selection Bias} = \sum_{u \in \mathcal{U}} E[Y_i(0)|U_i = u] \times \Big(Pr(U_i = u|D_i = 1) - Pr(U_i = u|D_i = 0) \Big)$$

Omitted Variable Bias

- Two elements to selection bias. First, if treatment assignment is independent of the confounder, then the bias is 0

$$\text{Selection Bias} = \sum_{u \in \mathcal{U}} E[Y_i(0)|U_i = u] \times \Big(Pr(U_i = u|D_i = 1) - Pr(U_i = u|D_i = 0) \Big)$$

- Second, if $Y_i(0)$ is independent of U_i, we have:

$$\text{Selection Bias} = \sum_{u \in \mathcal{U}} E[Y_i(0)] \times \Big(Pr(U_i = u|D_i = 1) - Pr(U_i = u|D_i = 0) \Big)$$

$$\text{Selection Bias} = E[Y_i(0)] \times \Big(\sum_{u \in \mathcal{U}} Pr(U_i = u|D_i = 1) - \sum_{u \in \mathcal{U}} Pr(U_i = u|D_i = 0) \Big)$$

$$\text{Selection Bias} = E[Y_i(0)] \times \Big(1 - 1 \Big) = 0$$

- We get OVB/confounding when:
  1. U_i is not independent of treatment
  2. U_i is not independent of the potential outcomes

Example: Smoking and Cancer

- Back when the link between smoking and cancer was being debated, some researchers suggested that cigarettes might be a "healthy" alternative to pipe smoking
- Cochran (1968) uses this to illustrate adjustment by stratification

TABLE 1

DEATH RATES PER 1,000 PERSON-YEARS

Smoking group	Study		
	Canadian	British	U. S.
Non-smokers	20.2	11.3	13.5
Cigarettes only	20.5	14.1	13.5
Cigars, pipes	35.5	20.7	17.4

1. What's the omitted confounder?
2. What's the direction of the bias due to the omitted confounder?

Propensity scores

Propensity scores

- With low dimensional $\mathbf{X}$ we can use a stratified difference-in-means estimator. But with high-dimensional $\mathbf{X}$ we run into problems
  - The **curse of dimensionality**!
- Can we adjust for a single scalar quantity regardless of how high-dimensional $\mathbf{X}$ is?
  - Yes: The **propensity score**

$$e(X_i) = P(D_i = 1|X_i)$$

- Rosenbaum and Rubin (1983) show that conditional on the propensity score, the treatment is independent of the covariates.

$$D_i \perp\!\!\!\perp X_i | e(X_i)$$

- Therefore, conditioning on the propensity score suffices to adjust for confounding due to X

$$\{Y_i(1), Y_i(0)\} \perp\!\!\!\perp D_i | e(X_i)$$

- The propensity score is a type of **balancing score** in that conditioning on it breaks the relationship between the covariates and treatment.

Weighting estimators

- One way of adjusting for the propensity score is to use it as a **weight** on each observation.
- We weight each unit by the **inverse** propensity of it receiving its particular treatment
  - Treated units receive a weight $\frac{1}{e(X_i)}$
  - Control units receive a weight $\frac{1}{1-e(X_i)}$
- This yields the Horvitz-Thompson estimator for the ATE (with known weights)

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

- **Intuition**: Weighting constructs a "pseudo-population" where the link between treatment and the observed covariates is broken
  - Which treated units get more weight? Those whose treatment status is counter to what we would predict from the propensity score.
  - Downweight the "overrepresented" and upweight the "underrepresented"

Why weighting works

- By iterated expectations

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

- Consistency

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

- Conditional ignorability

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

Why weighting works

- Definition of the propensity score

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

- Iterated expectations

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

Normalizing the propensity score

- Horvitz-Thompson behaves especially poorly when propensity scores are close to 0 and 1

$$\hat{\tau} = \frac{1}{n} \sum_{i=1}^{n} \frac{D_i Y_i}{\widehat{e(X_i)}} - \frac{(1 - D_i) Y_i}{1 - \widehat{e(X_i)}}$$

