Week 10: Causality with Measured Confounding

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Princeton

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$^1$These 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 → inference → regression → 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
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?

Confounders

\[ \begin{align*}
X & \\
T & \rightarrow X \\
Y & \rightarrow T
\end{align*} \]
Observational Studies and Experimental Ideal

- Randomization forms gold standard for causal inference, because it balances observed and unobserved confounders.

- Cannot always randomize so we do observational studies, where we adjust for the observed covariates and hope that unobservables are balanced.

- 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

- What is the causal relationship of interest?
- 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?
Experiment review

- An **experiment** is a study where assignment to treatment is controlled by the researcher.
  
  ▶ $p_i = \mathbb{P}[D_i = 1]$ 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**: $\mathbb{P}[D_i = 1|Y(1), Y(0)] = \mathbb{P}[D_i = 1]$
     
     ▶ Treatment assignment does not depend on any potential outcomes.
     ▶ 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

In an experiment we know that treatment is randomly assigned. Thus we can do the following:

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

When all goes well, an experiment eliminates selection bias.
Observational studies

- Many different sets of identification assumptions that we’ll cover.
- To start, focus on studies that are similar to experiments, just without a known and controlled treatment assignment.
  - No guarantee that the treatment and control groups are comparable.

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

2. **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.
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.
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:

$$
\mathbb{E}_X \left\{ \mathbb{E}[Y_i | D_i = 1, X_i] - \mathbb{E}[Y_i | D_i = 0, X_i] \right\} \\
= \left( \mathbb{E}[Y_i | D_i = 1, X_i = 1] - \mathbb{E}[Y_i | D_i = 0, X_i = 1] \right) \mathbb{P}[X_i = 1] \\
+ \left( \mathbb{E}[Y_i | D_i = 1, X_i = 0] - \mathbb{E}[Y_i | D_i = 0, X_i = 0] \right) \mathbb{P}[X_i = 0]
$$

Never used our knowledge of the randomization for this quantity.
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

<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>54.9</td>
<td>49.1</td>
<td>57.0</td>
</tr>
<tr>
<td>Cigarettes</td>
<td>50.5</td>
<td>49.8</td>
<td>53.2</td>
</tr>
<tr>
<td>Cigars/pipes</td>
<td>65.9</td>
<td>55.7</td>
<td>59.7</td>
</tr>
</tbody>
</table>
Stratification

To control for differences in age, we would like to compare different smoking-habit groups with the same age distribution.

One possibility is to use stratification:

- 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></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></td>
</tr>
</tbody>
</table>

What is the average death rate for Pipe Smokers?

\[
15 \cdot \left(\frac{11}{40}\right) + 35 \cdot \left(\frac{13}{40}\right) + 50 \cdot \left(\frac{16}{40}\right) = 35.5
\]
Stratification: Example

<table>
<thead>
<tr>
<th>Age Range</th>
<th>Death Rates Pipe Smokers</th>
<th># Pipe-Smokers</th>
<th># Non-Smokers</th>
</tr>
</thead>
<tbody>
<tr>
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<td>15</td>
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<td>16</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>40</td>
<td></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>
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:
    \[
    \mathbb{E}[Y_i|D_i = 1, X_i = 54134] - \mathbb{E}[Y_i|D_i = 0, X_i = 54134]
    \]
  - $\leadsto$ cannot stratify to each unique value of $X_i$:
- 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!
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

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

Therefore, under the common support condition:

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

Identification Assumption

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

Identification Result

Similarly,

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

\[
= \int \left( \mathbb{E}[Y | X, D = 1] - \mathbb{E}[Y | X, D = 0] \right) dP(X | D = 1)
\]

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

- \(Y_0 \independent D | X\) (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>
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</tr>
<tr>
<td>8</td>
<td></td>
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Stewart (Princeton)
Identification Under Selection on Observables

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<td>X = 1, D = 0]$</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 $X$:

\[
\mathbb{E}[Y_0|X = 0, D = 1] = \mathbb{E}[Y_0|X = 0, D = 0] \quad \text{and} \\
\mathbb{E}[Y_0|X = 1, D = 1] = \mathbb{E}[Y_0|X = 1, D = 0]
\]
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$(Y_1, Y_0) \perp D|X$ also implies

$$
\mathbb{E}[Y_1|X = 0, D = 1] = \mathbb{E}[Y_1|X = 0, D = 0] \quad \text{and} \\
\mathbb{E}[Y_1|X = 1, D = 1] = \mathbb{E}[Y_1|X = 1, D = 0]
$$
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
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.
How can we determine if no unmeasured confounding holds if we didn’t assign the treatment?

