Week 10: Causality with Measured Confounding

Brandon Stewart

Princeton

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1These slides are heavily influenced by Matt Blackwell, Jens Hainmueller, Erin Hartman, Kosuke Imai and Gary King.
Where We’ve Been and Where We’re Going...

- Last Week
  - intro to causal inference

- This Week
  - Monday:
    - experimental Ideal
    - identification with measured confounding
  - Wednesday:
    - regression estimation

- Next Week
  - identification with unmeasured confounding
  - instrumental variables

- Long Run
  - probability $\rightarrow$ inference $\rightarrow$ regression $\rightarrow$ causal inference

Questions?
1. The Experimental Ideal
2. Assumption of No Unmeasured Confounding
3. Estimation Under No Unmeasured Confounding
4. Regression Estimators
5. Regression and Causality
6. Regression Under Heterogeneous Effects
7. Fun with Visualization, Replication and the NYT
1. The Experimental Ideal
2. Assumption of No Unmeasured Confounding
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7. Fun with Visualization, Replication and the NYT
Lancet 2001: negative correlation between coronary heart disease mortality and level of vitamin C in bloodstream (controlling for age, gender, blood pressure, diabetes, and smoking)
Lancet 2002: no effect of vitamin C on mortality in controlled placebo trial (controlling for nothing)
Lancet 2003: comparing among individuals with the same age, gender, blood pressure, diabetes, and smoking, those with higher vitamin C levels have lower levels of obesity, lower levels of alcohol consumption, are less likely to grow up in working class, etc.
Why So Much Variation?
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Confounders

\[ X \rightarrow T \rightarrow Y \]
Observational Studies and Experimental Ideal

- Randomization forms gold standard for causal inference, because it balances observed and unobserved confounders.
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Better than hoping: design observational study to approximate an experiment.

“The planner of an observational study should always ask himself: How would the study be conducted if it were possible to do it by controlled experimentation” (Cochran 1965)
Angrist and Pishke’s Frequently Asked Questions
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- What is the experiment that could ideally be used to capture the causal effect of interest?
- What is your identification strategy?
- What is your mode of statistical inference?
An experiment is a study where assignment to treatment is controlled by the researcher.
Experiment review

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  - $p_i = P[D_i = 1]$ be the probability of treatment assignment probability.
  - $p_i$ is controlled and known by researcher in an experiment.

A randomized experiment is an experiment with the following properties:

1. Positivity: assignment is probabilistic: $0 < p_i < 1$
   - No deterministic assignment.
2. Unconfoundedness: $P[D_i = 1 | Y(1), Y(0)] = P[D_i = 1]$
   - Treatment assignment does not depend on any potential outcomes.
   - Sometimes written as $D_i \perp \perp (Y(1), Y(0))$
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     ▶ Sometimes written as \( D_i \perp (Y(1), Y(0)) \)
Why do Experiments Help?

Remember selection bias?

\[
E[Y_i|D_i = 1] - E[Y_i|D_i = 0] = E[Y_i(1)|D_i = 1] - E[Y_i(0)|D_i = 0] \\
= E[Y_i(1)|D_i = 1] - E[Y_i(0)|D_i = 1] + E[Y_i(0)|D_i = 1] - E[Y_i(0)|D_i = 0] \\
= E[Y_i(1) - Y_i(0)|D_i = 1] + E[Y_i(0)|D_i = 1] - E[Y_i(0)|D_i = 0]
\]

Average Treatment Effect on Treated + selection bias
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= \underbrace{E[Y_i(1) - Y_i(0)|D_i = 1]}_{\text{Average Treatment Effect on Treated}} + \underbrace{E[Y_i(0)|D_i = 1] - E[Y_i(0)|D_i = 0]}_{\text{selection bias}}
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When all goes well, an experiment eliminates selection bias.
Observational studies

- Many different sets of identification assumptions that we’ll cover.

- No guarantee that the treatment and control groups are comparable.

- Positivity (Common Support): assignment is probabilistic:
  \[ P[D_i = 1 | X, Y(1), Y(0)] < 1 \]

- No unmeasured confounding:
  \[ P[D_i = 1 | X, Y(1), Y(0)] = P[D_i = 1 | X] \]

- For some observed \( X \)

- Also called: unconfoundedness, ignorability, selection on observables, no omitted variables, exogenous, conditionally exchangeable, etc.
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Rubin (2008) argues that we should still “design” our observational studies:

- Pick the ideal experiment to this observational study.
- Hide the outcome data.
- Try to estimate the randomization procedure.
- Analyze this as an experiment with this estimated procedure.

Tries to minimize “snooping” by picking the best modeling strategy before seeing the outcome.
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Discrete covariates

• Suppose that we knew that $D_i$ was unconfounded within levels of a binary $X_i$. 

$$E_{X} \left\{ E\left[ Y_i | D_i = 1, X_i \right] - E\left[ Y_i | D_i = 0, X_i \right] \right\} = \left( E\left[ Y_i | D_i = 1, X_i = 1 \right] - E\left[ Y_i | D_i = 0, X_i = 1 \right] \right) \cdot \text{diff-in-means for } X_i = 1 \cdot P\left[ X_i = 1 \right] + \left( E\left[ Y_i | D_i = 1, X_i = 0 \right] - E\left[ Y_i | D_i = 0, X_i = 0 \right] \right) \cdot \text{diff-in-means for } X_i = 0 \cdot P\left[ X_i = 0 \right]$$

Never used our knowledge of the randomization for this quantity.
Suppose that we knew that $D_i$ was unconfounded within levels of a binary $X_i$.