- Common practice is to normalize the weights within the treated and control groups to sum to 1, which gives us the Hajek estimator

$$\hat{\tau} = \frac{1}{\sum_{i=1}^{n} \frac{D_i}{\widehat{e(X_i)}}} \sum_{i=1}^{n} \frac{D_i Y_i}{\widehat{e(X_i)}} - \frac{1}{\sum_{i=1}^{n} \frac{1 - D_i}{1 - \widehat{e(X_i)}}} \sum_{i=1}^{n} \frac{(1 - D_i) Y_i}{1 - \widehat{e(X_i)}}$$

- Weighted least squares will do this if we regress outcome on a binary indicator for treatment and supply the inverse propensity weights.

Inference

- We want to estimate the sampling variance $\mathbf{Var}(\hat{\tau})$ in order to do inference
  - Can't just use the regression standard error -- need to take into account uncertainty when estimating the propensity scores.
- Two options
  1. Closed form "sandwich" variance estimator $\widehat{\mathbf{Var}(\hat{\tau})}$ using M-estimation theory given the moment conditions from the propensity score model and the weighting estimator.
  2. Nonparametric bootstrap to estimate $\widehat{\mathbf{Var}(\hat{\tau})}$ via re-sampling.

Bootstrapping

- Bootstrapping is a useful tool for learning about the sampling distribution of a parameter when we don't want to use theory to figure out $\widehat{\mathbf{Var}(\hat{\tau})}$
  - Doesn't *always* work -- need a relatively well-behaved estimator that is smooth w.r.t. the data.
- **Intuition**: What drives uncertainty in $\hat{\tau}$? If we took another sample, we would calculate a different value.
  - We can't go and re-draw a sample from the population again (that's just a hypothetical).
  - But our sample is a pretty good approximation for the population...and we can resample from that.
  - So we can approximate the distribution of $\hat{\tau}$ under repeated draws from the population by taking repeated draws from the empirical distribution of the data in the sample.
- Under i.i.d. observations, we'll often use the "nonparametric" bootstrap in which we resample observations **with** replacement

The bootstrap

- The bootstrap procedure for IPTW:
  1. Draw a sample of the data by resampling observations (the rows of the data $\{Y_i, D_i, X_i\}$) **with replacement**
  2. Using the bootstrapped sample, estimate the propensity scores $e(\hat{X}_i)$.
  3. Using those propensity scores and the bootstrapped sample, compute an estimate of the average treatment effect $\hat{\tau}$. Store that estimate
  4. Repeat for many iterations.
- At the end, we have a bunch of draws that allow us to approximate the sampling distribution of $\hat{\tau}$.

Illustration: Washington (2008)

- Let's suppose we want to adjust for total number of children and party in the Washington (2008) study using IPTW.

```
# Fit a propensity score model (here, lm() works because of full saturation)
pscore_model <- lm(anygirls ~ as.factor(totchi) + as.factor(party) +
                  as.factor(totchi)*as.factor(party), data=wash_subset)

# Predict the propensity score
wash_subset$e <- predict(pscore_model, type="response")

# Generate the weights
wash_subset$iptw_weight <- 1/wash_subset$e
wash_subset$iptw_weight[wash_subset$anygirls == 0] <- 1/(1-wash_subset$e[wash_subset$anygirls =
```

Illustration: Washington (2008)

