

Causality

EC 421, Set 10

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Prologue

Schedule

Last Time

Autocorrelation and nonstationarity

Today

Causality

Upcoming

Assignment Due Friday

Next assignment Assigned this weekend

Causality

Causality

Intro

Most tasks in econometrics boil down to one of two goals:

$$y = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \cdots + \beta_k x_k + u$$

1. **Prediction:** Accurately and dependably predict/forecast y using on some set of explanatory variables—doesn't need to be x_1 through x_k . Focuses on \hat{y} . β_j doesn't really matter.
2. **Causal estimation:**[†] Estimate the actual data-generating process—learning about the true, population model that explains how y changes when we change x_j —focuses on β_j . Accuracy of \hat{y} is not important.

For the rest of the term, we will focus on **causally estimating** β_j .

[†] Often called *causal identification*.

Causality

The challenges

As you saw in the data-analysis exercise, determining and estimating the true model can be pretty difficult—both **practically** and **econometrically**.

Practical challenges

- Which variables?
- Which functional form(s)?
- Do data exist? How much?
- Is the sample representative?

Econometric challenges

- Omitted-variable bias
- Reverse causality
- Measurement error
- How precise can/must we be?

Many of these challenges relate to **exogeneity**, i.e., $E[u_i|X] = 0$. Causality requires us to **hold all else constant** (*ceterus paribus*).

Causality

It's complicated

Occasionally, **causal** relationships are simply/easily understood, *e.g.*,

- What **caused** the forest fire?
- **How** did this baby get here?

Generally, **causal** relationships are complex and challenging to answer, *e.g.*,

- What **causes** some countries to grow and others to decline?
- What **caused** President Trump's 2016 election?
- **How** does the number of police officers affect crime?
- What is the **effect** of better air quality on test scores?
- Do longer prison sentences **decrease** crime?
- How did cannabis legalization **affect** mental health/opioid addiction?

Causality

Correlation \neq Causation

You've likely heard the saying

Correlation is not causation.

The saying is just pointing out that there are violations of exogeneity.

Although correlation is not causation, **causation requires correlation**.

New saying:

Correlation plus exogeneity is causation.

Let's work through a few examples.

Causation

Example: The causal effect of fertilizer[†]

Suppose we want to know the causal effect of fertilizer on corn yield.

Q: Could we simply regress yield on fertilizer?

A: Probably not (if we want the causal effect).

Q: Why not?

A: Omitted-variable bias: Farmers may apply less fertilizer in areas that are already worse on other dimensions that affect yield (soil, slope, water).

Violates all else equal (exogeneity). Biased and/or spurious results.

Q: So what *should* we do?

A: **Run an experiment!** 🤖

[†] Many of the early statistical and econometric studies involved agricultural field trials.

Causation

Example: The causal effect of fertilizer

Randomized experiments help us maintain *all else equal* (exogeneity).

We often call these experiments **randomized control trials** (RCTs).[†]

Imagine an RCT where we have two groups:

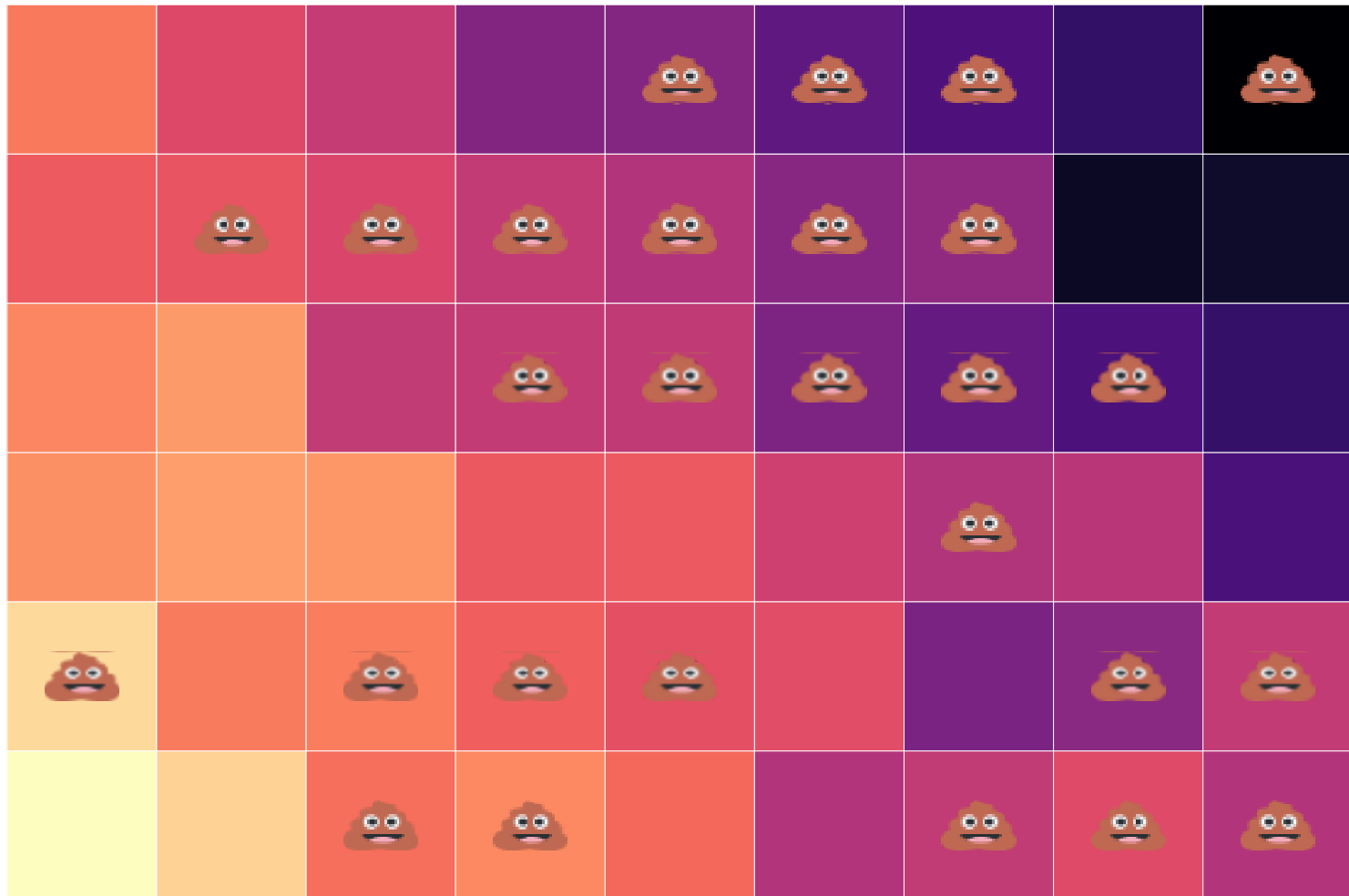
- **Treatment:** We apply fertilizer.
- **Control:** We do not apply fertilizer.

By randomizing plots of land into **treatment** or **control**, we will, on average, include all kinds of land (soild, slope, water, etc.) in both groups.

All else equal!

[†] Econometrics (and statistics) borrows this language from biostatistics and pharmaceutical trials.

54 equal-sized plots of varying quality plus randomly assigned treatment



Causation

Example: The causal effect of fertilizer

We can estimate the **causal effect** of fertilizer on crop yield by comparing the average yield in the treatment group (💩) with the control group (no 💩).

$$\overline{\text{Yield}}_{\text{Treatment}} - \overline{\text{Yield}}_{\text{Control}}$$

Alternatively, we can use the regression

$$\text{Yield}_i = \beta_0 + \beta_1 \text{Trt}_i + u_i \quad (1)$$

where Trt_i is a binary variable (=1 if plot i received the fertilizer treatment).

Q: Should we expect (1) to satisfy exogeneity? Why?

A: On average, **randomly assigning treatment should balance** trt. and control across the other dimensions that affect yield (soil, slope, water).

Causality

Example: Returns to education

Labor economists, policy makers, parents, and students are all interested in the (monetary) *return to education*.

Thought experiment:

- Randomly select an individual.
- Give her an additional year of education.
- How much do her earnings increase?

This change in earnings gives the **causal effect** of education on earnings.

Causality

Example: Returns to education

Q: Could we simply regress earnings on education?

A: Again, probably not if we want the true, causal effect.

1. People *choose* education based upon many factors, *e.g.*, ability.
2. Education likely reduces experience (time out of the workforce).
3. Education is **endogenous** (violates *exogeneity*).

The point (2) above also illustrates the difficulty in learning about educations while *holding all else constant*.

Many important variables have the same challenge—gender, race, income.

Causality

Example: Returns to education

Q: So how can we estimate the returns to education?

