Regression analysis of experiments
Regression analysis of an experiment

Recall using OLS to fit the following model:

\[ Y_i \approx \hat{\beta}_0 + \hat{\beta}_1 W_i, \]

where \( W_i \in \{0, 1\} \) is the assignment in a randomized experiment (0 is control, 1 is treatment), and \( Y_i \) is the corresponding observed outcome for individual \( i \).

As we showed:

- \( \hat{\beta}_0 \) is the average outcome in the control group.
- \( \hat{\beta}_0 + \hat{\beta}_1 \) is the average outcome in the treatment group.
- \( \hat{\beta}_1 = \hat{ATE} \).
Going further

In this lecture we consider what happens when we have additional covariates we can exploit in our analysis.

Suppose in addition to $Y(0), Y(1), \text{ and } W$, each individual also has a vector of observed covariates $\vec{X}$.
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There are two ways in which the regression approach to experimental analysis is powerful:

- Controlling for observed covariates helps improve estimation of the ATE.
- Interactions with the treatment effect allow us to see how the treatment effect varies among individuals with different covariate vectors.

Warning: The covariates $\vec{X}$ must be observed pre-treatment!
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**Warning:** The covariates $\vec{X}$ must be observed *pre-treatment!*
Controlling for observables: An example

I created a synthetic experiment where $n_0 = n_1 = 150$.

For each individual $i$, $X_i \sim \mathcal{N}(0, 1)$ is a pre-existing covariate, and $W_i$ is the treatment indicator.

I contructed $Y_i$ as:

$$Y_i = 10 + 0.5 \times W_i + X_i + \varepsilon_i,$$

where $\varepsilon_i \sim \mathcal{N}(0, 1)$.

In this example:

- The true ATE is 0.5—it does not vary depending on $X$.
- However, some of the variation in $Y_i$’s is explained the $X$’s as well.
Controlling for observables: An example

Suppose we regress $Y$ on the treatment indicator $W$ alone:

Call:
`lm(formula = Y ~ 1 + W, data = df)`

...  

Coefficients:

|            | Estimate | Std. Error | t value | Pr(>|t|)   |
|------------|----------|------------|---------|-----------|
| (Intercept)| 9.9807   | 0.1168     | 85.45   | < 2e-16   *** |
| W1         | 0.4608   | 0.1652     | 2.79    | 0.00561 ** |
Controlling for observables: An example

Now suppose we include the covariate $X$ in the regression:

Call:
\[ \text{lm(formula = Y } \sim \ 1 + W + X, \ data = df) \]

\[
\begin{array}{cccccc}
\text{Coefficients:} & \text{Estimate} & \text{Std. Error} & \text{t value} & \text{Pr(>|t|)} \\
(Intercept) & 9.91498 & 0.08688 & 114.123 & < 2e-16 *** \\
W1 & 0.61827 & 0.12314 & 5.021 & 8.88e-07 *** \\
X & 1.01032 & 0.06483 & 15.584 & < 2e-16 *** \\
\end{array}
\]

Notice that the standard error is smaller on the coefficient of $W$. 
Controlling for observables: Interpretation

In the specification $Y \sim 1 + W + X$, we still interpret the coefficient on $W$ as an estimate of the population-level ATE.

The point is that adding $X$ to the regression gives us a better estimate of the baseline $Y(0)$ for each individual.

Essentially, this regression says that for an individual with covariate $X$:

- $Y(0) \approx \hat{\beta}_0 + \hat{\beta}_2 X$.
- $Y(1) \approx \hat{\beta}_0 + \hat{\beta}_1 + \hat{\beta}_2 X$. 
Controlling for observables

Controlling for observed covariates has another effect as well:

If the randomization was less than perfect, controlling for observed covariates can reduce the sampling bias.

How this works:

▶ Suppose, e.g., individuals with higher $X$ were more likely to receive the treatment.

What are the limitations to this process?
Controlling for observables

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How this works:

▶ Suppose, e.g., individuals with higher $X$ were more likely to receive the treatment.
▶ Ignoring this fact will lead to a biased estimate of the ATE: part of the variation in the observed $Y$’s is explained by variation in the $X$’s, *not* by the variation in the treatment. (This is an omitted variable bias.)

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- Controlling for $X$ removes the omitted variable bias.

What are the limitations to this process?
The preceding slides suggest one limitation of merely controlling for observed covariates:

*What if the treatment effect itself varies depending on the covariates observed?*

To address this issue we employ interactions with the treatment indicator.
Suppose given a covariate $X$, we add the interaction term $W \times X$ to the model:

$$Y_i \approx \hat{\beta}_0 + \hat{\beta}_W W_i + \hat{\beta}_X X_i + \hat{\beta}_{WX} W_i X_i.$$ 

With the addition of this term we can interpret the model as follows:
Interactions

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For an individual with covariate $X$,

- $Y(0) \approx \hat{\beta}_0 + \hat{\beta}_X X$.
- $Y(1) \approx \hat{\beta}_0 + \hat{\beta}_W + (\hat{\beta}_X + \hat{\beta}_{WX}) X$.  