Put differently:

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

One way, from the assumption itself:

- \( \mathbb{P}[D_i = 1 | X, Y(1), Y(0)] = \mathbb{P}[D_i = 1 | X] \)
- 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.
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 $Y$ are common causes of $D$ and $Y$:

  $\begin{align*}
  &\quad X \\
  &\quad \downarrow \\
  &D \rightarrow Y
  \end{align*}$

- 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

- \( D \) is enrolling in a job training program.
- \( Y \) is getting a job.
- \( U \) is being motivated
- \( X \) is number of job applications sent out.
- Big assumption here: no arrow from \( U \) to \( Y \).

\[
\begin{align*}
U \rightarrow X \\
\downarrow \downarrow \\
D \rightarrow Y
\end{align*}
\]
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 $Y$. 

\[ U \rightarrow X \]
\[ \downarrow \]
\[ D \rightarrow Y \]
What’s the problem with backdoor paths?

A path is blocked if:

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

Unblocked backdoor paths $\Rightarrow$ confounding.

In the DAG here, if we condition on $X$, then the backdoor path is blocked.
Not all backdoor paths

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

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

Every time you do, a puppy cries.
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 $X \rightarrow$ opens the path and induces 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!
Backdoor criterion

• Can we use a DAG to evaluate no unmeasured confounders?
• 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.
Remove arrows out of $X$. 
Recall that paths are blocked by “unconditioned colliders” or conditioned non-colliders.
Example: Sufficient Conditioning Sets

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Example: Non-sufficient Conditioning Sets

Recall that paths are blocked by “unconditioned colliders” or conditioned non-colliders
Example: Non-sufficient Conditioning Sets
Implications (via Vanderweele and Shpitser 2011)

Two common criteria fail here:

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 $A \leftarrow C_3 \leftarrow U_3 \rightarrow Y$.)
No unmeasured confounders is not testable

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

\[
\frac{Y_i(0) | D_i = 1, X_i}{Y_i(0) | D_i = 0, X_i}
\]

- Here, \( \equiv \) means equal in distribution.
- No way to directly test this assumption without the counterfactual data, which is missing by definition!
- With backdoor criterion, you must have the correct DAG.
Assessing no unmeasured confounders

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

Della Vigna and Kaplan (2007, QJE): effect of Fox News availability on Republican vote share
  ▶ 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!
Where We’ve Been and Where We’re Going...

- **Last Week**
  - intro to causal inference

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  - 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?
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
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)

- ‘What’s 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.
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).
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 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.
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 \( \mathbb{E}[Y|D = 1, X] - \mathbb{E}[Y|D = 0, X] \). Approximation may be very poor if \( \mathbb{E}[Y|D, X] \) 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.
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

\[ Y_i = \beta_0 + \tau D_i + X_i' \beta + \epsilon_i \]
Ideal Case: Linear Constant Effects Model

Assume constant linear effects and linearly separable confounding:

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

- **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 \( \mathbb{E}[\epsilon_i | X_i] = 0 \).
- Under this model, \( (Y_1, Y_0) \perp \perp D | X \) implies \( \epsilon_i | X \perp \perp D \)
- As a result,

\[
Y_i = \beta_0 + \tau D_i + \mathbb{E}[\eta_i] \\
= \beta_0 + \tau D_i + X_i' \beta + \mathbb{E}[\epsilon_i] \\
= \beta_0 + \tau D_i + X_i' \beta \\
\]

- Thus, a regression where \( D_i \) and \( X_i \) are entered linearly can recover the ATE.
Implausible $\leadsto$ 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.