Then we could always estimate the causal effect using iterated expectations as in a stratified randomized experiment:

$$E\{E[Y_i|D_i=1, X_i] - E[Y_i|D_i=0, X_i]\} = \left( E[Y_i|D_i=1, X_i=1] - E[Y_i|D_i=0, X_i=1] \right) \cdot \frac{P[X_i=1]}{\text{share of } X_i=1} + \left( E[Y_i|D_i=1, X_i=0] - E[Y_i|D_i=0, X_i=0] \right) \cdot \frac{P[X_i=0]}{\text{share of } X_i=0}.$$
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\text{diff-in-means for } X_i=1 \\
+ \left( \mathbb{E}[Y_i|D_i = 1, X_i = 0] - \mathbb{E}[Y_i|D_i = 0, X_i = 0] \right) \mathbb{P}[X_i = 0] \\
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Stratification Example: Smoking and Mortality (Cochran, 1968)

**Table 1**

Death Rates per 1,000 Person-Years

<table>
<thead>
<tr>
<th>Smoking group</th>
<th>Canada</th>
<th>U.K.</th>
<th>U.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-smokers</td>
<td>20.2</td>
<td>11.3</td>
<td>13.5</td>
</tr>
<tr>
<td>Cigarettes</td>
<td>20.5</td>
<td>14.1</td>
<td>13.5</td>
</tr>
<tr>
<td>Cigars/pipes</td>
<td>35.5</td>
<td>20.7</td>
<td>17.4</td>
</tr>
</tbody>
</table>
Stratification Example: Smoking and Mortality (Cochran, 1968)

Table 2
Mean Ages, Years

<table>
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<td>54.9</td>
<td>49.1</td>
<td>57.0</td>
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<tr>
<td>Cigarettes</td>
<td>50.5</td>
<td>49.8</td>
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Stratification

To control for differences in age, we would like to compare different smoking-habit groups with the same age distribution.
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- for each country, divide each group into different age subgroups
- calculate death rates within age subgroups
- average within age subgroup death rates using fixed weights (e.g. number of cigarette smokers)
Stratification: Example

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Death Rates Pipe Smokers</th>
<th># Pipe-Smokers</th>
<th># Non-Smokers</th>
</tr>
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<tbody>
<tr>
<td>Age 20 - 50</td>
<td>15</td>
<td>11</td>
<td>29</td>
</tr>
<tr>
<td>Age 50 - 70</td>
<td>35</td>
<td>13</td>
<td>9</td>
</tr>
<tr>
<td>Age + 70</td>
<td>50</td>
<td>16</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
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What is the average death rate for Pipe Smokers?
### Stratification: Example

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What is the average death rate for Pipe Smokers?

\[
15 \cdot \left(\frac{11}{40}\right) + 35 \cdot \left(\frac{13}{40}\right) + 50 \cdot \left(\frac{16}{40}\right) = 35.5
\]
<table>
<thead>
<tr>
<th></th>
<th>Death Rates Pipe Smokers</th>
<th># Pipe-Smokers</th>
<th># Non-Smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age 20 - 50</td>
<td>15</td>
<td>11</td>
<td>29</td>
</tr>
<tr>
<td>Age 50 - 70</td>
<td>35</td>
<td>13</td>
<td>9</td>
</tr>
<tr>
<td>Age + 70</td>
<td>50</td>
<td>16</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>40</td>
<td>40</td>
</tr>
</tbody>
</table>

What is the average death rate for Pipe Smokers if they had same age distribution as Non-Smokers?
### Stratification: Example

<table>
<thead>
<tr>
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<th># Pipe-Smokers</th>
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<td>40</td>
<td>40</td>
</tr>
</tbody>
</table>

What is the average death rate for Pipe Smokers if they had same age distribution as Non-Smokers?

\[
15 \cdot \left(\frac{29}{40}\right) + 35 \cdot \left(\frac{9}{40}\right) + 50 \cdot \left(\frac{2}{40}\right) = 21.2
\]
### Table 3

**Adjusted Death Rates using 3 Age groups**

<table>
<thead>
<tr>
<th>Smoking group</th>
<th>Canada</th>
<th>U.K.</th>
<th>U.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-smokers</td>
<td>20.2</td>
<td>11.3</td>
<td>13.5</td>
</tr>
<tr>
<td>Cigarettes</td>
<td>28.3</td>
<td>12.8</td>
<td>17.7</td>
</tr>
<tr>
<td>Cigars/pipes</td>
<td>21.2</td>
<td>12.0</td>
<td>14.2</td>
</tr>
</tbody>
</table>

**Smoking and Mortality (Cochran, 1968)**
Continuous covariates

- So, great, we can stratify. Why not do this all the time?
Continuous covariates

- So, great, we can stratify. Why not do this all the time?
- What if $X_i =$ income for unit $i$?

Each unit has its own value of $X_i$: $54,134, $123,043, $23,842$.

If $X_i = 54134$ is unique, will only observe 1 of these:

$E[Y_i | D_i = 1, X_i = 54134] - E[Y_i | D_i = 0, X_i = 54134]$.

$\Rightarrow$ cannot stratify to each unique value of $X_i$.

Practically, this is massively important: almost always have data with unique values.

One option is to discretize as we discussed with age, we will discuss more later this week!
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  - If $X_i = 54134$ is unique, will only observe 1 of these:
    \[
    \mathbb{E}[Y_i|D_i = 1, X_i = 54134] - \mathbb{E}[Y_i|D_i = 0, X_i = 54134]
    \]
  - $\leadsto$ cannot stratify to each unique value of $X_i$:
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Identification Under Selection on Observables

Identification Assumption
1. \((Y_1, Y_0) \perp \perp D|X\) (selection on observables)
2. \(0 < \Pr(D = 1|X) < 1\) with probability one (common support)

Identification Result

Given selection on observables we have

\[
E[Y_1 - Y_0|X] = E[Y_1 - Y_0|X, D = 1]
= E[Y|X, D = 1] - E[Y|X, D = 0]
\]

Therefore, under the common support condition:

\[
\tau_{ATE} = E[Y_1 - Y_0] = \int E[Y_1 - Y_0|X] \, dP(X)
= \int (E[Y|X, D = 1] - E[Y|X, D = 0]) \, dP(X)
\]
Identification Under Selection on Observables