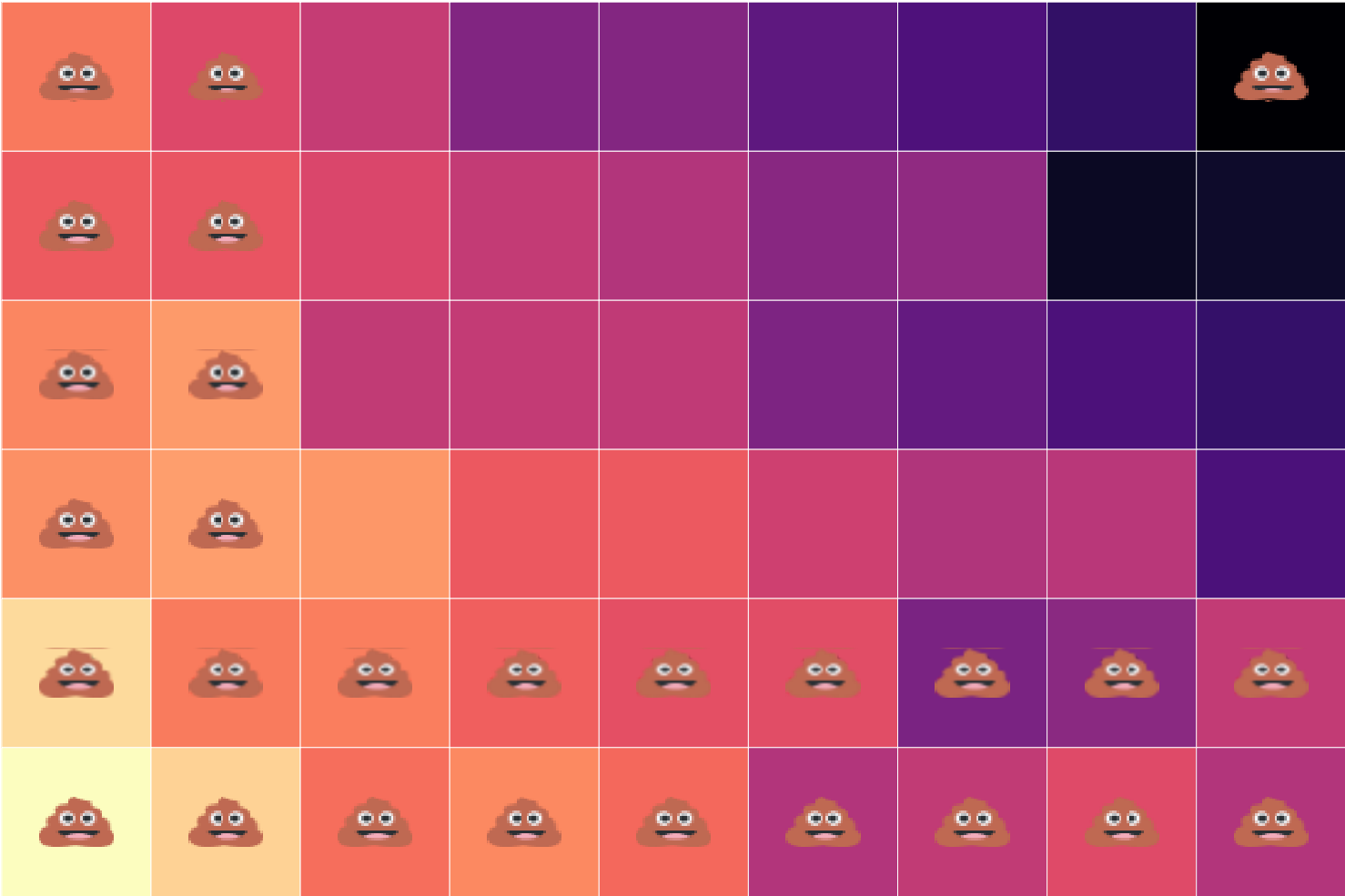
Option 1: Run an **experiment**.

- Randomly **assign education** (might be difficult).
- Randomly **encourage education** (might work).
- Randomly **assign programs** that affect education (e.g., mentoring).

Option 2: Look for a **natural experiment**—a policy or accident in society that arbitrarily increased education for one subset of people.

- Admissions **cutoffs**
- **Lottery** enrollment and/or capacity **constraints**

Unfortunate randomization



Causality

The ideal experiment

The **ideal experiment** would be subtly different.

Rather than comparing units randomized as **treatment** vs. **control**, the ideal experiment would compare treatment and control **for the same, exact unit**.

$$y_{\text{Treatment},i} - y_{\text{Control},i}$$

which we will write (for simplicity) as

$$y_{1,i} - y_{0,i}$$

This *ideal experiment* is clearly infeasible[†], but it creates nice notation for causality (the Rubin causal model/Neyman potential outcomes framework).