---

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- $Y(0) \approx \beta_0 + \beta_X X$.
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- The estimated causal effect is $\approx \beta_W + \beta_{WX} X$. 

This allows us to measure heterogeneous treatment effects across the population.
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- The estimated causal effect is $\approx \hat{\beta}_W + \hat{\beta}_{WX} X$.

This allows us to measure heterogeneous treatment effects across the population.
Interactions: Example

In the earlier example, there should be no meaningful change in the treatment effect across individuals with different $X$'s.

Call:
```
lm(formula = Y ~ 1 + W + X + X * W, data = df)
```

...  

Coefficients:

|                | Estimate | Std. Error | t value | Pr(> |t|)     |
|----------------|----------|------------|---------|---------|
| (Intercept)    | 9.91161  | 0.08705    | 113.860 | < 2e-16 *** |
| W1             | 0.61730  | 0.12323    | 5.009   | 9.4e-07 *** |
| X              | 1.06204  | 0.09365    | 11.340  | < 2e-16 *** |
| W1:X           | -0.09945 | 0.12986    | -0.766  | 0.444   |
Interactions: Example

Now suppose we change the model so that in the population, changing $X$ also changes the treatment effect.

In particular, suppose:

$$Y_i = 10 + (0.5 + X_i)W_i + X_i + \varepsilon_i,$$

where $\varepsilon_i \sim \mathcal{N}(0, 1)$.

What happens when we estimate a model with interactions on the resulting experimental data?
Interactions: Example

The result:

Call:
\( \text{lm(formula = Y \sim 1 + W + X + X * W, data = df)} \)

Residuals:

<table>
<thead>
<tr>
<th>Min</th>
<th>1Q</th>
<th>Median</th>
<th>3Q</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>-2.32757</td>
<td>-0.73146</td>
<td>0.05078</td>
<td>0.62216</td>
<td>2.85012</td>
</tr>
</tbody>
</table>

Coefficients:

| Term     | Estimate | Std. Error | t value | Pr(>|t|) |
|-----------|----------|------------|---------|---------|
| (Intercept) | 10.02695 | 0.08510 | 117.827 | < 2e-16 *** |
| W1        | 0.51447  | 0.12047 | 4.271   | 2.63e-05 *** |
| X         | 1.06476  | 0.09155 | 11.630  | < 2e-16 *** |
| W1:X      | 0.86899  | 0.12695 | 6.845   | 4.40e-11 *** |
SUTVA and interference
Implicitly throughout our discussion of causal inference, we have assumed there is no *interference* between treatment and control: Whether or not individual $i$ receives treatment or control has *no* *impact* on the causal effect of treatment on another individual $j$. When might this fail?
Suppose Airbnb decides to A/B test a new feature that dramatically simplifies the booking process for a guest.

In the test, guests are randomized at when they start the booking process; control is the old experience, treatment is the new experience.

It is found that customers with the new experience book much more frequently than customers with the old experience, but the estimated $\hat{ATE}$ is an overestimate. Why?
Interference

Both treatment and control see the *same* inventory of host listings!

So if treatment individuals book more often, that *reduces* the inventory available to control individuals, and implies their booking rates will be lower.
If interference is present, the “potential outcomes” for an individual are much more complicated: they depend on not just the treatment a single individual received, but also on the treatment other individuals received.

With $n$ individuals, this is $2^n$ potential outcomes for each individual!
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With $n$ individuals, this is $2^n$ potential outcomes for each individual!

The assumption that there is no interference between treatment and control is part of the stable unit treatment value assumption (SUTVA) in econometrics and causal inference.

The other part of SUTVA is that there is only one form of treatment or control: e.g., if treatment is “taking a drug”, there should be no variation in the treatment group as to how much of the drug is taken.
Paradoxes
A new treatment for a disease is introduced, and compared against the existing standard of care (control).

Let $W = 0, 1$ denote control or treatment, respectively.

Let $Y = 0, 1$ denote the outcome disease or no disease, respectively.

Let $Z$ be the gender of the individual ($M$ or $F$).
A puzzle

You run an experiment with a large sample size, and equal numbers of men and women.