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Identification Result

Similarly,

\[
\tau_{ATT} = \mathbb{E}[Y_1 - Y_0|D = 1] = \int (\mathbb{E}[Y|X,D = 1] - \mathbb{E}[Y|X,D = 0]) \, dP(X|D = 1)
\]

To identify \(\tau_{ATT}\) the selection on observables and common support conditions can be relaxed to:

- \(Y_0 \perp\!\!\!\!\!\!\!\!\!\!\!\perp D|X\) (SOO for Controls)
- \(\Pr(D = 1|X) < 1\) (Weak Overlap)
### Identification Under Selection on Observables

<table>
<thead>
<tr>
<th>unit</th>
<th>Potential Outcome under Treatment</th>
<th>Potential Outcome under Control</th>
<th>( D_i )</th>
<th>( X_i )</th>
</tr>
</thead>
<tbody>
<tr>
<td>i</td>
<td>( Y_{1i} )</td>
<td>( Y_{0i} )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>( \mathbb{E}[Y_1</td>
<td>X = 0, D = 1] )</td>
<td>( \mathbb{E}[Y_0</td>
<td>X = 0, D = 1] )</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>( \mathbb{E}[Y_1</td>
<td>X = 0, D = 0] )</td>
<td>( \mathbb{E}[Y_0</td>
<td>X = 0, D = 0] )</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>( \mathbb{E}[Y_1</td>
<td>X = 1, D = 1] )</td>
<td>( \mathbb{E}[Y_0</td>
<td>X = 1, D = 1] )</td>
</tr>
<tr>
<td>6</td>
<td></td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>( \mathbb{E}[Y_1</td>
<td>X = 1, D = 0] )</td>
<td>( \mathbb{E}[Y_0</td>
<td>X = 1, D = 0] )</td>
</tr>
<tr>
<td>8</td>
<td></td>
<td></td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>
Identification Under Selection on Observables

<table>
<thead>
<tr>
<th>unit</th>
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<th>$X_i$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, 2</td>
<td>$\mathbb{E}[Y_1</td>
<td>X = 0, D = 1]$</td>
<td>$\mathbb{E}[Y_0</td>
<td>X = 0, D = 1] = \mathbb{E}[Y_0</td>
</tr>
<tr>
<td>3, 4</td>
<td>$\mathbb{E}[Y_1</td>
<td>X = 0, D = 0]$</td>
<td>$\mathbb{E}[Y_0</td>
<td>X = 0, D = 0]$</td>
</tr>
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<tr>
<td>7, 8</td>
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</table>

$(Y_1, Y_0) \perp D|X$ implies that we conditioned on all confounders. The treatment is randomly assigned within each stratum of $X$:  
\[
\mathbb{E}[Y_0|X = 0, D = 1] = \mathbb{E}[Y_0|X = 0, D = 0] \quad \text{and} \\
\mathbb{E}[Y_0|X = 1, D = 1] = \mathbb{E}[Y_0|X = 1, D = 0]
\]
Identification Under Selection on Observables

<table>
<thead>
<tr>
<th>unit</th>
<th>Potential Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>under Treatment</td>
</tr>
<tr>
<td>i</td>
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</tr>
<tr>
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<td>( Y_{0i} )</td>
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\((Y_{1}, Y_{0}) \perp D | X\) also implies

\[ \mathbb{E}[Y_{1} | X = 0, D = 1] = \mathbb{E}[Y_{1} | X = 0, D = 0] \text{ and} \]

\[ \mathbb{E}[Y_{1} | X = 1, D = 1] = \mathbb{E}[Y_{1} | X = 1, D = 0] \]
The Experimental Ideal

Assumption of No Unmeasured Confounding

Estimation Under No Unmeasured Confounding

Regression Estimators

Regression and Causality

Regression Under Heterogeneous Effects

Fun with Visualization, Replication and the NYT
The Experimental Ideal

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Regression Estimators

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Fun with Visualization, Replication and the NYT
What is confounding?

- **Confounding** is the bias caused by common causes of the treatment and outcome.

Leads to "spurious correlation." In observational studies, the goal is to avoid confounding inherent in the data.

Pervasive in the social sciences:

- effect of income on voting (confounding: age)
- effect of job training program on employment (confounding: motivation)
- effect of political institutions on economic development (confounding: previous economic development)

No unmeasured confounding assumes that we've measured all sources of confounding.
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- No unmeasured confounding assumes that we’ve measured all sources of confounding.
How can we determine if no unmeasured confounding holds if we didn’t assign the treatment?

- What covariates do we need to condition on?
- What covariates do we need to include in our regressions?

One way, from the assumption itself:

\[ P[D_{i} = 1 | X, Y(1), Y(0)] = P[D_{i} = 1 | X] \]

Include covariates such that, conditional on them, the treatment assignment does not depend on the potential outcomes.

Another way: use DAGs and look at back-door paths.
Big problem

- How can we determine if no unmeasured confounding holds if we didn’t assign the treatment?
- Put differently:
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Backdoor paths and blocking paths

- **Backdoor path**: is a non-causal path from $D$ to $Y$. 
Backdoor paths and blocking paths

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  - Would remain if we removed any arrows pointing out of $D$. 