- Histogram of the weights

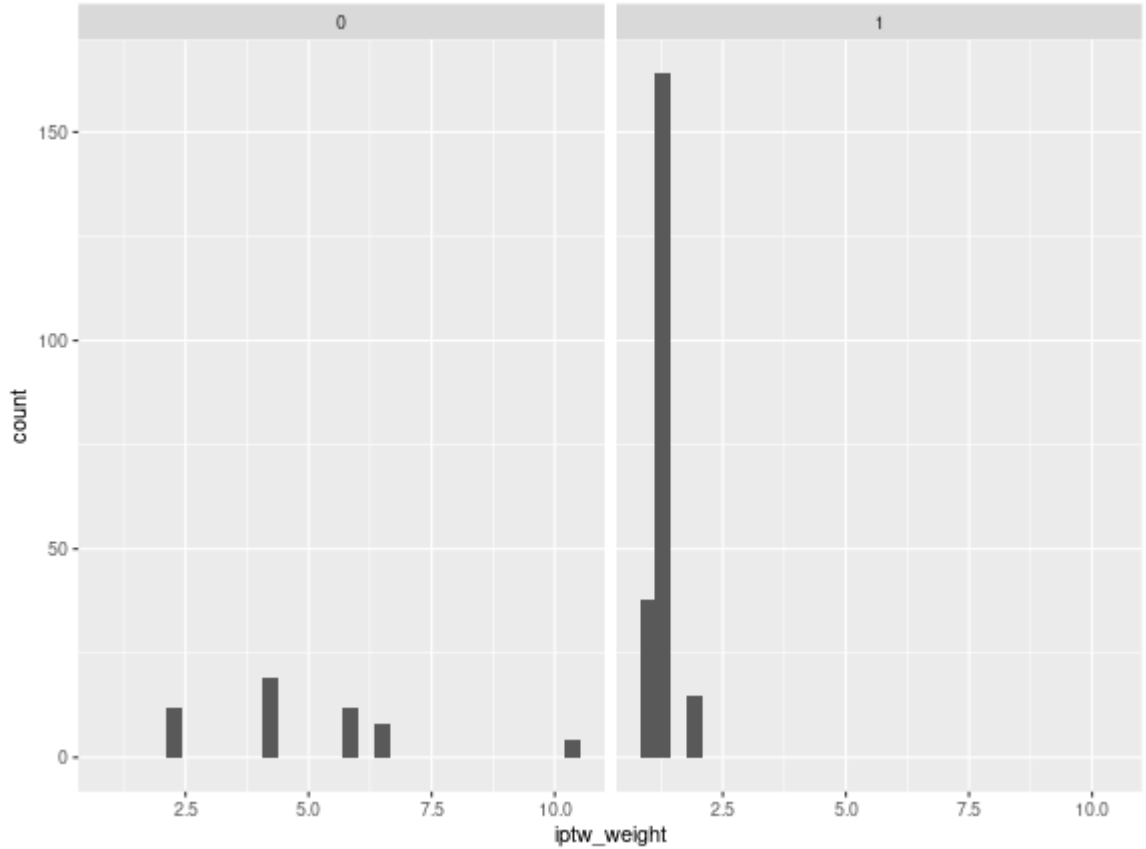


Illustration: Washington (2008)

- Calculate IPTW estimate of the ATE

```
# Weighted regression
iptw_model <- lm_robust(aauw ~ anygirls, data=wash_subset, weights=iptw_weight)
coef(iptw_model)[2] # Ignore the SE
```

```
## anygirls
##      7.5
```

```
# Weighted regression gives the Hajek (normalized weights) estimator
iptw_est <- weighted.mean(wash_subset$aauw[wash_subset$anygirls == 1], wash_subset$iptw_weight[
  weighted.mean(wash_subset$aauw[wash_subset$anygirls == 0], wash_subset$iptw_weight[wash_subse
iptw_est
```

```
## [1] 7.5
```

Illustration: Washington (2008)