- 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.
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Regression and causality

- Most econometrics textbooks: regression defined without respect to causality.
- But then when is $\hat{\beta}$ “biased”? What does this even mean?
- The question, then, is when does knowing the CEF tell us something about causality?
- Angrist and Pishke argue 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.
Linear constant effects model, binary treatment

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:

\[
Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0) \\
= Y_i(0) + ( Y_i(1) - Y_i(0) ) D_i \\
= \mathbb{E}[ Y_i(0) ] + \tau D_i + ( Y_i(0) - \mathbb{E}[ Y_i(0) ] ) \\
= \mu^0 + \tau D_i + \nu_i^0
\]

- Note that if ignorability holds (as in an experiment) for \( Y_i(0) \), then it will also hold for \( \nu_i^0 \), since \( \mathbb{E}[ Y_i(0) ] \) is constant. Thus, this satisfies the usual assumptions for regression.
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:

\[
Y_i(d) = \alpha + \tau \cdot d + \eta_i
\]

- 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:

\[
Y_i = \alpha + \tau D_i + \eta_i.
\]
Covariates in the error

- Let’s assume that $\eta_i$ is linear in $X_i$: $\eta_i = X_i' \gamma + \nu_i$
- New error is uncorrelated with $X_i$: $\mathbb{E}[\nu_i | X_i] = 0$.
- This is an assumption! Might be false!
- Plug into the above:

$$
\mathbb{E}[Y_i(d) | X_i] = E[Y_i | D_i, X_i] = \alpha + \tau D_i + E[\eta_i | X_i] \\
= \alpha + \tau D_i + X_i' \gamma + E[\nu_i | X_i] \\
= \alpha + \tau D_i + X_i' \gamma
$$
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, $\tau$:

$$Y_i = \alpha + \tau D_i + X_i' \gamma + \nu_i$$

- Works with continuous or ordinal $D_i$ if effect of these variables is truly linear.
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Heterogeneous effects, binary treatment

- Completely randomized experiment:

\[
Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0) \\
= Y_i(0) + (Y_i(1) - Y_i(0)) D_i \\
= \mu_0 + \tau_i D_i + (Y_i(0) - \mu_0) \\
= \mu_0 + \tau D_i + (Y_i(0) - \mu_0) + (\tau_i - \tau) \cdot D_i \\
= \mu_0 + \tau D_i + \varepsilon_i
\]

- Error term now includes two components:

1. “Baseline” variation in the outcome: \((Y_i(0) - \mu_0)\)
2. Variation in the treatment effect, \((\tau_i - \tau)\)

- We can verify that under experiment, \(\mathbb{E}[\varepsilon_i | D_i] = 0\)
- Thus, OLS estimates the ATE with no covariates.
Adding covariates

- What happens with no unmeasured confounders? Need to condition on $X_i$ now.
- Remember identification of the ATE/ATT using iterated expectations.
- ATE is the weighted sum of Conditional Average Treatment Effects (CATEs):

$$\tau = \sum_x \tau(x) \Pr[X_i = x]$$

- ATE/ATT are weighted averages of CATEs.
- What about the regression estimand, $\tau_R$? How does it relate to the ATE/ATT?
Let’s investigate this under a saturated regression model:

\[ Y_i = \sum_x B_{xi} \alpha_x + \tau_R D_i + e_i. \]

- Use a dummy variable for each unique combination of \( X_i \):
  \[ B_{xi} = \mathbb{I}(X_i = x) \]
- Linear in \( X_i \) by construction!
Investigating the regression coefficient

- How can we investigate \( \tau_R \)? Well, we can rely on the regression anatomy:

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

- \( D_i - E[D_i|X_i] \) is the residual from a regression of \( D_i \) on the full set of dummies.

- With a little work we can show:

\[
\tau_R = \frac{\mathbb{E}\left[\tau(X_i)(D_i - E[D_i|X_i])^2\right]}{\mathbb{E}[(D_i - E[D_i|X_i])^2]} = \frac{\mathbb{E}[\tau(X_i)\sigma^2_d(X_i)]}{\mathbb{E}[\sigma^2_d(X_i)]}
\]

- \( \sigma^2_d(x) = \text{Var}[D_i|X_i = x] \) is the conditional variance of treatment assignment.
ATE versus OLS

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

- Compare to the ATE:

\[ \tau = \mathbb{E}[\tau(X_i)] = \sum_x \tau(x) \mathbb{P}[X_i = x] \]

- Both weight strata relative to their size (\( \mathbb{P}[X_i = x] \))
- OLS weights strata higher if the treatment variance in those strata (\( \sigma^2_d(x) \)) is higher in those strata relative to the average variance across strata (\( \mathbb{E}[\sigma^2_d(X_i)] \)).
- The ATE weights only by their size.
Regression weighting

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

- Why does OLS weight like this?
- OLS is a minimum-variance estimator \(\rightsquigarrow\) more weight to more precise within-strata estimates.
- Within-strata estimates are most precise when the treatment is evenly spread and thus has the highest variance.
- If \(D_i\) is binary, then we know the conditional variance will be:

\[ \sigma_i^2(x) = \mathbb{P}[D_i = 1|X_i = x] (1 - \mathbb{P}[D_i = 1|X_i = x]) \]