---

Stewart (Princeton)  
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November 26 and 28, 2018  
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Backdoor paths and blocking paths

- **Backdoor path**: is a non-causal path from $D$ to $Y$.
  - Would remain if we removed any arrows pointing out of $D$.
- Backdoor paths between $D$ and $Y \sim \sim$ common causes of $D$ and $Y$:

![Diagram showing a backdoor path](image)

- $X$ is a common cause for the treatment and the outcome.
Backdoor paths and blocking paths

- **Backdoor path**: is a non-causal path from $D$ to $Y$.
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- Backdoor paths between $D$ and $Y \rightsquigarrow$ common causes of $D$ and $Y$:

\[
\begin{array}{c}
X \\
\bigtriangleup \\
D \rightarrow Y
\end{array}
\]

- Here there is a backdoor path $D \leftarrow X \rightarrow Y$, where $X$ is a common cause for the treatment and the outcome.
Other types of confounding

\[ U \rightarrow X \]
\[ \downarrow \]
\[ D \rightarrow Y \]

- \( D \) is enrolling in a job training program.
Other types of confounding

- $D$ is enrolling in a job training program.
- $Y$ is getting a job.
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\[ U \rightarrow X \]
\[
\downarrow \quad \downarrow
\]
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- $X$ is number of job applications sent out.
- Big assumption here: no arrow from $U$ to $Y$.

```
U → X
  ↓  ↓
  ↓  ↓
D → Y
```
Other types of confounding

- $D$ is exercise.
- $Y$ is having a disease.
- $U$ is lifestyle.
- $X$ is smoking.
- Big assumption here: no arrow from $U$ to $Y$. 

Diagram:

```
U → X
  ↓   ↓
D → Y
```
What’s the problem with backdoor paths?

A path is blocked if:

- we control for or stratify a non-collider on that path OR
- we do not control for a collider.

In the DAG here, if we condition on $X$, then the backdoor path is unblocked.
What’s the problem with backdoor paths?

A path is **blocked** if:

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- $\downarrow$
- $\downarrow$
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Unblocked backdoor paths $\leadsto$ confounding.
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Conditioning on the posttreatment covariates opens the non-causal path.
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- ↝ selection bias.
Not all backdoor paths

Conditioning on the posttreatment covariates opens the non-causal path.

- selection bias.
Don’t condition on post-treatment variables
Don’t condition on post-treatment variables

Every time you do, a puppy cries.
M-bias

Not all backdoor paths induce confounding.

- Sometimes called M-bias.

Controversial because of differing views on what to control for:
- Rubin thinks that M-bias is a “mathematical curiosity” and we should control for all pretreatment variables.
- Pearl and others think M-bias is a real threat.
- See the Elwert and Winship piece for more!
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- Not all backdoor paths induce confounding.
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M-bias

- Not all backdoor paths induce confounding.
- This backdoor path is blocked by the collider $X$ that we don’t control for.
- If we control for $X \sim \rightarrow$ opens the path and induces confounding.
M-bias

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  > See the Elwert and Winship piece for more!
Can we use a DAG to evaluate no unmeasured confounders?
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Pearl answered yes, with the backdoor criterion, which states that the effect of $D$ on $Y$ is identified if:

1. No backdoor paths from $D$ to $Y$.
   OR
2. Measured covariates are sufficient to block all backdoor paths from $D$ to $Y$.

First is really only valid for randomized experiments.

The backdoor criterion is fairly powerful. Tells us:

▶ if there is confounding given this DAG,
▶ if it is possible to remove the confounding, and
▶ what variables to condition on to eliminate the confounding.
Can we use a DAG to evaluate no unmeasured confounders?

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Example: Sufficient Conditioning Sets

Remove arrows out of $X$. 
Recall that paths are blocked by “unconditioned colliders” or conditioned non-colliders.
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Implications (via Vanderweele and Shpitser 2011)

1. Choose all pre-treatment covariates (would condition on $C_2$ inducing M-bias).

2. Choose all covariates which directly cause the treatment and the outcome (would leave open a backdoor path $A \leftarrow C_3 \leftarrow U_3 \rightarrow Y$).

\[\begin{align*}
U_3 & \rightarrow C_3 & \rightarrow A & \rightarrow Y \\
\quad & \quad & \quad & \\
U_1 & \quad & C_2 & \quad \\
\quad & \quad & \quad & \\
U_2 & \quad & \quad & \\
\end{align*}\]
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Two common criteria fail here:

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No unmeasured confounders is not testable

- No unmeasured confounding places no restrictions on the observed data.

\[
\begin{align*}
\left(Y_i(0) \mid D_i = 1, X_i\right) \overset{d}{=} \left(Y_i(0) \mid D_i = 0, X_i\right) \\
\text{unobserved} & \quad \text{observed}
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Assessing no unmeasured confounders

Can do “placebo” tests, where $D_i$ cannot have an effect (lagged outcomes, etc)

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<th>Dep. var.</th>
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<th>Placebo specifications</th>
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<tbody>
<tr>
<td></td>
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<tr>
<td>Availability of Fox News via cable in 2000</td>
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</tr>
<tr>
<td></td>
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<td>−0.0001</td>
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<tr>
<td></td>
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**TABLE VI**

THE FOX NEWS EFFECT: INTERACTIONS AND PLACEBO SPECIFICATIONS

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- Availability in 2000/2003 can’t affect past vote shares.

Unconfoundedness could still be violated even if you pass this test!
Alternatives to no unmeasured confounding

- Without explicit randomization, we need some way of identifying causal effects.

   - No unmeasured confounders \(\approx\) randomized experiment.
   - Identification results very similar to experiments.
   - With unmeasured confounding are we doomed? Maybe not!
   - Other approaches rely on finding plausibly exogenous variation in assignment of \(D_i\):
     - Instrumental variables (randomization + exclusion restriction)
     - Over-time variation (diff-in-diff, fixed effects)
     - Arbitrary thresholds for treatment assignment (RDD)
   - All discussed in the next couple of weeks!
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Where We’ve Been and Where We’re Going...