- Bootstrap

```
set.seed(60638)
nIter <- 1000
boot_iptw <- rep(NA,nIter)
for (i in 1:nIter){
  wash_subset_boot <- wash_subset[sample(1:nrow(wash_subset), size = nrow(wash_subset), replace
   # Fit a propensity score model
  pscore_model_boot <- lm(anygirls ~ as.factor(totchi) + as.factor(party) +
                as.factor(totchi)*as.factor(party), data=wash_subset_boot)

  # Predict the propensity score
  wash_subset_boot$e_boot <- predict(pscore_model_boot, type="response")

  # Generate the weights
  wash_subset_boot$iptw_weight_boot <-1/wash_subset_boot$e_boot
  wash_subset_boot$iptw_weight_boot[wash_subset_boot$anygirls == 0] <- 1/(1-wash_subset_boot$e_

  # Fit a model
  iptw_model_boot <- lm_robust(aauw ~ anygirls, data=wash_subset_boot, weights=iptw_weight_boot
  boot_iptw[i] <- coef(iptw_model_boot)[2]
}
```

Illustration: Washington (2008)

- Bootstrap SE and percentile intervals

```
# Estimated standard error
boot_se <- sd(boot_iptw)
boot_se
```

```
## [1] 3.75
```

```
# Asymptotic CI
c(iptw_est - qnorm(.975)*boot_se, iptw_est + qnorm(.975)*boot_se)
```

```
## [1]  0.136 14.855
```

```
# Percentile interval
quantile(boot_iptw, c(.025, .975))
```

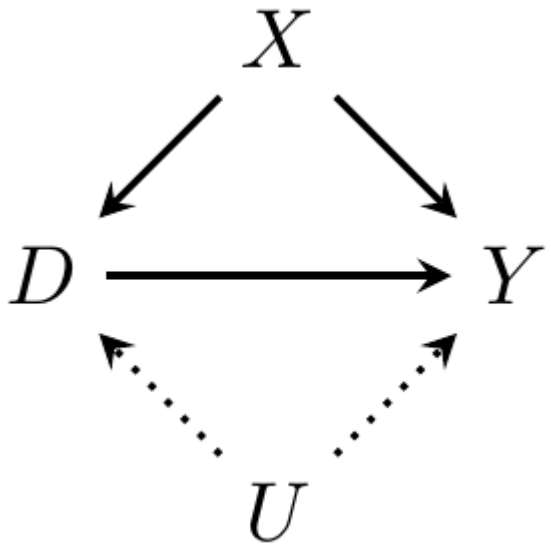
```
##   2.5%  97.5%
##  0.644 15.712
```

Directed Acyclic Graphs

What to condition on?

- The main challenge in designing an observational study is figuring out what $\mathbf{X}$ is.
  - What do you need to control for in order to make $\{Y_i(1), Y_i(0)\} \perp\!\!\!\perp D_i | \mathbf{X}_i$ plausible?
- One useful tool: **graphical models**
  - Represent causal relations in terms of vertices/nodes (V) and edges (E) on a graph G.
  - **Vertices** represent variables
  - Directed **edges** denote non-zero causal effects.
- We will use DAGs to reason about (conditional) dependencies between variables
  - Slight conceptual/terminology differences from potential outcomes, but fundamentals of graphical approaches are the same
  - See: Imbens (2020; JEL) for comments on the differences. Richardson and Robins (2013) for a unification.

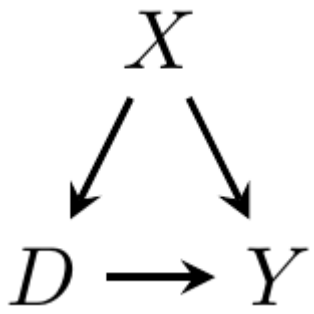
Directed Acyclic Graphs



1. **Directed**: Edges have arrows
2. **Acyclic**: A node can't be its own descendant
3. **Graph**: Comprised of nodes and edges

- DAGs encode causal assumptions
  - Absence of an edge - assume **no** causal effect
  - Direction of the arrow - direction of effect