[†] Without (1) God-like abilities and multiple universes or (2) a time machine.

Causality

The ideal experiment

The *ideal* data for 10 people

```
#>      i trt  y1i  y0i effect_i
#> 1    1   1  5.01  2.56      2.45
#> 2    2   1  8.85  2.53      6.32
#> 3    3   1  6.31  2.67      3.64
#> 4    4   1  5.97  2.79      3.18
#> 5    5   1  7.61  4.34      3.27
#> 6    6   0  7.63  4.15      3.48
#> 7    7   0  4.75  0.56      4.19
#> 8    8   0  5.77  3.52      2.25
#> 9    9   0  7.47  4.49      2.98
#> 10  10   0  7.79  1.40      6.39
```

Calculate the causal effect of trt.

$$\tau_i = y_{1,i} - y_{0,i}$$

for each individual i .

The mean of τ_i is the
average treatment effect (ATE).

Thus, $\bar{\tau} = 3.82$

Causality

The ideal experiment

This model highlights the fundamental problem of causal inference.

$$\tau_i = y_{1,i} - y_{0,i}$$

The challenge:

If we observe $y_{1,i}$, then we cannot observe $y_{0,i}$.

If we observe $y_{0,i}$, then we cannot observe $y_{1,i}$.

Causality

The ideal experiment

So a dataset that we actually observe for 6 people will look something like

```
#>      i trt  y1i  y0i
#> 1    1   1 5.01   NA
#> 2    2   1 8.85   NA
#> 3    3   1 6.31   NA
#> 4    4   1 5.97   NA
#> 5    5   1 7.61   NA
#> 6    6   0  NA 4.15
#> 7    7   0  NA 0.56
#> 8    8   0  NA 3.52
#> 9    9   0  NA 4.49
#> 10 10   0  NA 1.40
```

We can't observe $y_{1,i}$ and $y_{0,i}$.

But, we do observe

- $y_{1,i}$ for i in 1, 2, 3, 4, 5
- $y_{0,j}$ for j in 6, 7, 8, 9, 10

Q: How do we "fill in" the `NA`s and estimate $\bar{\tau}$?

Causality

Causally estimating the treatment effect

Notation: Let D_i be a binary indicator variable such that

- $D_i = 1$ if individual i is treated.
- $D_i = 0$ if individual i is not treated (*control group*).

Then, rephrasing the previous slide,

- We only observe $y_{1,i}$ when $D_i = 1$.
- We only observe $y_{0,i}$ when $D_i = 0$.

Q: How can we estimate $\bar{\tau}$ using only $(y_{1,i} | D_i = 1)$ and $(y_{0,i} | D_i = 0)$?

Causality

Causally estimating the treatment effect

Q: How can we estimate $\bar{\tau}$ using only $(y_{1,i} | D_i = 1)$ and $(y_{0,i} | D_i = 0)$?

Idea: What if we compare the groups' means? *I.e.,*

$$Avg(y_i | D_i = 1) - Avg(y_i | D_i = 0)$$

Q: When does this simple difference in groups' means provide information on the **causal effect** of the treatment?

Q_{2.0}: Is $Avg(y_i | D_i = 1) - Avg(y_i | D_i = 0)$ a *good* estimator for $\bar{\tau}$?

Time for math! 🎉

Causality

Causally estimating the treatment effect

Assumption: Let $\tau_i = \tau$ for all i .

This assumption says that the treatment effect is equal (constant) across all individuals i .

Note: We defined

$$\tau_i = \tau = y_{1,i} - y_{0,i}$$

which implies

$$y_{1,i} = y_{0,i} + \tau$$

Q3.0: Is $Avg(y_i \mid D_i = 1) - Avg(y_i \mid D_i = 0)$ a good estimator for τ ?

Difference in groups' means

$$= Avg(y_i \mid D_i = 1) - Avg(y_i \mid D_i = 0)$$

$$= Avg(y_{1,i} \mid D_i = 1) - Avg(y_{0,i} \mid D_i = 0)$$

$$= Avg(\tau + y_{0,i} \mid D_i = 1) - Avg(y_{0,i} \mid D_i = 0)$$

$$= \tau + Avg(y_{0,i} \mid D_i = 1) - Avg(y_{0,i} \mid D_i = 0)$$

$$= \text{Average causal effect} + \text{Selection bias}$$

So our proposed group-difference estimator give us the sum of

1. τ , the **causal, average treatment effect** that we want
2. **Selection bias:** How much trt. and control groups differ (on average).

Next time: Solving selection bias.

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