- Last Week
  - intro to causal inference

- This Week
  - Monday:
    - experimental Ideal
    - identification with measured confounding
  - Wednesday:
    - regression estimation

- Next Week
  - identification with unmeasured confounding
  - instrumental variables

- Long Run
  - probability → inference → regression → causal inference

Questions?
The Experimental Ideal

Assumption of No Unmeasured Confounding

Estimation Under No Unmeasured Confounding

Regression Estimators

Regression and Causality

Regression Under Heterogeneous Effects

Fun with Visualization, Replication and the NYT
1. The Experimental Ideal

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3. Estimation Under No Unmeasured Confounding

4. Regression Estimators

5. Regression and Causality

6. Regression Under Heterogeneous Effects

7. Fun with Visualization, Replication and the NYT
Identification vs. Estimation

An approximately ordered causal workflow:

1) Question ← the thing we care about
2) Ideal Experiment ← what's the counterfactual we care about
3) Estimand ← the causal quantity of interest
4) Identification Strategy ← how we connect features of a probability distribution of observed data to causal estimand.
5) Estimation ← how we estimate a feature of a probability distribution from observed data.
6) Inference/Uncertainty ← what would have happened if we observed a different treatment assignment? (and possibly sampled a different population)

'Whats your identification strategy?' means 'what are the assumptions that allow you to claim that the association you've estimated has a causal interpretation?'

Selection on observables is an identification strategy

Identification depends on assumptions not statistical models.
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- Identification depends on assumptions not statistical models.
Estimation

Estimation is secondary to identification. Selection on observables generally requires estimating at least one conditional expectation function and there are many ways to do that. An incomplete list of strategies:

- matching
- weighting
- regression
- combinations of the above

Today we will talk about regression because that's the subject of the class. A big topic I'm skipping over as outside the scope of class is the propensity score (conditional expectation of the treatment given the covariates).
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David Freedman:

*I sometimes have a nightmare about Kepler. Suppose a few of us were transported back in time to the year 1600, and were invited by the Emperor Rudolph II to set up an Imperial Department of Statistics in the court at Prague. Despairing of those circular orbits, Kepler enrolls in our department. We teach him the general linear model, least squares, dummy variables, everything. He goes back to work, fits the best circular orbit for Mars by least squares, puts in a dummy variable for the exceptional observation - and publishes. And that’s the end, right there in Prague at the beginning of the 17th century.*
Regression and Causality
Regression and Causality

- Regression is an estimation strategy that can be used with an identification strategy to estimate a causal effect.
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When is regression causal?
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Regression and Causality

- Regression is an estimation strategy that can be used with an identification strategy to estimate a causal effect.
- When is regression causal? When the CEF is causal.
- This means that the question of whether regression has a causal interpretation is a question about identification.
Identification under Selection on Observables: Regression

Consider the linear regression of \( Y_i = \beta_0 + \tau D_i + X_i' \beta + \epsilon_i \).

Given selection on observables, there are mainly three identification scenarios:

1. Constant treatment effects and outcomes are linear in \( X \) ▶ \( \tau \) will provide unbiased and consistent estimates of ATE.

2. Constant treatment effects and unknown functional form ▶ \( \tau \) will provide well-defined linear approximation to the average causal response function \( E[Y | D = 1, X] - E[Y | D = 0, X] \). Approximation may be very poor if \( E[Y | D, X] \) is misspecified and then \( \tau \) may be biased for the ATE.

3. Heterogeneous treatment effects (\( \tau \) differs for different values of \( X \)) ▶ If outcomes are linear in \( X \), \( \tau \) is unbiased and consistent estimator for conditional-variance-weighted average of the underlying causal effects. This average can be different from the ATE.
Consider the linear regression of \( Y_i = \beta_0 + \tau D_i + X_i' \beta + \epsilon_i \).

Given selection on observables, there are mainly three identification scenarios:

1. **Constant treatment effects and outcomes are linear in \( X \)**: \( \tau \) will provide unbiased and consistent estimates of the average treatment effect (ATE).

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3. **Heterogeneous treatment effects (\( \tau \) differs for different values of \( X \))**: If outcomes are linear in \( X \), \( \tau \) is an unbiased and consistent estimator for the conditional-variance-weighted average of the underlying causal effects. This average can be different from the ATE.
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Identification under Selection on Observables: Regression

Identification Assumption

1. Constant treatment effect: \( \tau = Y_{1i} - Y_{0i} \) for all \( i \)
2. Control outcome is linear in \( X \): \( Y_{0i} = \beta_0 + X_i'\beta + \epsilon_i \) with \( \epsilon_i \perp \perp X_i \) (no omitted variables and linearly separable confounding)

Identification Result

Then \( \tau_{ATE} = \mathbb{E}[Y_1 - Y_0] \) is identified by a regression of the observed outcome on the covariates and the treatment indicator
\[
Y_i = \beta_0 + \tau D_i + X_i'\beta + \epsilon_i
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Ideal Case: Linear Constant Effects Model

Assume constant linear effects and linearly separable confounding:

$$Y_i(d) = Y_i = \beta_0 + \tau D_i + \eta_i$$
Ideal Case: Linear Constant Effects Model

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- **Linearly separable confounding:** assume that \( \mathbb{E}[\eta_i|X_i] = X_i'\beta \), which means that \( \eta_i = X_i'\beta + \epsilon_i \) where \( \mathbb{E}[\epsilon_i|X_i] = 0 \).
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- Under this model, \((Y_1, Y_0) \perp \perp D|X\) implies \( \epsilon_i|X \perp \perp D \)
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- Under this model, \((Y_1, Y_0) \perp D|X\) implies \(\epsilon_i|X \perp D\)
- As a result,

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Ideal Case: Linear Constant Effects Model

Assume constant linear effects and linearly separable confounding:

\[ Y_i(d) = Y_i = \beta_0 + \tau D_i + \eta_i \]

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- Thus, a regression where \( D_i \) and \( X_i \) are entered linearly can recover the ATE.
Implausible $\leadsto$ Plausible
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- Constant effects and linearly separable confounding aren’t very appealing or plausible assumptions
Implausible ⇝ Plausible

- **Constant effects and linearly separable confounding** aren’t very appealing or plausible assumptions.
- To understand what happens when they don’t hold, we need to understand the properties of regression with minimal assumptions: this is often called an agnostic view of regression.
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The Aronow and Miller book (and lecture 7) provide some context but essentially as long as we have iid sampling, we will asymptotically obtain the best linear approximation to the CEF.
1. The Experimental Ideal
2. Assumption of No Unmeasured Confounding
3. Estimation Under No Unmeasured Confounding
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5. Regression and Causality
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Regression and causality

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The question, then, is when does knowing the CEF tell us something about causality?