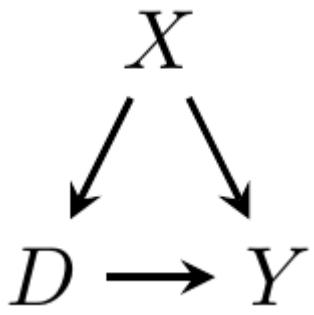
Directed Acyclic Graphs



- Chain: $X \to D \to Y$
- Fork: $D \leftarrow X \to Y$
- Collider: $X \to Y \leftarrow D$

- A **parent** node is a direct cause of a **child** node.
- An **ancestor** node is a direct or indirect cause of a **descendent** node.
- **Path**: A sequence of nodes connected by edges (either direction!)
  - Causal path: All arrows are pointed in the same direction
  - Non-causal path: Some arrows go in the opposite direction

Directed Acyclic Graphs



$$Y = f_Y(D, X, \epsilon_Y)$$
$$D = f_D(X, \epsilon_D)$$

- DAGs are a way of representing a particular **nonparametric** structural equation model
- The DAG also represents a factorization of the joint distribution if it's compatible with the DAG.

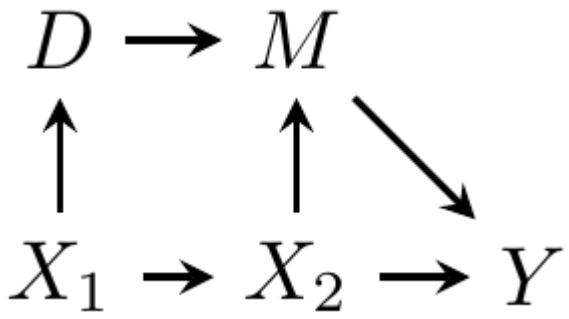
$$P(X_1, X_2, \ldots, X_K) = \prod_{k=1}^{K} P(X_K | \text{parents}(X_K))$$

- This lets us read (conditional) independence relationships directly from the DAG.

D-separation

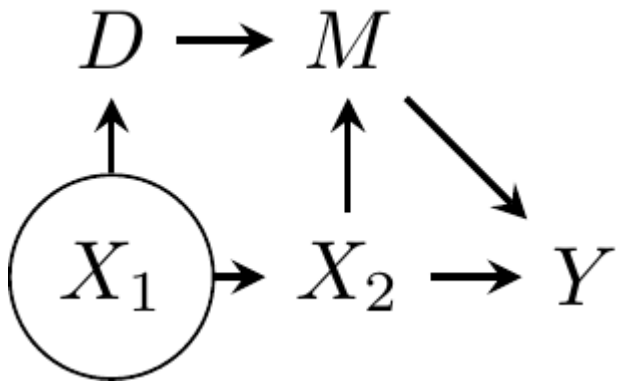
- How do we know if two nodes A and B are conditionally dependent or independent conditional on a third set of nodes C?
- We can read this right off of the DAG using the "*d*-separation" criterion.
  - If A and B are *d*-separated given C, then $A \perp\!\!\!\perp B | C$
  - Otherwise they are *d*-connected
- When are two nodes d-separated?
  - When there are no **unblocked** paths between them given C.
- When are paths unblocked?
  - When there is a chain or fork that is *not* conditioned on in C.
  - When there is a collider (or descendent of a collider) that *is* conditioned on in C.
- Conditioning on causal chains or common causes (forks) blocks a path.
- Conditioning on common effects (colliders) unblocks a path.