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

- Binary covariate:

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\mathbb{P}[X_i = 1])</td>
<td>0.75</td>
<td>0.25</td>
</tr>
<tr>
<td>(\mathbb{P}[D_i = 1</td>
<td>X_i = 1])</td>
<td>0.9</td>
</tr>
<tr>
<td>(\sigma^2_d(1))</td>
<td>0.09</td>
<td>0.25</td>
</tr>
<tr>
<td>(\tau(1))</td>
<td>1</td>
<td>-1</td>
</tr>
</tbody>
</table>

- Implies the ATE is \(\tau = 0.5\)
- Average conditional variance: \(\mathbb{E}[\sigma^2_d(X_i)] = 0.13\)
- \(\Rightarrow\) weights for \(X_i = 1\) are: \(0.09/0.13 = 0.692\), for \(X_i = 0\): \(0.25/0.13 = 1.92\).

\[\tau_R = \mathbb{E}[\tau(X_i)W_i] \]
\[= \tau(1)W(1)\mathbb{P}[X_i = 1] + \tau(0)W(0)\mathbb{P}[X_i = 0] \]
\[= 1 \times 0.692 \times 0.75 + (-1) \times 1.92 \times 0.25 \]
\[= 0.039\]
When will OLS estimate the ATE?

- When does $\tau = \tau_R$?
- Constant treatment effects: $\tau(x) = \tau = \tau_R$
- Constant probability of treatment: $e(x) = \mathbb{P}[D_i = 1|X_i = x] = e$.
  - Implies that the OLS weights are 1.
- Incorrect linearity assumption in $X_i$ will lead to more bias.
Other ways to use regression

- What’s the path forward?
  - Accept the bias (might be relatively small with saturated models)
  - Use a different regression approach

- Let $\mu_d(x) = \mathbb{E}[Y_i(d)|X_i = x]$ be the CEF for the potential outcome under $D_i = d$.
- 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 $X_i = x$.
- How can we use this?
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.
- More mathematically, look like this:
  \[
  \tau_{imp} = \frac{1}{N} \sum_i \hat{\mu}_1(X_i) - \hat{\mu}_0(X_i)
  \]
- Sometimes called an **imputation estimator**.
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
```
Notes on imputation estimators

- If $\hat{\mu}_d(x)$ are consistent estimators, then $\tau_{imp}$ is consistent for the ATE.
- Why don’t people use this?
  - 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:
  - Kernel regression, local linear regression, regression trees, etc
  - Easiest is generalized additive models (GAMs)
Imputation estimator visualization

Treated
Control

Stewart (Princeton)
Imputation estimator visualization

- Treated
- Control

Stewart (Princeton)
Week 10: Measured Confounding
November 26 and 28, 2018
Imputation estimator visualization
Nonlinear relationships

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

![Graph showing nonlinear relationship between treated and control]
Nonlinear relationships

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

![Graph showing nonlinear relationship between $X$ and $Y$]
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.
- 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)
```

```
##
## 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

- Treated
- Control

Week 10: Measured Confounding
November 26 and 28, 2018
Using GAMs
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
- 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
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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.
Alternate Graphs

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

<table>
<thead>
<tr>
<th>Country</th>
<th>1930s</th>
<th>1980s</th>
<th>'30s</th>
<th>'80s</th>
<th>'30s</th>
<th>'80s</th>
<th>'30s</th>
<th>'80s</th>
<th>'30s</th>
<th>'80s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweden</td>
<td></td>
<td></td>
<td>75%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>100%</td>
<td></td>
<td>75%</td>
<td>60%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Netherlands</td>
<td>95%</td>
<td></td>
<td></td>
<td>50%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>United States</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>100%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Zealand</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>95%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Britain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>100%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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.
@RyanDEnos They also stop at the 80s 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

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

Respondent age

Australia
Netherlands
New Zealand
Sweden
United States
Alternate Graphs

How important is it for you to live in a country that is governed democratically? United States, 2011

Born in 1980s: Red
Born in 1940s: Blue

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

Dimiter Toshkov  @DToshkov · 31m
my take on the democratic deconsolidation graph that scared everyone yesterday. Blue is 1940s cohort, red is 1980s.
First, United States
Thoughts

Two stories here:

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