Angrist and Pishke argues that a regression is causal when the CEF it approximates is causal. Identification is king.

We will show that under certain conditions, a regression of the outcome on the treatment and the covariates can recover a causal parameter, but perhaps not the one in which we are interested.
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- We will show that under certain conditions, a regression of the outcome on the treatment and the covariates can recover a causal parameter, but perhaps not the one in which we are interested.
Now with the benefit of covering agnostic regression, let’s review again the simple case.

- **Experiment**: with a simple experiment, we can rewrite the consistency assumption to be a regression formula:

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

Note that if ignorability holds (as in an experiment) for \( Y_i(0) \), then it will also hold for \( v_0_i \), since \( E[Y_i(0)] \) is constant. Thus, this satisfies the usual assumptions for regression.
Linear constant effects model, binary treatment

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Now with covariates

Now assume no unmeasured confounders: $Y_i(d) \perp D_i | X_i$. 

Remember that linearity isn’t an assumption if $D_i$ is binary. Effect of $D_i$ is constant here, the $\eta_i$ are the only source of individual variation and we have $E[\eta_i] = 0$. Consistency assumption allows us to write this as: $Y_i = \alpha + \tau D_i + \eta_i$. 

Stewart (Princeton)

Week 10: Measured Confounding

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Now with covariates

- Now assume no unmeasured confounders: \( Y_i(d) \perp D_i | X_i \).
- We will assume a linear model for the potential outcomes:

\[
Y_i(d) = \alpha + \tau \cdot d + \eta_i
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Covariates in the error

- Let’s assume that \( \eta_i \) is linear in \( X_i \): \( \eta_i = X_i' \gamma + \nu_i \)
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- Let’s assume that $\eta_i$ is linear in $X_i$: $\eta_i = X_i'\gamma + \nu_i$
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Summing up regression with constant effects

- Reviewing the assumptions we’ve used:
  - no unmeasured confounders
  - constant treatment effects
  - linearity of the treatment/covariates

Under these, we can run the following regression to estimate the ATE, \( \tau \):

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Y_i = \alpha + \tau D_i + X_i' \gamma + \nu_i
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Works with continuous or ordinal \( D_i \) if effect of these variables is truly linear.
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- Thus, OLS estimates the ATE with no covariates.
Adding covariates

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- ATE/ATT are weighted averages of CATEs.
- What about the regression estimand, $\tau_R$? How does it relate to the ATE/ATT?
Heterogeneous effects and regression

Let’s investigate this under a saturated regression model:

\[ Y_i = \sum_x B_{xi} \alpha_x + \tau R D_i + e_i. \]
Heterogeneous effects and regression

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Use a dummy variable for each unique combination of \( X_i \):

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- Let’s investigate this under a saturated regression model:
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- Linear in \( X_i \) by construction!
How can we investigate $\tau_R$? Well, we can rely on the regression anatomy:

$$\tau_R = \frac{\text{Cov}(Y_i, D_i - E[D_i|X_i])}{\text{Var}(D_i - E[D_i|X_i])}$$
Investigating the regression coefficient

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- With a little work we can show:
  \[
  \tau_R = \frac{\mathbb{E}\left[\tau(X_i)(D_i - E[D_i|X_i])^2\right]}{\mathbb{E}[(D_i - E[D_i|X_i])^2]} = \frac{\mathbb{E}[\tau(X_i)\sigma_d^2(X_i)]}{\mathbb{E}[\sigma_d^2(X_i)]}
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- \( \sigma_d^2(x) = \text{Var}[D_i|X_i = x] \) is the conditional variance of treatment assignment.
ATE versus OLS

\[ \tau_R = \mathbb{E}[\tau(X_i)W_i] = \sum_x \tau(x) \frac{\sigma^2_d(x)}{\mathbb{E}[\sigma^2_d(X_i)]} P[X_i = x] \]
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- Compare to the ATE:

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- Both weight strata relative to their size \((\mathbb{P}[X_i = x])\)
ATE versus OLS

\[ \tau_R = \mathbb{E}[\tau(X_i)W_i] = \sum_x \tau(x) \frac{\sigma_d^2(x)}{\mathbb{E}[\sigma_d^2(X_i)]} \mathbb{P}[X_i = x] \]

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- The ATE weights only by their size.
Regression weighting

\[ W_i = \frac{\sigma^2_d(X_i)}{\mathbb{E}[\sigma^2_d(X_i)]} \]

Why does OLS weight like this?
Regression weighting

\[ W_i = \frac{\sigma^2_d(X_i)}{E[\sigma^2_d(X_i)]]} \]

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\[ \sigma^2_d(x) = \mathbb{P}[D_i = 1|X_i = x] \left(1 - \mathbb{P}[D_i = 1|X_i = x]\right) \]
Regression weighting

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- Maximum variance with \( \mathbb{P}[D_i = 1|X_i = x] = 1/2 \).
OLS weighting example

- Binary covariate:
OLS weighting example

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<table>
<thead>
<tr>
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<tr>
<td>[P[X_i = 1] = 0.75]</td>
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\[\tau = \frac{0.09}{0.13} = 0.692, \quad \frac{0.25}{0.13} = 1.92.\]

\[
\tau_R = E[\tau(X_i)W_i] = \tau(1)W(1)P[X_i = 1] + \tau(0)W(0)P[X_i = 0] = 0.039
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### OLS weighting example

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Implies the ATE is \( \tau = 0.5 \).

Average conditional variance:
\[
E[\sigma_d^2(X_i)] = 0.13
\]

\( \Rightarrow \) weights for \( X_i = 1 \) are: \( \frac{0.09}{0.13} = 0.692 \), for \( X_i = 0 \): \( \frac{0.25}{0.13} = 1.92 \).