D-separation



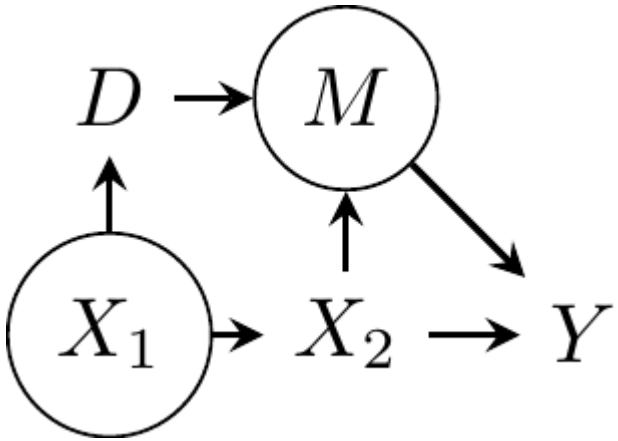
- Are X_2 and D d-separated?
- What are the paths?
  - $D \rightarrow M \rightarrow Y \leftarrow X_2$ (Blocked by collider at Y)
  - $D \rightarrow M \leftarrow X_2$ (Blocked by collider at M)
  - $D \leftarrow X_1 \rightarrow X_2$ (Unblocked)

D-separation



- Are X_2 and D d-separated conditional on X_1?
- Three paths
  - $D \to M \to Y \leftarrow X_2$ (Blocked by collider at Y)
  - $D \to M \leftarrow X_2$ (Blocked by collider at M)
  - $D \leftarrow X_1 \to X_2$ (Blocked by conditioning on X_1)

D-separation



- Are X_2 and D d-separated conditional on X_1 and M?
- Three paths
  - $D \rightarrow M \rightarrow Y \leftarrow X_2$ (Blocked by collider at Y)
  - $D \rightarrow M \leftarrow X_2$ (Unblocked by a conditioned collider at M)
  - $D \leftarrow X_1 \rightarrow X_2$ (Blocked by conditioning on X_1)

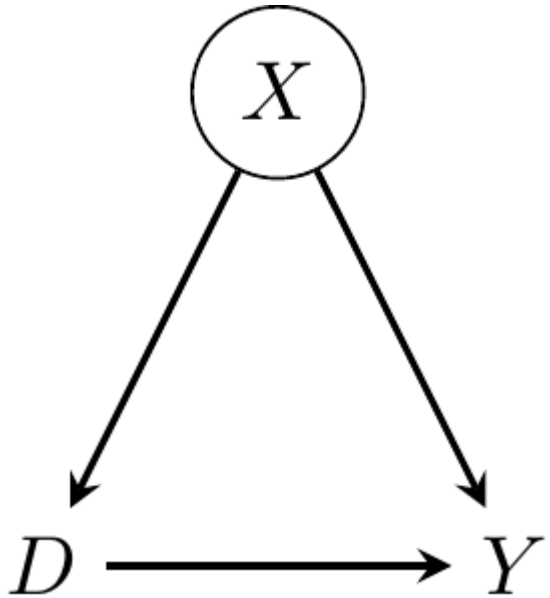
Defining causal effects

- The graph lets us read conditional probabilities on the observed quantities $P(Y|D, X)$.
- However, we don't just want $P(Y|D)$, we want a *counterfactual*. Within Pearl's graph framework, an intervention is represented by the "do" operator applied to a node.
  - $\text{do}(X = x)$ denotes an intervention that sets X to a particular level x
  - Represented in a **counterfactual graph** that removes all arrows into D.
- We want to learn about the post-intervention distribution $P(Y|\text{do}(X = x))$, but we only have the observed distribution/DAG.
  - We want to **identify** the counterfactual distribution (or a functional thereof) from the observed distribution.
  - How do we do this: Pearl's "do-calculus"
- Similar idea to our identification problem when defining effects in terms of potential outcomes.
  - We observe $Y|D, X$ and want to identify $Y(d)$
  - In fact, $Y_i(d)$ and $Y|do(D = d)$ are essentially representing the same counterfactual concept.