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No disease ((Y = 1))</td>
<td>Disease ((Y = 0))</td>
</tr>
<tr>
<td></td>
<td>0.1500 0.2250</td>
<td>0.1000 0.0250</td>
</tr>
<tr>
<td>Treatment ((W = 1))</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.0375 0.0875</td>
<td>0.2625 0.1125</td>
</tr>
<tr>
<td>Control ((W = 0))</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Here the numbers are the fractions of individuals in each category.)
A puzzle

Analyzing the results:

- On average, \( \mathbb{P}(Y = 1|W = 1) = 0.5 \) while \( \mathbb{P}(Y = 1|W = 0) = 0.6 \), so the treatment appears detrimental.

- On the other hand, \( \mathbb{P}(Y = 1|W = 1, Z = M) = 0.4 \) while \( \mathbb{P}(Y = 1|W = 0, Z = M) = 0.3 \), so the treatment appears to be beneficial to men.

- In addition, \( \mathbb{P}(Y = 1|W = 1, Z = F) = 0.8 \) while \( \mathbb{P}(Y = 1|W = 0, Z = F) = 0.7 \), so the treatment appears to also be beneficial to women as well!

What happened? (This is called Simpson's paradox.)
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What happened? (This is called *Simpson’s paradox.*)
Potential outcomes, causal effects, and sampling bias

Each man and woman has two potential outcomes \( Y(0) \) and \( Y(1) \), associated to control and treatment, respectively.

If we presume there was no sampling bias among men, (so \( W \) is uncorrelated with \( Y \) given \( Z = M \)) then the average causal effect among men is:

\[
\mathbb{E}[Y(1) - Y(0)|Z = M] = \mathbb{E}[Y(1)|Z = M, W = 1] - \mathbb{E}[Y(0)|Z = M, W = 0] = P(Y = 1|Z = M, W = 1) - P(Y = 1|Z = M, W = 0) = 0.1
\]
Potential outcomes, causal effects, and sampling bias

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If we presume there was no sampling bias among men, (so $W$ is uncorrelated with $Y$ given $Z = M$) then the average causal effect among men is:

$$
\mathbb{E}[Y(1) - Y(0) | Z = M] \\
= \mathbb{E}[Y(1) | Z = M, W = 1] - \mathbb{E}[Y(0) | Z = M, W = 0] \\
= \mathbb{P}(Y = 1 | Z = M, W = 1) - \mathbb{P}(Y = 1 | Z = M, W = 0) \\
= 0.1
$$

Similarly the average causal effect among women is

$$
\mathbb{E}[Y(1) - Y(0) | Z = F] = 0.1.
$$
So what is the average causal effect overall?

\[
\mathbb{E}[Y(1) - Y(0)] \\
= \mathbb{E}[Y(1) - Y(0)|Z = M]P(Z = M) \\
+ \mathbb{E}[Y(1) - Y(0)|Z = F]P(Z = F) \\
= 0.1
\]

So there is no paradox: if the causal effect for men and women is separately positive, it must be positive overall.
Potential outcomes, causal effects, and sampling bias

The issue is that in this example:

\[ \mathbb{E}[Y(1) - Y(0)] \neq \mathbb{E}[Y(1)|W = 1] - \mathbb{E}[Y(0)|W = 0]. \]

The reason is that if we ignore gender, there is a sampling bias:

- **Women** are more likely to be in control than treatment; men are more likely to be in treatment than control.
- And women have *higher* potential outcomes on average than men: the average outcome of a woman in treatment (resp. control) is 0.8 (resp., 0.7), while the same for a man in treatment is 0.4 (resp., 0.3).
- This combination of effects lowers the average outcome in the treatment group relative to the overall population (since the treatment group is primarily men), and raises the average outcome in the control group relative to the overall population (since the control group is primarily women).
Potential outcomes, causal effects, and sampling bias

The preceding analysis shows that ignoring gender creates an \textit{omitted variable bias} in our estimate of the average treatment effect.

Note that we assumed no further sampling bias beyond gender; the example makes clear that any such bias would only further cloud the true causal effect.
Another example: Berkeley admissions

Berkeley was sued for gender bias in admissions based on 1973 statistics: 44% of men were admitted, while only 35% of women were admitted.

But based on individual departments’ admissions statistics, there did not appear to be statistically significant gender-based discrimination (in fact if anything, some departments tended to favor women).

What happened is that there was a sampling bias: women were systematically applying to majors that were much more competitive.
The moral

This example is meant to illustrate how to use potential outcomes to carefully describe the causal effect of interest.

Perfect randomization makes up for a lot of deficiencies, but sometimes things are less than perfect.

Taking care to think through potential outcomes and sampling bias carefully can help avoid incorrect inference!
Observational data
Natural experiments

How can we make causal inferences *without* randomized experiments?

As the preceding lecture shows, we need to find other ways to eliminate sampling bias.

The phrase “natural experiment” refers to the fact that we look for structure in the data we are given that “mimics” an experiment we would have wanted to conduct.
Examples

Some examples include:

- Regression discontinuity analysis
- Propensity score matching
- Instrumental variables