\( \tau_R = E[\tau(X_i)W_i] = \tau(1)W(1)P[X_i = 1] + \tau(0)W(0)P[X_i = 0] = 1 \times 0.692 + (-1) \times 1.92 = 0.039 \).
### OLS weighting example

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**Average conditional variance:**

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**$\tau_R = E[\tau(X_i)W_i] = \tau(1)W(1)P[X_i = 1] + \tau(0)W(0)P[X_i = 0]$**

$= 1 \times 0.692 + (-1) \times 1.92 = 0.039$. 
OLS weighting example

- Binary covariate:

  
  \[
  \begin{array}{c|cc}
  & \text{Group 1} & \text{Group 2} \\
  \mathbb{P}[X_i = 1] & 0.75 & 0.25 \\
  \mathbb{P}[D_i = 1 | X_i = 1] & 0.9 & 0.5 \\
  \sigma^2_d(1) & 0.09 & 0.25 \\
  \tau(1) & 1 & -1 \\
  \end{array}
  \]

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When will OLS estimate the ATE?

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Implies that the OLS weights are 1. Incorrect linearity assumption in $X_i$ will lead to more bias.
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Other ways to use regression

- What’s the path forward?

\[ \mu_d(x) = \mathbb{E}[Y_i | D_i = d, X_i = x] \]

By consistency and n.u.c., we have \( \mu_d(x) = \mathbb{E}[Y_i | D_i = d, X_i = x] \).

Estimate a regression of \( Y_i \) on \( X_i \) among the \( D_i = d \) group. Then, \( \hat{\mu}_d(x) \) is just a predicted value from the regression for \( X_i = x \).
Other ways to use regression

- What’s the path forward?
  - Accept the bias (might be relatively small with saturated models)
Other ways to use regression

- What’s the path forward?
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- Let \( \mu_d(x) = \mathbb{E}[Y_i(d)|X_i = x] \) be the CEF for the potential outcome under \( D_i = d \).
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- How can we use this?
Imputation estimators

- Impute the treated potential outcomes with $\hat{Y}_i(1) = \hat{\mu}_1(X_i)$!
Imputation estimators

- Impute the treated potential outcomes with $\hat{Y}_i(1) = \hat{\mu}_1(X_i)$.
- Impute the control potential outcomes with $\hat{Y}_i(0) = \hat{\mu}_0(X_i)$.

Procedure:

- Regress $Y_i$ on $X_i$ in the treated group and get predicted values for all units (treated or control).
- Regress $Y_i$ on $X_i$ in the control group and get predicted values for all units (treated or control).
- Take the average difference between these predicted values.

Mathematically, this looks like:

$$\tau_{imp} = \frac{1}{N} \sum_i \hat{\mu}_1(X_i) - \hat{\mu}_0(X_i)$$

Sometimes called an imputation estimator.
Imputation estimators

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Simple imputation estimator

- Use `predict()` from the within-group models on the data from the entire sample.
Simple imputation estimator

- Use `predict()` from the within-group models on the data from the entire sample.
- Useful trick: use a model on the entire data and `model.frame()` to get the right design matrix:

```r
# heterogeneous effects
y.het <- ifelse(d == 1, y + rnorm(n, 0, 5), y)
mod <- lm(y.het ~ d + X)
mod1 <- lm(y.het ~ X, subset = d == 1)
mod0 <- lm(y.het ~ X, subset = d == 0)
y1.imps <- predict(mod1, model.frame(mod))
y0.imps <- predict(mod0, model.frame(mod))
mean(y1.imps - y0.imps)
## [1] 0.61
```
Simple imputation estimator

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mod1 <- lm(y.het ~ X, subset = d == 1)
mod0 <- lm(y.het ~ X, subset = d == 0)

y1.imps <- predict(mod1, model.frame(mod))
y0.imps <- predict(mod0, model.frame(mod))
mean(y1.imps - y0.imps)
```

## [1] 0.61
Notes on imputation estimators

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  - Most people don’t know the results we’ve been talking about.

- Harder to implement than vanilla OLS.
- Can use linear regression to estimate $\hat{\mu}_d(x) = x'\beta_d$
- Recent trend is to estimate $\hat{\mu}_d(x)$ via non-parametric methods such as:
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Imputation estimator visualization
Imputation estimator visualization

- Treated
- Control

Week 10: Measured Confounding

November 26 and 28, 2018
Imputation estimator visualization
Nonlinear relationships

- Same idea but with nonlinear relationship between $Y_i$ and $X_i$:
Nonlinear relationships

- Same idea but with nonlinear relationship between $Y_i$ and $X_i$: 

![Graph showing nonlinear relationship between $x$ and $y$ with two lines and two sets of data points (Treated and Control).]
Nonlinear relationships

- Same idea but with nonlinear relationship between $Y_i$ and $X_i$:
Using semiparametric regression

- Here, CEFs are nonlinear, but we don’t know their form.

```r
library(mgcv)
mod0 <- gam(y ~ s(x), subset = d == 0)
summary(mod0)
```

```
##
## Family: gaussian
## Link function: identity
##
## Formula:
## y ~ s(x)
##
## Parametric coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   -0.0225     0.0154   -1.46   0.16

## Approximate significance of smooth terms:
## edf Ref.df F p-value
## s(x) 6.03    7.08 41.3 <2e-16 ***
```

Stewart (Princeton)

Week 10: Measured Confounding

November 26 and 28, 2018
Using semiparametric regression

- Here, CEFs are nonlinear, but we don’t know their form.
- We can use GAMs from the mgcv package to for flexible estimate:

```r
library(mgcv)
mod0 <- gam(y ~ s(x), subset = d == 0)
summary(mod0)
```

```markdown
##
## Family: gaussian
## Link function: identity
##
## Formula:
## y ~ s(x)
##
## Parametric coefficients:
## Estimate Std. Error t value Pr(>|t|)
## (Intercept) -0.0225  0.0154  -1.46  0.16
##
## Approximate significance of smooth terms:
## edf Ref.df  F p-value
## s(x) 6.03  7.08 41.3 <2e-16 ***
## ---
## Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1
##
```
Using GAMs
Using GAMs

The graph illustrates the application of Generalized Additive Models (GAMs) with a scatter plot comparing treated and control groups.

- **Treated** group data is marked with red dots.
- **Control** group data is marked with blue triangles.

The x-axis represents the independent variable, while the y-axis represents the dependent variable. The curves suggest a non-linear relationship between the variables for both groups.

The presence of confounding variables is explored, and the GAMs are used to control for these effects, allowing for a clearer comparison between treated and control groups.
Using GAMs
‘Wait...so what are we actually doing most of the time?’

A Discussion
Conclusions

- Regression is mechanically very simple, but philosophically somewhat complicated
Conclusions

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- It is a useful descriptive tool for approximating a conditional expectation function
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- Once again though, the estimand of interest isn’t necessarily the regression coefficient.
Conclusions

- Regression is mechanically very simple, but philosophically somewhat complicated.
- It is a useful descriptive tool for approximating a conditional expectation function.
- Once again though, the estimand of interest isn’t necessarily the regression coefficient.
- There are many other approaches to estimation, but identification is key.
Next Week

- Causality with Unmeasured Confounding

- Reading:
  - Angrist and Pishke Chapter 4 Instrumental Variables and Chapter 6 on Regression Discontinuity Designs
  - Morgan and Winship Chapter 9 Instrumental Variable Estimators of Causal Effects
  - Optional: Hernan and Robins Chapter 16 Instrumental Variable Estimation
1. The Experimental Ideal

2. Assumption of No Unmeasured Confounding

3. Estimation Under No Unmeasured Confounding

4. Regression Estimators

5. Regression and Causality

6. Regression Under Heterogeneous Effects

7. Fun with Visualization, Replication and the NYT
1. The Experimental Ideal
2. Assumption of No Unmeasured Confounding
3. Estimation Under No Unmeasured Confounding
4. Regression Estimators
5. Regression and Causality
6. Regression Under Heterogeneous Effects
7. Fun with Visualization, Replication and the NYT
WASHINGTON — Yascha Mounk is used to being the most pessimistic person in the room. Mr. Mounk, a lecturer in government at Harvard, has spent the past few years challenging one of the bedrock assumptions of Western politics: that once a country becomes a liberal democracy, it will stay that way.

His research suggests something quite different: that liberal democracies around the world may be at serious risk of decline.

Mr. Mounk’s interest in the topic began rather unusually. In 2014, he published a book, “Stranger in My Own Country.” It started as a memoir of his experiences growing up as a Jew in Germany, but became a broader investigation of how contemporary European nations were struggling to construct new, multicultural national identities.
Visualization in the New York Times

The Danger of Deconsolidation

THE DEMOCRATIC DISCONNECT

Roberto Stefan Foa and Yascha Mounk

Roberto Stefan Foa is a principal investigator of the World Values Survey and fellow of the Laboratory for Comparative Social Research. His writing has appeared in a wide range of journals, books, and publications by the UN, OECD, and World Bank. Yascha Mounk is a lecturer on political theory in Harvard University’s Government Department and a Carnegie Fellow at New America, a Washington, D.C.–based think tank. His dissertation on the role of personal responsibility in contemporary politics and philosophy will be published by Harvard University Press, and his essays have appeared in Foreign Affairs, the New York Times, and the Wall Street Journal.
Visualization in the New York Times

Percentage of people who say it is “essential” to live in a democracy

<table>
<thead>
<tr>
<th>Sweden</th>
<th>Australia</th>
<th>Netherlands</th>
<th>United States</th>
<th>New Zealand</th>
<th>Britain</th>
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</thead>
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</tr>
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</table>

Decade of birth: 1930s, 1980s, '30s, '80s

Visualization in the New York Times

Ryan D. Enos @RyanDEnos · 19h
Lots of worried chatter a/b @amandataub article on work of @Yascha_Mounk. Important, but want to raise cautions 1/

How Stable Are Democracies? ‘Warning Signs Are Flashing Red’

New research tries to spot the collapse of liberal democracies before they happen, and it suggests that Western democracy may be seriously ill.

tytimes.com
Alternate Graphs

Percentage of people who say it is “essential” to live in a democracy

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| 95% confidence intervals

Alternate Graphs

@RyanDEnos Compare NYT/JoD (left) to the very same data analysed differently by Bartels and Achen (2016) (right). Extreme score vs means.

Across numerous countries, including Australia, Britain, the Netherlands, New Zealand, Sweden and the United States, the percentage of people who say it is “essential” to live in a democracy has plummeted, and it is especially low among younger generations.

[Graphs showing data trends for various countries]
@RyanDEnos They also stop at the 80's cohort. The data has the 90's as well. I wonder why they would stop there...
Alternate Graphs

Percentage of people who say it is extremely important to live in a country that is governed democratically

Decade of birth

Source: ESS Wave 6

In reply to Ryan D. Enos
Benjamin Sack @bcsack · 15h
@RyanDEnos Same analysis strategy with comparable data from @ESS_Survey (similar item, 0-10 scale) shows slightly different pattern, too.
Alternate Graphs

How important is it for you to live in a country that is governed democratically?

Australia
Netherlands
New Zealand
Sweden
United States

Respondent age

Absolutely important
Not at all important
How important is it for you to live in a country that is governed democratically? United States, 2011

Data: World Values Survey Wave 6
Author: @DToshkov, http://www.dIMITER.eu

Alternate Graphs
Thoughts

Two stories here:
Thoughts

Two stories here:

1. Visualization and data coding choices are important
Thoughts

Two stories here:

1. Visualization and data coding choices are important
2. The internet is amazing (especially with replication data being available!)