Adjustment criterion

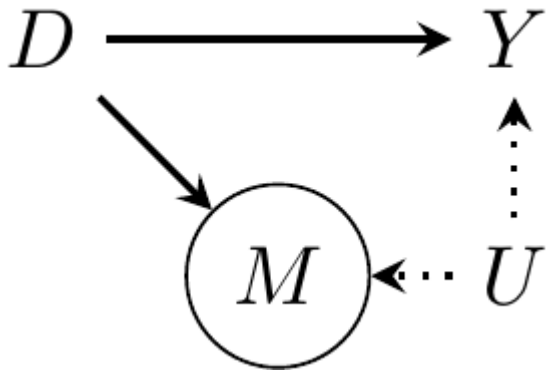
- If we condition on covariates X can we non-parametrically identify the effect of D on Y?
  - Yes, if we block all **non-causal paths** from Y to D.
- **Adjustment criterion** *(Shpitser, VanderWeele, and Robins, 2012)*
  - All non-causal paths from D to Y are blocked by the set X
  - No element in X is a node or a descendant of a node on a causal path from D to Y.
- Intuition:
  - Block non-causal paths from Y to D
  - Don't **open up** non-causal paths from Y to D
  - Don't control for variables along the causal path from D to Y.
- If the adjustment criterion holds then, the observed distributions identify the counterfactual distribution
  - $P(Y|\text{do}(d)) = \sum_z P(Y|d,z)P(z)$
  - **Conditional ignorability!**

Good controls



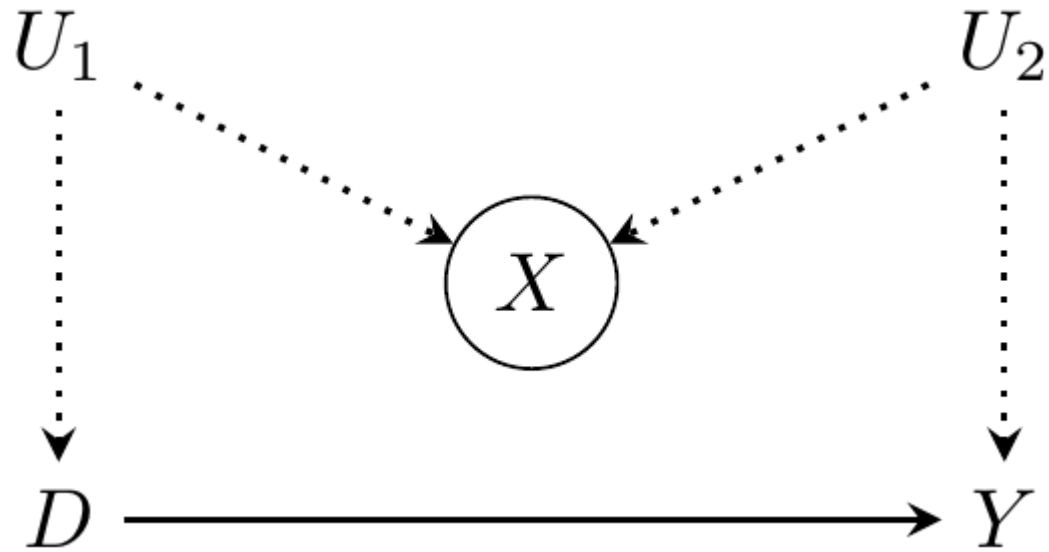
- One non-causal path from D to Y: $D \leftarrow X \rightarrow Y$.
- Conditioning on X blocks that path

Bad controls



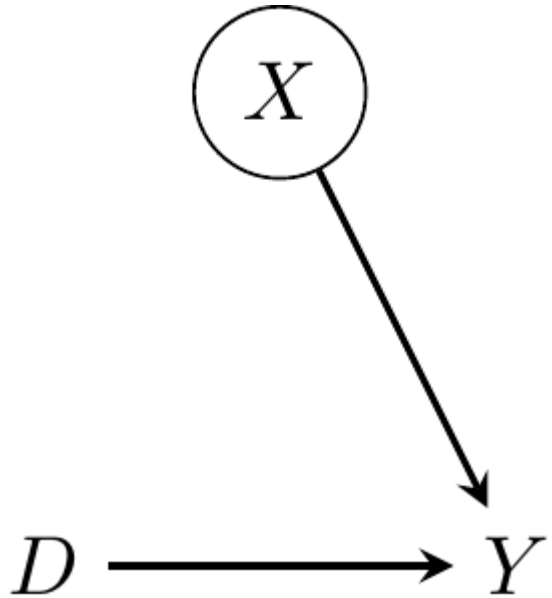
- No non-causal paths between D and Y w/o conditioning
- But conditioning on M opens up a non-causal path between D and Y
  - $D \rightarrow Z \leftarrow U \rightarrow Y$

Bad controls



- Even though X is not causally related to D or Y ("irrelevant control"), conditioning on it still induces bias if there are common causes of X and treatment and X and outcome
- "M-bias"

Neutral Controls



- X is not a confounder but is predictive of Y
- Might improve precision!

Are graphs and P.O. incompatible?

> So, what is it about epidemiologists that drives them to seek the light of new tools, while economists (at least those in Imbens's camp) seek comfort in partial blindness, while missing out on the causal revolution? Can economists do in their heads what epidemiologists observe in their graphs? Can they, for instance, identify the testable implications of their own assumptions? Can they decide whether the IV assumptions (i.e., exogeneity and exclusion) are satisfied in their own models of reality? Of course they can't; such decisions are intractable to the graph-less mind. (I have challenged them repeatedly to these tasks, to the sound of a pin-drop silence)

Pearl (2014) (http://causality.cs.ucla.edu/blog/index.php/2014/10/27/are-economists-smarter-than-epidemiologists-comments-on-imbenss-recent-paper/)

SWIGs - a Unification

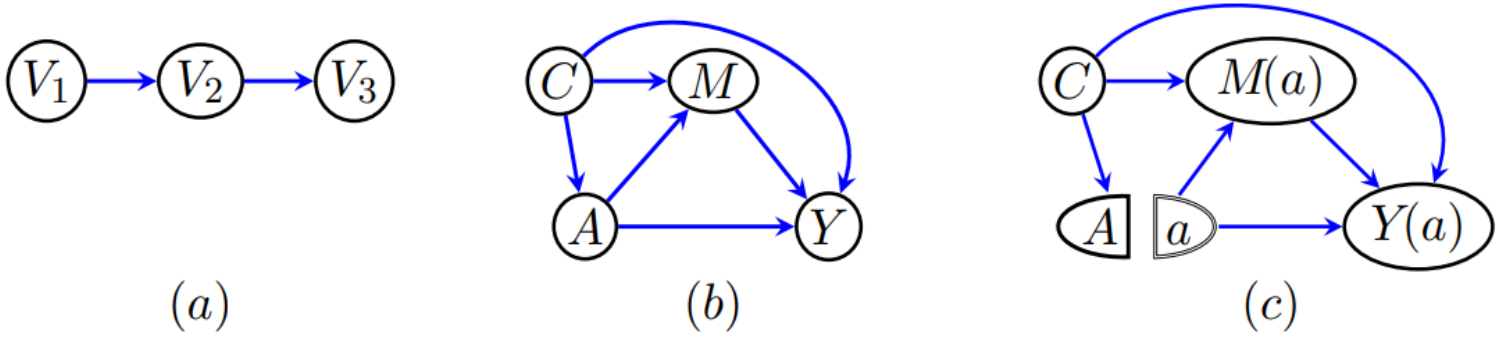


Figure 1: (a) A DAG representing a simple NPSEM. (b) A simple causal DAG $\mathcal{G}$, with a treatment A, an outcome Y, a vector C of baseline variables, and a mediator M. (c) A SWIG $\mathcal{G}(a)$ derived from (a) corresponding to the world where A is intervened on to value a.

Shpitser, Richardson and Robins (2021)

DAG Practice